Digitized by the Internet Archive
in 2010 with funding from
University of Toronto

http://www.archive.org/details/p3proceedingsmed04roya

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY
JOHN NACHBAR, M.A., M.D.
UNDER THE DIRECTION OF
THE EDITORIAL COMMITTEE

VOLUME THE FOURTH

SESSION 1910-11

PART III

ODONTOLOGICAL SECTION
OTOLOGICAL SECTION
PATHOLOGICAL SECTION
SURGICAL SECTION
THERAPEUTICAL AND PHARMACOLOGICAL SECTION

INDEX

1805-1834
1907

128620
10/7/13

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1911

LONDON:

JOHN BALE, SONS AND DANIELSSON, LTD.,
OXFORD HOUSE,
GREAT TITCHFIELD STREET, OXFORD STREET, W.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FOURTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1910-11

ODONTOLOGICAL SECTION

1805-1834
1907

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1911

Odontological Section.

OFFICERS FOR THE SESSION 1910-11.

President—

W. B. Bacon.

Vice-Presidents—

(Resident)

A. E. Clayton Woodhouse. Sidney Spokes. J. F. Colyer.

(Non-resident)

Edward Bogue. Walter Harrison. Rees Price.

Hon. Secretaries—

E. B. Dowsett (*Council*). W. W. James, F.R.C.S. (*Section*).
J. Howard Mummery (*Foreign*).

Curator—

J. F. Colyer.

Other Members of Council—

(Resident)

J. Lewin Payne.
P. F. Henry.
E. Bartlett.
H. W. Trewby.
C. F. Peyton Baly.
Douglas P. Gabell.
Stanley P. Mummery.
H. E. Cribb.
E. Balding.

(Non-resident)

C. A. Hayman.
G. R. Shiach.
S. G. Yates.
C. F. Sutcliffe.
J. Mountford.
H. R. F. Brooks.
L. Austen.
J. C. Storey.
J. A. Woods.

Representative on Library Committee—

Ashley Densham.

Representative on House and Finance Committee—

H. Lloyd Williams.

Representative on Editorial Committee—

W. W. James, F.R.C.S.

ODONTOLOGICAL SECTION.

CONTENTS.

October 24, 1910.

PAGE
Presidential Address. By W. B. Bacon 1
The Destruction of Teeth by Milk Germs. By F. J. Bennett, M.R.C.S., L.D.S. 3

November 28, 1910.

Pneumococcal Stomatitis. By Sidney Spokes, M.R.C.S., L.D.S. 11
By H. A. T. Fairbank, F.R.C.S.
(1) Endothelioma of the Gum 12
(2) Cyst of the Jaw, the Result of Traumatism 14
A Successful Surgical and Prosthetic Treatment of a Case of Excessive Hypertrophy of the Gums and Alveolar Processes of the Jaws. By Frederick Sleep, L.D.S. 18

January 23, 1911.

Some Inflammatory Eye Conditions due to Oral Sepsis. By Charles Goulden, F.R.C.S. 27

February 27, 1911.

Absorption of a Permanent Molar due to an Unerupted Premolar. By T. Stordy, M.R.C.S., L.D.S. 37
(1) Three Cases of Torus Palatinus. (2) Skull from a Barrow, showing somewhat Symmetrical Mandibular Exostoses. By W. de C. Prideaux L.D.S. 39
Model of a Maxilla with Exostoses. By P. J. Proud, L.D.S.... 41
Multiple Supernumerary Teeth preceding a Central Incisor. By A. A. H. Johnson, L.D.S. 42

March 27, 1911.

PAGE

A short Preliminary Note on the Distribution of the Nerve-fibres of the Dental Pulp. By J. HOWARD MUMMERY, M.R.C.S., L.D.S. 51

The Comparative Elasticity and Internal Structure of Cast and Rolled Gold Plate. By H. J. MORRIS, L.D.S., and ANDREW MCWILLIAM, A.R.S.M., M.Met. 57

April 24, 1911.

(1) An Erosion Cavity on the Lingual Aspect of a Lower Incisor. (2) Dislocation of the Pulp of a Temporary Incisor, the Result of a Fall, with subsequent Removal of the Pulp and Apex intact. By C. F. PEYTON BALY, M.R.C.S., L.D.S. 69

The Relation between the Teeth and certain Diseases of the Skin and Mucous Membrane. By W. KNOWSLEY SIBLEY, M.D. 71

May 22, 1911.

A Dentigerous Cyst with Cellular Contents. By W. W. JAMES, F.R.C.S., L.D.S. 85

Experiments and Observations on Bread, with Special Reference to the Causation and Prevention of Dental Caries. By J. SIM WALLACE, M.D., L.D.S. .. 90

June 26, 1911.

A possible Cause of Facial Asymmetry. By W. RUSHTON, L.D.S. 105

Cases of Mal-occlusion of Permanent Molars. By F. ST. J. STEADMAN, M.R.C.S., L.D.S. 110

A Case of Infection of the Cavernous Sinus due to Oral Sepsis. By GORDON TAYLOR, M.S. 113

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

JOHN BALE, SONS & DANIELSSON, LTD., 83-91, Great Titchfield Street, London, W.

Odontological Section.

October 24, 1910.

Mr. W. B. Bacon, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

I am deeply touched by the great personal compliment conveyed by your confidence in me as a successor, so early in the career of the Odontological Section of the Royal Society of Medicine, to such illustrious presidents as Mr. Mummery, Mr. Matheson, and Mr. Hern. It will ever be memorable that to Mr. Mummery we owe the present honourable and distinguished position of our Section, as representing an acknowledged and highly important speciality of general medicine and surgery. His successors have had, and will ever have, an onerous task in maintaining the high dignity and prestige of the office.

But I am fully aware that, after all, the Council of your Section, in nominating me as worthy of the highest honour at their disposal, have but followed the salutary traditions of the late Odontological Society. By alternating with London practitioners in the presidency those who represent the great body of provincial practitioners, and who, as practical exponents of our profession, have its welfare, its solidarity and progress no less at heart than the London or resident members, the Council have paid due honour to all classes of the profession. If, therefore, I cannot add to the scientific speculations of those more closely in touch with the opportunities for clinical research in the great metropolis, I have at least the satisfaction of bringing to you the ripe experience of many years of actual practice, during which time I have had the privilege of intimate and continuous friendship with very many worthy colleagues both within and without our Section, throughout the United Kingdom.

Since the very earliest years of the old Society one of the best appreciated traditions of our Section has been the avoidance of the certain febrile conditions, during which milk alone had been the diet, teeth which had hitherto been free from caries had been attacked in a rapid and unusual manner. I therefore set about testing the matter by direct experiment. For this purpose I considered the following questions should be answered:—

(1) Could milk, under favourable conditions, produce any or all the appearances of dental caries?

(2) Could the milk exist under such conditions in the mouth?

In order to answer the first question I obtained some sound teeth, and, having broken them into fragments, they were placed in glass-covered pots and covered with from 50 cc. to 100 cc. of ordinary cow's milk, and left in an incubator kept at a uniform temperature of 35° C. At various intervals the milk was changed—in some cases daily, in others after two or more days, up to a week. In all these cases the milk became curdled and an acid reaction to litmus was found after a few hours, becoming strongly marked in two or three days and ultimately intense, after which an alkaline reaction set in and with a characteristic putrescent smell. In these cases there was no admixture of saliva or other ingredient except that of milk.

The specimens, on examination, showed a gradual softening and flexibility of the denture, with patches of white opacity in the enamel. After a varying period, according to size, the dentine became sufficiently soft to be cut into sections on the microtome. These were stained by Gram's method and mounted for observation. I may here say that I find the earliest visible change in the enamel in these specimens, as also in others decalcified in bread and saliva, is not that of an opaque white spot, but whilst still translucent there is a ribbed and wavy pattern on the enamel not unlike the marking of the skin seen in finger-prints. This is due to the enamel prisms being seen in relief, and it is not until later, when more isolated, that the air surrounding the prisms so disturbs the light as to produce opacity. I think you will see by these specimens that the patches of opacity in the enamel do not differ in any essential feature from teeth decalcified in bread and saliva, or even in a weak mineral acid.

I will now show you some slides illustrating the result on the dentine of the fermented milk, as revealed by the microscope; but before doing so I will show two or three slides of natural caries for comparison, and also one or two specimens of artificial caries produced in the usual manner by the action of bread and saliva. The first slides (figs. 1 and 2) illustrate the action of the acid milk on the dentine, and you will

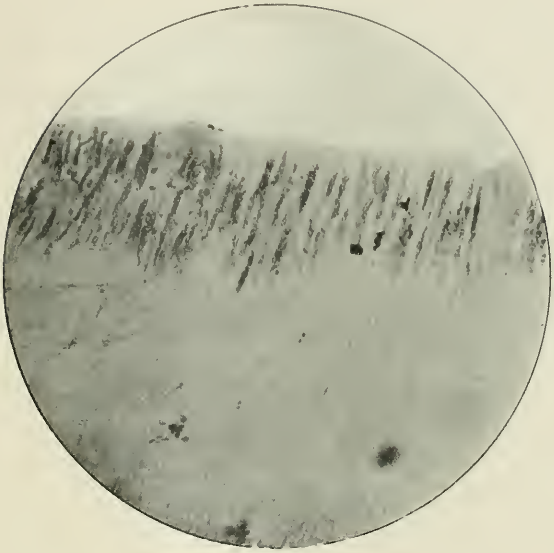


Fig. 1.

Photomicrograph from tooth decalcified by acid milk. Dentinal tubes occupied by cocci of milk. (Low power.)

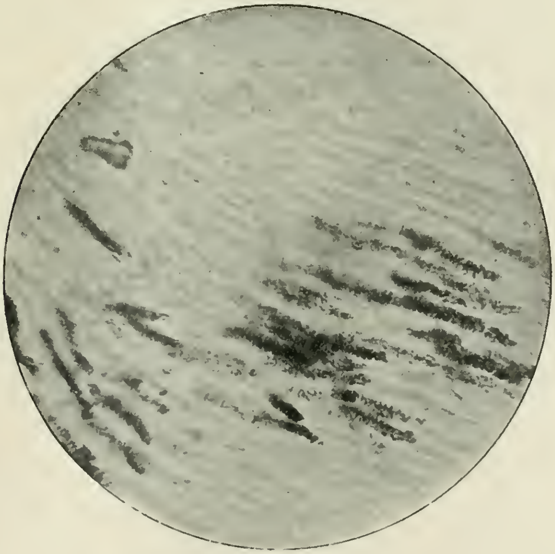


Fig. 2.

The same under high power.

see the bacteria have penetrated the dentinal tubes in various places, chiefly occupying the surface area, and there are also considerable portions of the decalcified dentine in which no germs are seen. Under a higher magnification the dentinal tubes are seen occupied by micrococci, sometimes three or four being crowded together in the width of the tubes. Some of the slides (figs. 3 and 4) also show the entrance of bacteria from the pulp surface, and where there are portions of the pulp *in situ* bacilli are here and there seen passing some distance into the neighbouring dentine. Another slide shows a section of the decalcified dentine, in which no germs are to be seen. This may be due to some defect in the cultivation of the germs in this particular specimen, but I think not; indeed, I believe it really throws light on the nature of the process which goes on, both in these specimens of milk caries and also in those of natural caries itself, for if the whole of this specimen has been decalcified by the milk, which has been converted into an acid by means of germs, and yet no germs are present in the specimen, then it is evident there must be two separate actions going on—one due to the specimen being in a bath of lactic acid, acting in the same way as a dilute mineral acid would do, and a second process due to the direct action of the germs when they come to occupy the dentinal tubes. In this particular specimen the second process is absent. I think these two actions, acting with different intensities, will help to explain the variations, both in the rapidity and the appearances of natural caries which we meet with in practice.

In returning to these experiments and considering the appearances in reference to those recognized as present in natural caries, one must remember that in the milk specimens the change has been brought about only by the acid milk and by the organisms of milk. In natural caries one has always to consider that unknown factor, the action of the saliva—as to what extent it favours or retards the action of the caries germs, and to what extent the liquefaction or digestion of the dental cartilage is due to the saliva or to the mouth organisms which it contains, and it has been my object to avoid introducing this factor of the saliva into the experiments, in order to simplify the problem by reducing the active agents to the smallest number.

It is therefore no matter of surprise that some of the appearances of natural caries—namely, the complete liquefaction of the dentine—are absent in my specimens. In other respects, I am bound to say I find very little difference in the microscopical appearances of these milk specimens and those of natural caries. But I need not here remind you

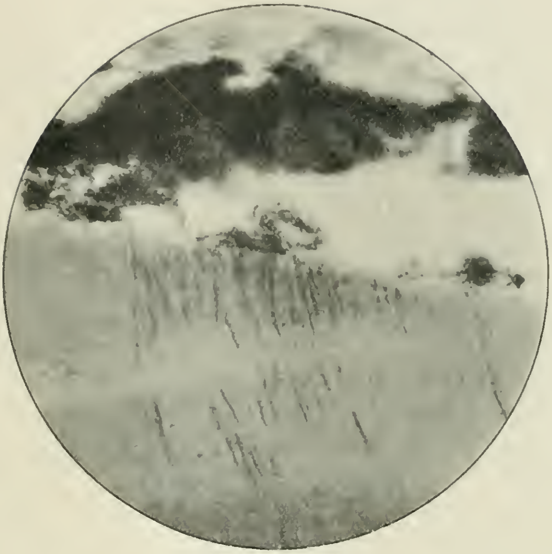


FIG. 3.

Bacilli from milk passing from pulp into enlarged dentinal tubes.

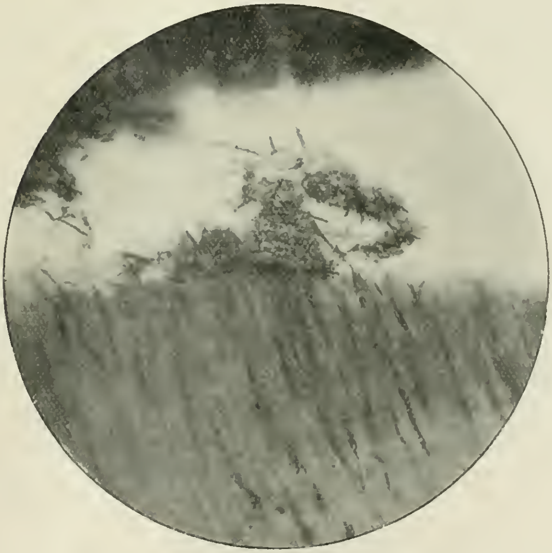


FIG. 4.

The same under high power.

that it is one thing to find germs in a tissue and another thing to cultivate them and learn their habits and species.

There are several varieties of the lactic-acid bacillus which grow in milk, and at least one form is found in the human mouth. Milk as used for household purposes is often acid to litmus paper, and one can readily believe that milk germs are conveyed to the mouth in ordinary milk; the oral cavity acts as an incubator, and therefore all the factors for the complete acid fermentation of milk may be present. But is there any reason to suppose the conditions in the mouth sufficiently resemble those mentioned in my experiments to make it probable that milk is really an important factor in producing dental caries?

In the first place, provided the milk is not curdled, we are dealing with a fluid and not with a solid substance, which is, therefore, less liable to be lodged between the teeth than is bread, although a mixture of bread and milk would find a ready lodgment. Milk mixes with the saliva, and would thus cling to any spot where viscid saliva lodges. Nevertheless it is not easy to find traces in the mouth of milk after it has been taken, provided it be fresh milk. But, by experiment, with glass tubes of capillary dimensions, I have proved that fragments of the skim of boiled milk can be retained in the mouth for ten hours and still show an acid reaction to litmus paper. When the glass tube is shaped into a minute cup, with a small opening, into which is poured ordinary fresh milk, it will be retained in the mouth in spite of the movement of the muscles and action of the saliva, and will remain acid for eight hours or more. Under ordinary circumstances, as Professor Pavlov has shown, milk appears, in dogs, to excite a copious flow of saliva which flushes away the milk, but I am not satisfied that this takes place in man.

On the whole it is not difficult to imagine that milk, taken as it often is three or four times daily, does at times lodge in the mouth, especially in mouths which are not frequently cleansed or where the milk has turned sour. But the mention of this word reminds me of the extensive use of sour milk as a form of diet and of its possible result on the teeth, and also suggests an inquiry into the habits employed in preparing the milk used by the peasants in Eastern Europe, though amongst these people it is a *vinous* fermentation which is encouraged, at the expense of the lactic acid. But all these topics must be left for further investigation.

I have to thank Mr. Hopewell-Smith for valuable assistance in photographing these specimens and also for cutting most of them; also to add that the slides of true caries shown for comparison are from the collection of the late Mr. Storer Bennett.

DISCUSSION.

Mr. STANLEY MUMMERY said he had long thought that the question of milk in relation to dental caries was one that needed investigation. In the ordinary course of events the milk would be probably flushed away by the saliva and have very little effect, but in those cases where a ropy condition of the saliva obtained, the milk probably became mixed with the saliva and adhered to the teeth. Also in febrile conditions where the saliva was of an acid nature the milk became curdled, and remained sticking round the teeth in the form of curds. He had often seen that condition in the mouths of patients suffering from typhoid and other fevers. It would be interesting to know whether Mr. Bennett had made any examination of the degree of acidity in connexion with his experiments. A very small percentage of lactic acid, 0·75 per cent., he himself had found, in some experiments made last year, was sufficient to decalcify certain teeth in twenty-four hours, and he would like to know the period of time required for decalcification in Mr. Bennett's experiments.

Mr. SIDNEY SPOKES was rather disappointed to find that Mr. Bennett had not included in his experiments some dealing with the particular lactic-acid bacillus which was so much recommended by the medical faculty nowadays to patients. It was not so very long ago that he ventured to state that he had seen certain suggestive cases where patients had been taking the lactic-acid milk and had neglected to wash the mouth out carefully afterwards, and that in those cases there had been a very great outbreak of rapid decay of the teeth. At the meeting of the British Medical Association in July[1] he had ventured again to make some allusions to the subject, and Mr. Kenneth Goadby, who was then present, stated that a weak solution of lactic acid was really a very good way to sterilize the mouth and get rid of other germs. That was analogous to the treatment recommended for the intestinal canal, because he understood the lactic-acid bacillus of a certain kind had the duty of clearing out undesirable bacilli and occupying their place. Perhaps, in that way, Mr. Goadby's sterilization by a weak solution of lactic acid might get rid of the other germs, but decalcify the teeth, as had been suggested by Mr. Stanley Mummery. He believed it was understood that the lactic-acid milk recommended by the medical faculty ought to be in a curdled condition, and, therefore, it would be in the best condition to remain around the patient's teeth. Dental surgeons had been doing everything possible in recent years to keep lactic acid out of people's mouths, but now that it was taken into the mouth in such a wholesale manner it certainly was time that somebody should carry out experiments, and bring forward the results of the inquiry.

Mr. F. J. BENNETT, in reply, said it had not been his desire to fix exactly the percentage strength of the acid developed, because he wished as far as possible to imitate the conditions in the mouth. It could hardly be imagined that the strength of the acid produced, either from bread fermentation or any other fermentation, was a constant quality; it varied, being weak at one time, strong at another. Therefore, it did not seem to him worth occupying much time upon that point in a preliminary communication; he would, however, bear it in mind, and would endeavour to fix the strength at which the greatest activity was present. He believed it was recognized pretty generally that anything above 2 per cent. of lactic acid began to destroy the germs. He could quite understand Mr. Sidney Spokes's disappointment, but it should be remembered he was trying to clear the ground by finding out what could be done on the teeth. When it was understood from experiments that there was a considerable possibility of milk affecting the teeth, then the ground was cleared to begin with clinical observations to see whether the teeth had suffered from acid-milk treatment or not. By proceeding in any other manner it would have been impossible to state definitely that the cause was due to the milk or to the bread, because, as a matter of fact, no one fed upon acid milk alone. He thought he was right in first going into the histology and pathology, and afterwards taking up the question of diet, &c. He hoped Mr. Spokes would bear with him until he was able to supply the facts desired. He remembered Mr. Goadby's remark that a weak acid was possibly deleterious to the lactic-acid germs. As Mr. Spokes had said, the dental profession had been accustomed to look upon an alkaline reaction of the saliva as the ideal to be aimed at, but according to Mr. Goadby that was not so. It should, however, be borne in mind that laboratory results were not quite the same as clinical results, and therefore he should not advise any member to try to maintain an acid reaction in his own saliva with a view to killing germs, as he might in that way decalcify his own teeth, which would then become an easy prey to germs.

---

[1] *Brit. Med. Journ.*, 1910, i, pp. 769, 770.

Odontological Section.

November 28, 1910.

Mr. SIDNEY SPOKES, Vice-President of the Section, in the Chair.

A Case of Pneumococcal Stomatitis.

By SIDNEY SPOKES, M.R.C.S., L.D.S.

THE Chairman exhibited a little girl, aged 15, who had complained of bad teeth, and for the last three months had suffered from headache and giddiness, pains in the stomach, diarrhœa, and occasional nausea. The appetite was good and the bowels and other functions were regular. She had suffered from epileptic fits, the last having occurred some two years ago, and she had also suffered from scarlet fever, whooping-cough, and chicken-pox. The father was healthy, the mother suffered from fainting fits, and there was one crippled sister, aged 12, who might be possibly tuberculous. There was nothing abnormal in the heart and lungs. The patient came to University College Hospital on August 3. The condition was diagnosed as pneumococcal stomatitis, treated for some time, and then sent to the dental department. It was found that she had been vaccinated twice by Mr. Goadby, and that on the closing of the out-patient department at the National Dental Hospital the girl was transferred to the University College Hospital. She was there treated with tonics and mouth washes, and when she was first seen in the dental department the gums on the right side of the mandible were hanging down in a very dreadful condition, looking like a piece of raw beef. There were two loose bicuspids, which were extracted, and that side of the mouth had cleared up and become very much more healthy; but from the first there was an extension in front of the mouth along the incisor region to the canine, and he fancied that had become a little worse within the last few days. She was also suffering from a conjunctivitis in the right eye that had come on quite recently, and probably had nothing whatever to do with her mouth. The gums were swabbed with dioxygen and the patient attended regularly, and then she was sent on to the ophthalmic surgeon. There was no history of any pathological examination having been made at University College Hospital, but Mr. Goadby, who remembered the patient, informed him that another case had responded very well indeed to the vaccine treatment and was reported to be cured. The patient was now shown in order to give members an opportunity of seeing the actual condition of a stomatitis which was due to pneumococcal infection.

DISCUSSION.

Mr. STANLEY MUMMERY asked whether any of the missing teeth were the result of the pneumococcal infection. There seemed to be but little pus there now.

Mr. SPOKES said he had stated that two of the bicuspids of the right mandible had been removed because they were very loose, but he did not know anything about other missing teeth.

Mr. CARL SCHELLING said there seemed to be some little ulcers and a considerable quantity of tartar, and he asked whether there was any reason why the ulcers should not be treated and the tartar removed.

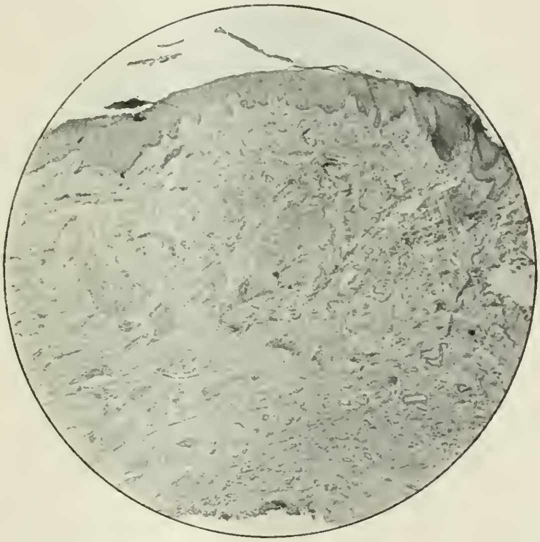
Mr. COLEMAN considered the conjunctivitis was due to direct infection from the mouth, by the child wiping its eye after placing its finger in its mouth.

A Case of Endothelioma of the Gum.

By H. A. T. FAIRBANK, F.R.C.S.

MISS J. V., aged 13, was sent to me by Mr. T. Stordy in December, 1909, with the following history: Two weeks before she had sudden bleeding from the gums after eating a biscuit. She was obliged to call in Dr. Sully, of Claygate, to stop the bleeding. The bleeding recurred, but stopped in ten minutes without treatment. She had had four teeth removed for crowding, the last extraction being performed eight months previously. There had been no pain. On examination a bright red vascular tumour behind, and extending round slightly to the sides of, the upper left second bicuspid tooth was seen. The tumour was not more than $\frac{1}{8}$ in. in width from the tooth, the posterior half of which it encircled. The tooth was absolutely healthy. The first molar was missing; it was one of the teeth extracted for crowding. The second molar was healthy. There was no oral sepsis of any kind. There was no sign of any enlargement of the alveolus or antrum. No enlarged glands. Since there was no sepsis, this swelling was regarded as a neoplasm which required immediate removal. A skiagram, taken by Dr. Ironside Bruce, showed the antrum to be normal and the alveolus apparently free from growth.

Operation: With the help of Dr. Sully the upper left second bicuspid and second molar were extracted, and then the tumour, the surrounding gum, and a piece of the alveolus, including the socket of the second bicuspid and the anterior half of the socket of the second molar, as well as that which corresponded to the absent first molar, were removed in one piece.



[illegible] Section of endothelioma of gum, showing the columns of endothelial cells in section beneath the epithelial covering of the gum.

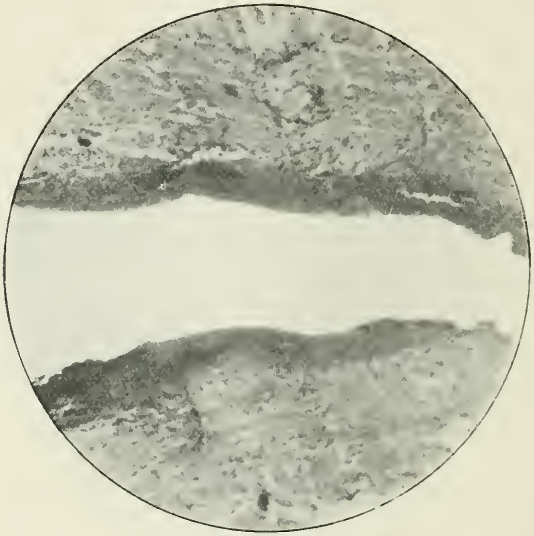
Examination of the tumour: Sections showed the growth to be an endothelioma apparently growing from the walls of the blood-vessels of the gum. It had not extended up the socket of the bicuspid tooth. The alveolus beneath it was only slightly roughened. Microscopical sections of this bone showed that the growth had not invaded it.

Last report: A few days ago Dr. Sully reported that there was no sign of recurrence—i.e., eleven months after the operation. The special interest of the case lies in the fact that, thanks to Mr. Stordy, the operation was performed while the growth was still minute.

Cyst of the Jaw, the Result of Traumatism.

By H. A. T. Fairbank, F.R.C.S.

C. B., aged 6, was brought to the Children's Hospital, Great Ormond Street, last August with the history of having two years previously accidentally knocked out two or three front temporary teeth. Six months ago a swelling of the right incisor region of the jaw was first noticed, and this had gradually increased. Examination revealed the presence of an unmistakable cyst, the size of a walnut, in this region, with very little bone in its anterior wall. The cyst was situated well above the alveolar margin, which was not displaced from its normal position. The left central permanent incisor was erupted and normal in position. The right central and lateral incisors were unerupted; their corresponding temporary teeth had apparently been knocked out. The right temporary canine was in its normal position.



Section of two portions of the wall of the cyst, showing epithelial lining consisting of several layers of cells.

Operation: A horizontal incision was made high up over the swelling and the cyst opened. There was no tooth projecting into the cavity of the cyst. The cyst wall was enucleated and removed. The crowns of the central and lateral incisors were then seen to be exposed, but these lay outside the cyst wall, and the part of the cyst wall covering them was of the same thickness as elsewhere. The lateral lay horizontally, the cutting edge being directed outwards; it was removed. The enamel of this tooth seemed to be perfectly formed, but only a little of its root was developed. The central was placed more or less vertically, and was therefore left undisturbed. The cavity was packed for a day and gradually filled up entirely.

Microscopical examination of cyst wall shows it to consist of fibrous tissue lined by epithelial cells, several layers in thickness, such as are seen in many dental cysts.

Origin of the cyst: It is suggested that the cyst has arisen by proliferation of epithelial remains of the tooth band, the proliferation being the result of inflammation following the traumatism which had been sufficiently severe to displace the lateral incisor. Since the cyst wall over these teeth was as thick as elsewhere, the teeth being definitely outside the cyst, the latter should not be included in the class of follicular odontomes.

DISCUSSION.

Mr. JAMES said, with regard to the first case, that he supposed it was an endothelioma of the lymph spaces, and he would like to know whether that was so. With regard to the second case, it was of very great interest, having regard to the paper read to the Section recently by Mr. Hopewell-Smith[1] and the two classes of odontocele there described. He himself thought that it corresponded very closely with an ordinary dental cyst which arose in epithelium. Malassez pointed out long ago that epithelium might give rise to a cyst of the same character. Dental cysts occurred very much later in life as a rule, and occurred as the result of irritation, the irritation usually being of an infective character. In the present case the cyst was clearly due to traumatism, although it did not follow, of course, that an infective process did not exist. Malassez described various positions in which the epithelium was found, and pointed out that cyst formation might occur in any one of these groups, and among them those situated in the position through which the tooth passed during its process of eruption. He himself had gone into the subject very carefully about a year ago,[2] and had read a paper before the Section referring to the collections of epithelium described by Malassez. In his original paper Malassez stated that the cysts were of epithelial origin. Previous to that the pathology was not clearly understood. He thought the cyst described that evening might be clearly classed with one of the cysts then described.

---

[1] *Proceedings*, 1910, iii, p. 121.

[2] *Proceedings*, 1909, ii, p. 166.

Mr. HOPEWELL-SMITH said he had not been quite clear as to the presence or absence of the capsule (or the dental follicle) in Mr. Fairbank's second case. If the cyst was outside that capsule, he thought it might be called, perhaps, an extra-capsular odontocele. He could not quite agree with Mr. James's remarks, because he had always understood that a dental cyst arose from septic irritation of the epithelial remnants in the periodontal membrane, and, of course, he was not certain whether sepsis had occurred in the case under discussion. Naturally he hoped that it would turn out to be an extra-capsular odontocele.

Mr. DOWSETT said he had it in mind to make a somewhat similar remark to that which Mr. James had made—namely, that the cyst seemed to be very like what was generally looked upon as a dental cyst. Some years ago he himself had shown before the Society a case of a dental cyst arising from a living tooth, and, although that was freely discussed at the time, it seemed to be fairly well established that it did actually arise from a tooth with a living pulp. There had been a tooth extracted close by many years previously, but the swelling had never become apparent until a year or so before the case came into his hands. There were definite reasons for thinking at the time that it did arise from the tooth—the shape of the tooth, for instance, and the position. Since that time he had seen another case, strangely enough, in exactly the same position, a lower premolar, and again, from all appearances, it seemed to be definitely a dental cyst, and microscopical examination confirmed this. It seemed to arise definitely from the premolar tooth, which was also a living tooth with no sign of caries whatever. He thought those two cases would fairly indicate that a dental cyst might arise from a living tooth, and not merely as the result of septic infection from a dead pulp. For that reason it seemed possible that the case under discussion might be a dental cyst arising, as Mr. James suggested, from a traumatism. The epithelial remnants of the epithelial sheath of Hertwig were simply little masses of epithelium which could be easily stimulated to growth by anything, or one might also say nothing, because growths did arise without any apparent cause. Such growths might start as the result of some injury, as well as from septic infection, and therefore he would rather like to classify the particular case Mr. Fairbank had shown as a so-called dental cyst.

Mr. COLEMAN said some years ago he had a case of a cyst in a boy aged about 7, and thought it compared in many respects to the type of cyst Mr. Fairbank had shown. He looked up the literature for the last fifty years on account of the early age of his patient, and could only find two earlier cases recorded, one by Heath and another by a practitioner in the country whose name he could not remember. In the boy referred to he (Mr. Coleman) removed the front of the cyst wall freely, and at the time of the operation failed to see a tooth or even feel one in the cyst wall. The tooth must have been in a somewhat similar position to that of Mr. Fairbank's case—viz., lying outside the cyst—because two or three days later, when he saw the boy, the enamel of the tooth was plainly exposed in the wall of the cyst. The only explanation he could give was that the tooth was originally lying outside the cyst wall, and in some way the lining over it had ulcerated, and so exposed the enamel. He scraped the cyst, and probably that was the cause of the epithelium disappearing, and subsequently exposing the subjacent tooth. With regard to the endothelioma, the perforations showing the limits of operation reminded him of an operation he had seen done some time ago by Mr. Clayton-Greene, which seemed to be a practical way of performing a partial excision of the mandible. Mr. Clayton-Greene made a linear series of perforations through the jaw by means of the dental engine, marking out the area of the jaw he desired to cut out, and the portion of jaw so defined certainly came away in a most pleasing manner and left a clean wound.

Mr. SPOKES said there appeared to be no sign of any one of the teeth said to have been knocked out having been driven up into the jaw, but he imagined that the temporary teeth with the roots absorbed to some extent might offer some sharp point which might possibly be septic at the time and provide the old-fashioned means of explaining a true dental cyst—that a septic root of a tooth was really the cause. It was a very early age to find such a case. The sharp point might have made an inoculation and afterwards been removed, but perhaps that was rather searching for an explanation.

Mr. FAIRBANK, in reply, said he had no definite information with regard to the condition of the mouth before the injury, the only history being that two or three teeth were removed by the injury. There was always the possibility of previous origin of the cyst from infection. With regard to Mr. James's remarks, was it not possible that Mr. James and Mr. Hopewell-Smith really meant the same thing and only called it by a different name? On reading the papers of Mr. James and Mr. Hopewell-Smith, it seemed to him that they were really describing the same thing but that they had chosen different names to designate it. It was admitted that there were many epithelial remains in the jaw as the result of the degeneration of the tooth band, and whether such a case as this were called a cystic swelling arising from those remains, an extracapsular odontocele, or a dental cyst, seemed to him to be of secondary importance. With regard to the two teeth, they were quite outside the capsule of the cyst, and there was nothing over them, so far as he could see after removal of the cyst. The tooth capsule, of course, might have come away with the cyst wall, for the two might have become blended by the pressure. With regard to Mr. James's query as to the origin of the endothelioma, he thought it arose from the blood-vessels. The tumour was very vascular indeed. As to the method of removing a piece of jaw, he was very interested to hear Mr. Coleman's account of what Mr. Clayton-Greene had done. In his own case he simply removed the second bicuspid and second molar teeth, and with bone forceps cut through the wall of one side of the socket and then the other, and did the same with the molar socket; then, with cross-cutting forceps, he made a horizontal incision and took the jaw, the gum and everything away in one piece so as to avoid the possibility of opening up the growth in any sort of way.

A successful Surgical and Prosthetic Treatment of a Case of excessive Hypertrophy of the Gums and Alveolar Processes of the Jaws.

By Frederick Sleep, L.D.S.

The subject which I am about to introduce to your notice is one which may, from its very rarity, fail to appeal at first sight to the practical mind of the average dental practitioner, distressed as he is with the thousand and one commoner problems which occur and recur with periodic and systematic regularity.

Whilst admitting, however, that the common and frequent vexations and distresses should be the subject-matter of our most constant attention, I yet think we must all own that an extended view of the ætiology and philosophy underlying the graver and rarer phases of oral abnormalities and disease tends frequently to throw unexpected sidelights on the minor everyday questions adverted to, and, by unravelling causes, helps to suggest new and unexpected methods of treatment of simpler and more frequent cases.

The more general studies of comparative anatomy, embryology, or bacteriology have all in their turn seemed, at their onset, to most of us as interesting academic departments of science, until speculative philosophy, by inference and deduction, has served actually to revolutionize our preconceived opinions and open up unexpected methods of future practice.

I propose, however, to present to you something more than the mere theoretical aspect of the case I shall deal with, and exhibit not merely a successful surgical treatment, but a prosthetic replacement, which has not only restored impaired function, but at the same time altered a marked deformity into normal appearance and removed a cruel ban from an otherwise ostracized individual.

Twenty years ago I was consulted on behalf of the patient, whose photographs and mouth models are hereby presented for your contemplation. The boy (fig. 1) exhibited a mouth so prognathous that the lips could not be comfortably strained over the hypertrophied gum and process. Suspecting the teeth were abnormal and irregular, I excised a strip of thickened gum from the labial surface of the superior maxilla, reaching from canine to canine. To my surprise I discovered, in most irregular position, merely the tips of the crowns of the front six permanent teeth, and so loose in the submucous tissue that I could have easily enucleated them with my thumb-nail; such development, in fact, as might have been expected in a child half his age. Remembering the old aphorism that "meddlesome surgery was worse than none," I determined to dismiss the case for some years and await further developments, with a possible view of eventually removing the fully-formed crowns and roots, and, by packing the sockets with some antiseptic material, inducing a new gum formation and an absorption of the external plates of the alveoli.

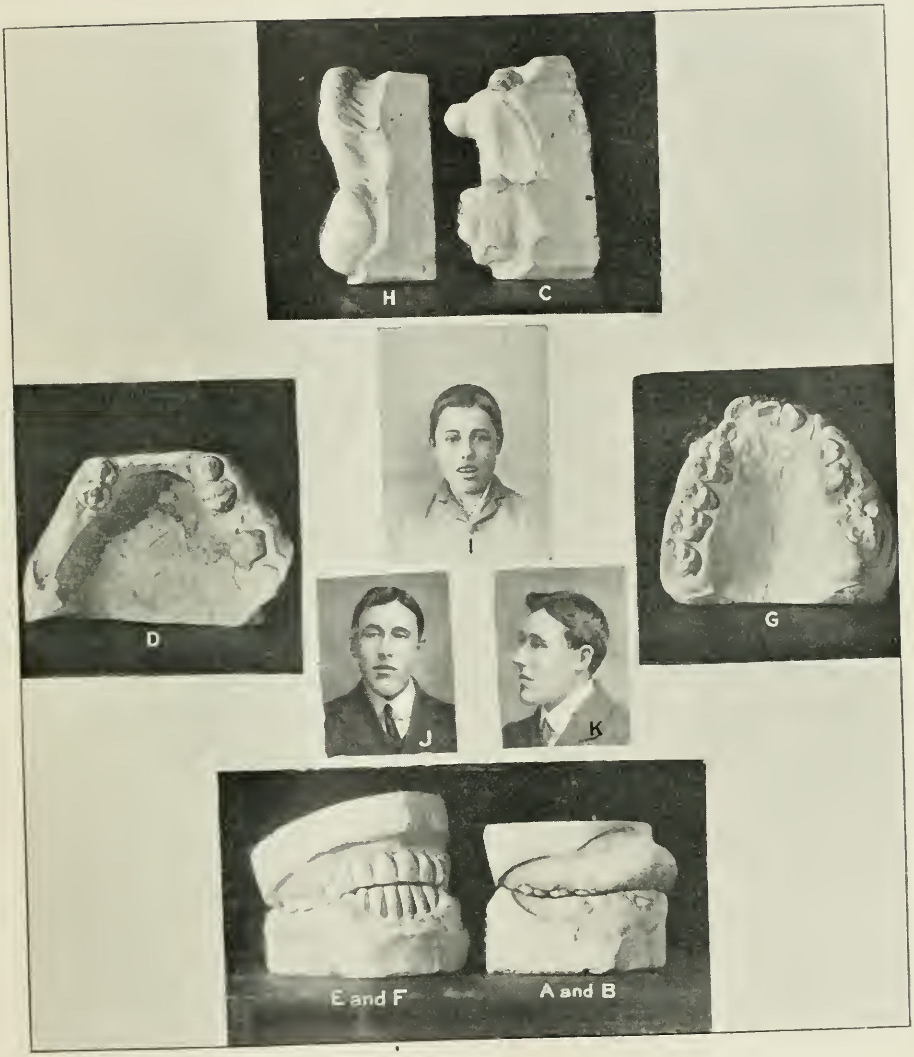
After a period of twenty years I received a letter from the patient, who was then in South Wales, informing me that he was anxious to know what could be done for him. He had found his condition such a serious bar to his advancement, that in despair he had taken a situation in a mine. By this time the six anterior teeth had only sufficiently risen to show just the tips of their misplaced crowns through the thickened bone and gum, although, as may be noticed, the bicuspids and molars had erupted very fairly. The condition of the deformity in front was however, if anything, more prognathous, owing, we may imagine, to the *vis a tergo* tendency of the additional permanent teeth, induced by their formation between the palate bone, which is being pushed forward by the pterygoid processes, in the more extensive relative growth of the greater over the lesser wings of the sphenoid. The increasing deformity was however somewhat disguised by the compensating length of lip, which naturally increases with the formation of the permanent teeth, and contracts with senility and the approximation of the wasting maxillæ through their loss. The patient was etherized, and the deeply set teeth removed, chiefly by compression of the forceps placed perpendicularly outside the gums, over nearly the whole length of their roots. The hypertrophied gum was next seized with a tenaculum and artery forceps, deliberately cut off with a bistoury, and the alveolar borders gouged and trimmed away with bone forceps. The patient was now dismissed for a short time to observe the effect of the pruning.

On the next visit it was evident that enough had not been removed, especially in the lower jaw, so, having injected novocain, I recommenced the trimming, and desisted only when I felt assured that the whole hypertrophied area of gum tissue was cleared beyond the probability of reformation, and the alveolus likely to retract in the healing process sufficiently to allow artificial teeth to be inserted, and the lips to meet normally. The raw surfaces healed kindly enough, and without any excessive granulations to cauterize. The only after-trouble was a recurrent hæmorrhage of a passive nature, which came on the night after the operation. The patient took train early to show me what he called "a tumour," formed during the night, which was, as you may guess, a lump of fibrinated coagulum over the seat of the trouble. The lump being removed, the surface was syringed with hot water and a wad saturated with turpentine was applied with pressure. The young man stayed with me till the evening, when, seeing that the matter was under control, he was dismissed.

Models **A** and **B** show the mouth twenty years before operating, whilst **C** and **D** exhibit the more normal appearance of the upper and lower maxillæ twelve months after the extractions and operations, at which time the impressions were taken for the artificial teeth. As may be partially seen in the imperfect lower model, the mouth was undercut not only on the inside, but, strange to say, on the outside as well. The removal of any composition impression material in the ordinary way that set hard, was impossible. On the other hand, anything ductile, like wax, could not be lifted without extreme distortion. To circumvent the difficulty, a smooth new lower impression tray was vaselined, filled with plaster of Paris, and inverted into position. When set hard, the tray was removed, a small circular saw was revolved on the engine in various directions, chiefly longitudinal, and the pieces prised off, collected, and re-arranged in the impression tray in the same manner as before, and cast.

It became possible, as may be seen from models 5 and 6, to grind the front teeth directly on to the gum without intervening vulcanite. My assistant was somewhat amused at my directions to make the lower set in the regular manner, insisting that I could never get it into place, leaving its removal from the mouth out of the question. The Gordian knot was however easily cut, by passing a hair saw between the central incisors, and coming through on the inside, dividing the set, of course into two parts, each section being hooked into place separately, from inside outward. There was no question as to the fixity of either, for the patient came to my table straight forth and ate a hearty meal.

The models **E** and **F** show the cases in the mouth. The two photographs (**J** and **K**), side and full face, were taken on the same day, and it will be readily seen that not only do the lips meet, but the contour of the face is perfectly restored. In a letter received from him afterwards, he cheerfully remarked, that he only suffered from one inconvenience—that people who had known him previous to the operations, had difficulty in recognizing him as the same individual.



Successful surgical and prosthetic treatment of a case of excessive hypertrophy of the gums and alveolar processes of the jaws.

The disease is by no means common, as may be inferred from the fact that Dr. Gross, of Philadelphia, confessed he had never met with but one case in all his vast experience. A short review therefore of such recorded cases as I could gather, may I hope be interesting, and serve as a preliminary to an investigation as to its cause.

Mr. Alfred Canton related another such an instance as I have brought forward; a case, however, in which no treatment was attempted. The lips could not be closed over the gums. The patient was described as half-witted, and with such a strong manifestation of the strumous diathesis, as to have raised the presumption in his mind that that disease was in itself the primary cause of the malformation.

In a case operated on by Mr. Gillivray, of Bendigo Hospital, the patient, aged 10, had several teeth extracted without result, and although several portions of gum were cut off at various times, it grew again persistently. He considered the origin in his case to be from the palatal portion of the gums, as the labial surface was comparatively sound. After removing the hypertrophied gum and alveoli there was no recurrence.

Mr. Pollock, in a similar case, excised the gum and cut away bone in successive operations, and cauterized; but, although he extracted the temporary teeth, there was a tendency to recurrence of the growth. The patient, a child, had an abnormal growth of hair and a tendency to tegumentary hypertrophy, and the centrals were described as abnormally large, the gums showing a tendency to be lobulated.

Mr. Erichsen, of University College, operated on a child, aged 2½, paring off hypertrophied gum, extracting some teeth, and cauterizing the cut surface. The enlarged gum invaded the incisive portions only, but it was remarkable that the temporary teeth had enlarged also. Five years after the operation it was entirely reproduced again, and completely covered the teeth. The patient presented peculiarities of the skin, subcutaneous connective tissue, periosteum, and ends of fingers and toes, whilst the patient's brother was similarly affected. He could associate no cause other than that the parents were first cousins.

Dr. Gross, of Philadelphia, has recorded a case which showed a tendency for some time to grow again after operation, and, considering the habit of the disease to enlarge papillary structures, such as the gum and teeth, it is interesting to observe that in this case there was an associated hypertrophy of the papillæ of the tongue.

Mr. Heath related a case of a child, aged 4½. Two years before operating the swelling began by the side of the temporary molars, which were just erupting, and spread right round the jaw. At this time she had fits about once a week; they were epileptic, and continued at the time he recorded the case, though at more irregular intervals. The gums covered the teeth except at the tip, which were, as usual, very irregular, and, what is unusual, the patient seemed to feel a comfort in cooling the mouth with cold, hard substances. He removed the gums and thickened alveolar border, leaving all the permanent teeth except a molar, which accidentally came away. The hæmorrhage was pretty free, and was stopped by actual cautery. There are no later remarks in this case, so probably a cure was effected.

The structure of the gums in these cases has always presented the same form, that of simple hypertrophy, just as we might find the enlarged heart, thyroid, tongue, or pregnant uterus. Mr. Charles Tomes declared it to be identical, microscopically, with the small polypi proliferating into a sharp or ragged-edged cavity, which is on, or just below, the level of the junction of the external with the peridental periosteum, and to be evolved chiefly from its fibrous stroma within the margin of the alveoli. In a strip of hypertrophied gum examined by Mr. Salter, not only was there an immense thickening of the fibrous tissue of the gum, but its papillæ had increased from five to fifteen times their normal size. The ætiology of such malformation is admittedly obscure, and its very scarcity has, I suspect, induced the pathologist to fasten the cause on any diathesis or disease which may accompany the hypertrophy. Personally, I do not consider the lesion to be contingent on any exciting cause, but that the potentiality was contained *ab initio* in the germinal vesical itself. Mr. Erichsen has pointed out that a great number of such patients manifest weak intellects, an observation confirmed by others and not altogether strange, for common observation will endorse the observations as to the occurrence of defects, deficiencies, and deformities, associated with the feeble-minded by Dr. Langdon Down and others.

I gathered that there was a tradition in the family of the patient that a great-grandfather had suffered from a similar trouble, and this is as far as it is possible to trace the pedigree of the labouring classes; its characteristic heredity, though often asserted with an air of finality, neither explains nor proves anything. The congenital symptoms of syphilis have been generally absent, though scrofula and rickets, from their frequent association, have excited the suspicion of a few. The real cause, however, would seem to lie deeper than mere constitutional debility, infection, or intermarriage, though it is generally understood that the latter is capable of awaking latent proclivities to disease and intensifying vicious tendencies, physical and psychical. In the case however I have brought to your notice, there is nothing in the history, or physical appearance of the family, that may help to suggest an adequate cause, and although the father and brother have both suffered in a minor degree from the same trouble, neither they, nor the rest of the family, exhibit any other deviation from the normal, but appear healthy, with a high moral tone and excellent mentality.

With a view of ascertaining a possibly remote or proximate cause, I subjected the following list of questions, and elicited the replies subjoined:—

(1) About what age did the gums first enlarge: was it when teething?

Answer.—In teething.

(2) What was his age (about) when he first called on me?

Answer.—About ten years.

(3) What is his age now?

Answer.—Thirty.

(4) Was there any great trouble with him when teething?

Answer.—No more than ordinary.

(5) Are you and your wife cousins, or related distantly?

Answer.—No relation.

(6) Has there been any history of gout or rheumatism in the family?

Answer.—None that we know of.

(7) Did any of either family suffer from epileptic fits, or become insane?

Answer.—No.

(8) Has any of either family at any time suffered from rickets, skin diseases, or enlarged fingers or toes?

Answer.—We don't know of any.

Mr. C. Tomes pointed out that the growth seemed to spring from the alveolar margins, and has suggested the experimental removal of the teeth without further operation, in the hope that, a corrective activity being excited, the parts may recede to a normal state. The argument would seem to gain weight from the fact that in the present case, and another recorded by Mr. Pollock, the hypertrophy was first noticed at the time of teething. The analogy, too, of cases of epulis, which are not observed to arise where the mouth is edentulous, and which, with other minor tumours of the alveolar border, osseous or otherwise, are often cured by extraction alone, tend to give a rational basis to the supposition.

As a counterweight, however, it must not be forgotten that where teeth of abnormal size are evolved, the cause of their enlargement must have preceded their formation. Fortunately, in the brother of the patient I have commented on, I found a case which served somewhat as a test to the theoretical value of the extraction theory as a working hypothesis.

Model G exhibits the condition of the brother's upper jaw prior to his extractions, and fig. D some eight months afterwards. In his case no further surgical treatment was adopted, owing to the modified condition of the deformity; nevertheless, the case serves the purpose happily, for, as may be seen, the teeth, if somewhat irregular in front, were all fully erupted, and the prognathism much less pronounced.

The experience, however, derived from this last case has convinced me that the constitutional perversion is a developing force that precedes and accompanies the advent of the teeth germs from their genesis to their maturity, and that a radical treatment of the gums and alveoli should accompany the removal of the teeth.

Although the patient expressed great pleasure at his changed appearance, the case did not compare favourably with the other, in which I had effected the removal of the hypertrophied parts. A side view of the upper models of the two brothers in their states A and G previous to operating, and C complement to A and H to G, when ready for the artificial substitutes, serve to reveal that the prognathism remains actually greater in the case in which it was primarily less. The gum was still so prominent (*vide* H) that, although short teeth were ground in on the crest, the lips would not fall sufficiently far to cover the gum line without voluntary effort on the part of the patient.

One peculiarity has attracted my notice, which seems to more frequently attend a variation of the disease. When the gums have been lobular, the teeth are generally described as of unusual size and formation, and more frequently normal when smooth; from which we may infer the former to be the graver lesion.

The teeth in both my patients' cases were perfectly normal in appearance and size, and did not suggest, by crown corrugations or root abnormality, manifestations of any pathological process or excitement in the fœtal or infant life which might have induced hypertrophy of the formative epithelium constituting the enamel organ, or of the submucous tissue of the papilla forming the dentine. Such observations would lead me to infer that the aberrant process underlying the disease may precede the advent of the dental organs and grow with it, and that they should not be suspected as the possible cause of the diseased excitement either by or through their mere presence.

Time and further observation can alone unravel these perplexities. The fact that the teeth are dermal appendages will serve to explain the association of excessive hairiness and fibroid enlargements of the skin, but the peculiarities of another disease in which there is found osteal hypertrophy of the maxillæ, and that of the connective tissue, and periosteal ends of the fingers and toes, seem to me to link the matter somewhat with diseases connected with disturbed function of the ductless glands. In acromegaly, for instance, we have, together with a facial enlargement not peculiar to the disease we have discussed, an enlargement of the upper jaw with a peculiar hypertrophy of the lower maxilla, and a general thickening of the extremities. Again, associated with this peculiar disease (acromegaly) we find either shrinkage or enlargement of the thyroid gland, with hypertrophy, or sarcomatous change of the anterior pituitary body. In gigantism, too, skiagrams have demonstrated decisive enlargements and hyperfunction with the same body, and great enlargement of the sella turcica to receive the hypophysis. As a significant variation, too, it is frequently noticeable that giants bear a distinctly acromegalic appearance, although they may scarcely be considered as diseased. Until such time, therefore, as the cause be determined, the subject must remain a surgical matter, and beyond the realm of purely medical aid.

I can only hope that if my efforts in this field have not succeeded in pointing out the certain cause of the trouble, and indicating methods of prevention, I yet may claim, at any rate, to have undertaken what may be practically considered a curative treatment in the graver case which I have had the pleasure of presenting to your notice.

Odontological Section.

January 23, 1911.

Mr. W. B. BACON, President of the Section, in the Chair.

Some Inflammatory Eye Conditions due to Oral Sepsis.

By CHARLES GOULDEN, F.R.C.S.

IN 1883, Mr. Henry Power read a paper before the Odontological Society "On the Relations between Dental Lesions and Diseases of the Eye."[1] In that paper, after referring to earlier observations that had been made in connexion with the relation of disease of the teeth and eyes, he went on to discuss the sympathetic nervous relations between the teeth and eyes, using as his text the analogy of sympathetic inflammation of an eye following injury to its fellow. It was then thought that sympathetic ophthalmitis was purely a reflex nervous phenomenon, and on these grounds were discussed paralysis of the various ocular muscles, which was in certain cases stated to be due to irritation of the branches of the fifth cranial nerve. Many diseases of the eye have been attributed to disease of the teeth, such as blindness, squint, paralysis of muscles supplied by the third and seventh cranial nerves, and glaucoma. There was a long discussion after this paper in which many speakers quoted cases in their own experience, and mention was made, besides the above conditions, of phlyctenular ophthalmia, cataract, conjunctivitis, spasm of the orbicularis, hypermetropia, lachrymation, and others. Such conditions as these will not be dealt with in this paper; it is with diseases of the eye due to infection either with organisms or their products, and with those cases especially in which these causes of diseases seem to have originated in connexion with the teeth—namely, with oral sepsis.

The eye may become infected in two ways, either by a perforating wound—exogenous infection—or by way of the blood-stream—endogenous infection.

---

[1] *Trans. Odonto. Soc. Great Britain* (1883-84), 1884, N.S., xvi, pp. 11, 69.

Panophthalmitis as a result of septicæmia has been long known, and occurs most frequently in septic conditions following labour. It also occurs after surgical operations, and in the course of specific infectious diseases. Such cases can be proved in a pathological examination by the discovery of the organism that is the cause of the septicæmia, and the same organism in the tissues of the eye that has been diseased.

There are also cases of chronic inflammatory eye diseases in which organisms have been found in the retinal and choroidal capillaries, and cases occur of chronic panophthalmitis which resolve without total destruction of the globe. Again, a patient may have one eye attacked by acute panophthalmitis, whereas the other may suffer from a milder inflammation which will resolve without suppuration. In such cases organisms have been found in the inflammatory foci in either eye, but in other cases it is reasonable to suppose that the less damaged eye was only injured by the toxins of the organism which caused suppuration in its fellow.

There are many cases of inflammatory eye disease in which no organism can be found on pathological examination, and it would seem that the organisms which caused the inflammation have died out, or that disease was merely due to toxins.

There are several diseases well known and acknowledged to be the cause of chronic inflammatory eye affections, and passing mention may be made of syphilis, gonorrhœa, and diabetes, each of which will cause a condition in many cases to be distinguished by the clinical picture of the eye affected.

If note be taken of a series of cases of iritis, iridocyclitis, choroiditis, and certain cases in which an exudate is present far forward in the vitreous, it will be found that only a certain, and that a comparatively small, number can be attributed to one of these causes. There is a large number, more especially of inflammation of the iris and ciliary body, due to some other cause or causes, and these are the cases which will be dealt with. Below will be found a series of cases of iritis, iridocyclitis with deposits on the back of the cornea, cases doubtless of cyclitis, with opacities far forward in the vitreous, and of choroiditis. These cases are not offered as statistics showing the relative incidence of the causes of these diseases, the number is far too small, but they do show what a large number of cases are unexplained. The cases number in all thirty-nine:—

5 of iritis.
21 of iridocyclitis.
4 of vitreous opacity far forward.
8 of choroiditis.
1 of panophthalmitis.

Of the five cases of iritis, syphilis was responsible for three, gonorrhœa for one, and diabetes for one; of the twenty-one cases of iridocyclitis, syphilis was responsible for four, one was a case of sympathetic ophthalmitis, which leaves sixteen cases for which a cause is to be found. It is this class of case that has been variously labelled plastic iritis, rheumatic iritis, and gouty iritis, and they are the cases most difficult to treat. This marks them out in contrast with iritis and iridocyclitis due to syphilis, and the majority of those cases due to gonorrhœa, which respond to treatment well and rapidly. A search was made for a focus of septic absorption. In one case the focus found was in the tonsils, the patient improving rapidly, so soon as these received attention, in spite of more than twelve months' treatment previously which had caused no improvement. In two cases no cause could be discovered, but in both cases the patients were women, and no actual examination was made of the uterus. In thirteen cases sepsis in connexion with the teeth was present, and although not every case improved as rapidly as one could wish after the teeth were removed, yet in many cases the improvement was so marked as to leave little doubt as to the connexion between the oral sepsis and the iridocyclitis. Here will be mentioned two rather striking cases, especially as one, the first, would certainly seem at first sight to support the opinion that many of the cases are due to rheumatism :—

A gentleman, aged 31, was seen in consultation with Dr. Radcliffe, of Oldham, on April 21, 1910, and gave me the following history: Sixteen months previously he was incapacitated from work owing to an effusion into each knee-joint. There had been no venereal infection at any time, and his doctor, finding he had a septic condition of his teeth, sent him to a dental surgeon, who removed all his teeth, and after a lapse of some months fitted him with an artificial set. This was done eight months previously to his consulting me. His knees improved a great deal, but yet a considerable amount of fluid remained. About this time also he consulted Mr. Robert Jones, of Liverpool, who diagnosed arthritis due to septic absorption. The cause of his consulting me was that his right eye had been red and painful for a week. Upon examination there was intense conjunctival and ciliary injection. There were no deposits on the back of the cornea. The aqueous was hazy. The iris was dull and hyperæmic. The pupil was partially dilated with atropine. No view of the deeper parts could be obtained with the ophthalmoscope owing to the haziness of the aqueous humour. The tension was normal and the vision $\frac{6}{12}$. The left vision was $\frac{6}{6}$. I inquired very carefully as to any venereal infection, especially as the effusion into his knee-joints was still present and the simultaneous presence of synovitis, especially of a knee-joint, and iritis is strongly suggestive of gonorrhœa as a cause. I also asked about his teeth, and he told me all had been removed eight months previously. I did not ask him to remove his plates to see if any stumps had been left as he had been under the care of a first-rate dental surgeon. He was treated in the routine way with atropine and hot bathings. Three days later (April 23) I saw him again. The eye was not so well. The pupil was fairly dilated but the cornea was becoming hazy. There were no deposits on the back of the cornea. The tension was normal, but the eye was very tender and he had been in great pain ever since I had seen him before. He was given a mixture containing 15 gr. of salicylate of soda to each dose to take three times a day. On April 29, six days later, I again saw him. The cornea by now was very hazy and there was a fair amount of infiltration, much as one sees in the early stages of interstitial keratitis in congenital syphilitics. The corneal epithelium was irregular and roughened. The pupil was wide, the tension normal, and the pain less. My notes for May 4 and May 10 are to the effect that the eye remains "in statu quo." On May 12 he came to my rooms considerably brighter and told me he had just been to his dental surgeon to complain of a little tenderness in the upper jaw on the right side and of a bad taste in his mouth, which he had noticed both before and after the clearance of his mouth ten months previously. He was examined and a septic root of a right upper premolar was found and removed. The patient declared to me that he was already better, although the extraction had been performed but four hours previously. My note for May 25 and June 1 states that the eye is much better and that the cornea has sufficiently cleared to see a large crop of deposits on the back of it. On June 14 he was very much improved and there was practically no injection at all. On July 1 I saw him again, and advised him still to continue with his atropine. On August 4 the vision in the affected eye was $\frac{6}{5}$. It should be stated that his knees improved as his eye improved, confirming the diagnosis that the effusion was due to septic absorption, as had been suspected from the early days of his illness. I saw him again on September 19 and October 3, when he was perfectly well. I examined him again on January 16, 1911, and he still continued quite well, and his knees are also quite well.

I have quoted this case at some length, as it seemed one of considerable interest and affording a somewhat striking connexion between disease and causative agent.

The other case in this connexion is one of a gentleman, aged 29, whom I saw on August 2, 1909, in consultation with Dr. Menzies, of Rochdale. He had had iridocyclitis in the right eye for two years, and in the left for eighteen months. When I saw him his condition was advanced and his eyes were very seriously damaged. The case had been considered one of tuberculous nature. Both eyes were much injected, the pupils extensively bound down, and there were copious deposits on the back of the cornea. In the right eye there was a small yellowish mass below at the angle of the anterior chamber. He had previously been treated with mercurials, iodide of potassium, salicylate of soda, and also with cacodylate of guaiacol, besides local treatment with atropine. He had benefited but little by these remedies. His teeth were in a very bad state and he was advised to see his dental surgeon. He was next seen on August 20—that is, eighteen days later. He had had all the septic teeth removed the day after he had been advised, and already reported himself very much improved. The eyes were certainly very much better and the small yellowish mass in the right eye had disappeared. Since then I have not seen him, but Dr. Menzies, of Rochdale, under whose care he is, tells me he is very much improved, and that the eyes are now quiet.

Of those cases in which an exudate is seen in the vitreous far forward the search for a septic focus in the four cases revealed the nose in one instance, in one case a recent attack of gastritis, and in two more sepsis in relation to the teeth. As the two cases in which the mouth was at fault are still under treatment, the result of cleansing the mouth cannot yet be ascertained, but these cases are closely allied to iridocyclitis.

Of the eight cases of choroiditis, six were due to syphilis, and in two no other cause could be found save the septic condition of the teeth. One case will be mentioned as being of a class not often seen, but still distinct clinically. It is a case in which iridocyclitis was present in the left eye, and on a further investigation a mass of exudate was found in the choroid.

The patient was a woman, aged 40, who had oral sepsis. She was immediately sent to the dentist, who removed several lower incisors and also some septic stumps. The iridocyclitis quickly disappeared, but the choroiditis resulted in great loss of vision.

Closely allied to these cases of choroditis is the one case of panophthalmitis in this series, and it is a case in which the evidence is very strong in connecting septic conditions of the teeth with ocular disease.

The patient was a woman, aged 40, who was admitted to the Oldham Infirmary on April 18, 1910. She gave the following history: About ten days previously she had had bad toothache, and four days before admission she had had two teeth extracted after injection of the gum. Twenty-four hours later she became extremely ill and had to go to bed. Twelve hours later—i.e., thirty-six hours after the extraction—the right eye became red and painful. The eye increased in pain and redness. She was seen on the night of April 19. Her temperature was 103·2° F. She was very ill, and was apparently suffering from septicæmia. The right eye was in a condition of panophthalmitis. So far as could be judged, she had had an extraction in the region of the two left lower premolars. The gum was considerably lacerated and swollen, and was in a very foul condition in that neighbourhood, and the odour of the breath was very objectionable. The condition was explained to the husband, and the right eye was removed early the following morning. At the operation 2 dr.

of blood were withdrawn from the right median basilic vein for pathological examination. The patient died the next day at 10 p.m., her temperature before death reaching 107° F. There was no post-mortem examination. The blood was sent to the Pathological Department of the University of Manchester, and the eye to the laboratory of Moorfields Eye Hospital for examination. The report from Manchester showed that the *Streptococcus pyogenes* had been cultivated from the specimen of blood, and Mr. Coats, of Moorfields, reported that the eye showed a typical condition of panophthalmitis, and that, by suitable staining processes streptococci could be seen in the vitreous.

If I may sum up what I have attempted to show, it is this: That from an ophthalmic surgeon's point of view, we have in the mouth a source of infection which in all probability is at the root of much mischief that occurs in the eye, and although the number of cases enumerated is small, it is not on these cases alone that conviction rests, but upon a very great number of cases that have come under notice at different times and which have all been treated on the supposition that the mouth was at fault, in many cases with marked success, and the persistent teaching at Middlesex and Moorfields.

DISCUSSION.

Mr. W. W. JAMES said it was an exceedingly difficult thing definitely to associate a condition of oral sepsis with any disease whatever unless the organism was discovered to be present in the mouth and also in the lesion that occurred. In the last case mentioned by the author, for instance, the organism was found in the blood and also in the eye, in fact it was a case of general septicæmia in which the eye was involved, but the organism was not actually cultivated from the mouth. He would like to know how far the author thought he was really justified in drawing a conclusion that cases of eye infection were definitely associated with septic mouth conditions. It was an exceedingly difficult question to answer.

Mr. F. J. BENNETT congratulated the author upon bringing new matter before the Section. People, though at first incredulous, were beginning to regard pyorrhœa alveolaris as a formidable disease, and to recognize its possible extension to other parts. He did not quite gather from the author whether he included only cases in which there was septic absorption from the teeth and secondary infection of the eye, or if he also included cases of direct infection through the hand rubbing the eye or in any other way. He alluded to that point, because years ago the subject was dealt with by Sir Jonathan Hutchinson, and was reported in the *Transactions* of the old Odontological Society.[1] Sir Jonathan pointed out that accompanying pyorrhœa alveolaris, or as it was then known, Riggs's disease, there were occasionally affections of the eyelashes and eyebrows, and even of the hair itself. He spoke vaguely of the pathology, putting forward more as a clinical fact that he had observed, in the course of his long practice, that those two conditions co-existed. He guarded himself, however, against the assumption that it was due to the direct spread of the disease by means of rubbing with the hand, though he was inclined to think it was a disease of the hair-follicles. Again, lately, in the *British Medical Journal*, Dr. Pagan Lowe, of Bath, in answer to some letters on the subject of gonorrhœal iritis, considered that many of the cases recorded were to be classed as due to pyorrhœa alveolaris. If direct infection occurred, how was it that dentists as a class escaped? It was a rare thing to hear of any dentist suffering from suppurative conditions of the eye.

---

[1] *Trans. Odont. Soc. Great Britain*, 1889, N.S., xxi, p. 155.

Mr. W. B. PATERSON said he was not wholly convinced by the statements made in connecting the teeth with eye disease. In the first case quoted by the author, that of a gentleman with synovitis of the knee-joints and iritis, he had pictured to himself some venereal infection as a cause, and he could not trace from the descriptions given by the author any connexion between the solitary premolar root in process of extrusion and the eye and joint troubles; nor in the absence of bacteriological examination, more than coincidence in the clearing up of the iritis and the disappearance of the synovitis after the removal of the root. If the cause was to be explained by absorption of *materies morbi* into the blood he had nothing more to say than that it was a possible one: but from the description of that case, as he had understood it, he did not see any direct connexion between the tooth root, the eye, and the knee-joints. With regard to the other case where, as the patient got better in health, the eye cleared up, there also seemed to be no dental connexion. It was possible, of course, that there was a greater connexion between diseases of the dental periosteum and other organs of the body than was supposed, but he was reminded of a discussion that took place in the Society many years ago when his old teacher, the late Mr. Henry Power, traced a great many conditions of the eye to teeth.[1] In those days nervous influence entered very largely into the question. Some few years afterwards a house surgeon of Mr. Power's, now Sir William Collins,[2] read a paper before the Society in which he was most iconoclastic, pulling down a great many idols: in fact, he disputed altogether the connexion between affections of the teeth and the eyes except in cases of blepharospasm and lachrymation, and those where direct spread of inflammation from the mouth upwards through the tissues of the cheek, or through the maxilla, occurred. As a dental surgeon attached to a large general hospital, he (Mr. Paterson) could say that it was a fact that in the course of the year few eye cases were seen in the dental department for treatment. In Mr. Power's day there were more, but the results of dental treatment were often difficult to verify or to trace. Still he was not prepared to say that in cases of iritis or of any eye disease a patient with a septic mouth would not be benefited in general health by proper dental treatment, and his eyes improved thereby—he thought he would.

---

[1] *Trans. Odont. Soc. Great Britain* (1883-84), 1884, N.S., xvi, pp. 11 69.

[2] *Ibid.*, 1891, N.S., xxiii, p. 161.

Mr. KENNETH GOADBY said Mr. Henderson, who was very interested in the question of the relation of diseases of the eye to diseases of the teeth, had sent him from time to time at the National Dental Hospital a number of cases of diseases of the eye of the type which he thought were associated with diseases of the mouth. He had been collecting notes and had particulars of several cases, one a case of keratitis with early cyclitis, a case of recurrent keratitis, and a case of keratitis profunda. In all those cases there was found to be distinct infection in the mouth from various micro-organisms; in some cases staphylococci and in some streptococci. The cases had been treated at the Eye Hospital in the ordinary way and had not improved, and they were at once put under inoculation treatment and as soon as possible the condition of the mouth was cleared up. Of the eight cases he had had sent to him during the last year—the cases that had failed to respond to previous treatment, which he thought was always a fair criterion as to whether any given treatment was of value—every case was stated at the Moorfields Hospital to be cleared up entirely, and so far there had been no recurrence. There seemed, therefore, to have been some definite connexion between the eye and the infective condition of the dental periosteum.

The PRESIDENT asked whether Mr. Goadby could mention the conditions of the mouth in those cases.

Mr. GOADBY said the first case of episcleritis had been under treatment at intervals for four years at Moorfields. The mouth was filthy. There were two teeth from which pus was exuding. The right side of the lip and the mouth was swollen and œdematous and there was a large ulcer on the right-hand side of the tongue. The patient, a woman, was complaining of rheumatism and swollen legs and a good deal of crepitus of the right knee-joint. The rheumatism cleared up with the treatment in that case. The other case, that of recurrent keratitis, had tingling pains in the hands and up the arms, especially at night. She was a washerwoman, and the pains prevented her doing her ordinary work. She had a very septic mouth, pus exuding from a large number of teeth: there was no loosening of the teeth. There was very foul breath, with a foul tongue and other concomitants of septic infection from the mouth in the way of constipation and occasional diarrhœa. She had no teeth extracted at all, and was merely treated by means of streptococcic vaccine. In the case of episcleritis there was distinct ulceration. There were four teeth discharging pus in the lower jaw—the two lower centrals and the two laterals. The case was inoculated with mixed vaccines of *Micrococcus catarrhalis* and pneumococcus. Another case of recurrent episcleritis: The patient was a woman, aged 46, and she had had the right eye and then the left eye affected. Six years previously she had had several teeth removed, and at the present time had only one remaining in the upper jaw; that tooth was very septic. There was also evidence of infection of the alveolus. He was able to pass a needle into the alveolus, and notwithstanding that the teeth had been removed some time ago there was evidence of rarefaction and old infection into the alveolus—a condition that was no doubt keeping up the general infection.

It was absorption from the tissue rather than direct active pus formation. It was rather an interesting case, because there were no organisms to which she reacted, but he curetted and cleared out a soft place very thoroughly and isolated a streptobacillus, and the condition cleared up.

Mr. ASHLEY DENSHAM thought there was evidence to convince most people that there was a relation between septic conditions of the mouth and arthritis of various joints, especially the knee-joint. There was also a definite connexion between inflammations of the synovial membrane and inflammations of the eye. Therefore it seemed reasonable that the cases the author had brought forward should be considered from the point of view of oral sepsis. The relation to eye affections was so new to dentists that a bacteriological study would no doubt shortly be undertaken, and definite evidence would be brought forward which would convince them of the part played by oral sepsis in some inflammatory conditions of the eye. With regard to septic conditions of the mouth causing arthritis, he had a relative who suffered from acute arthritis which was regarded as rheumatic. There were excruciating pains, with effusion in the knees and shoulders, and she was eventually sent to Bath, where she had a bad attack in both knees. It was recognized by the physician that the mouth was not quite healthy. A plate had been made covering a large number of septic stumps. In the middle of an acute attack of arthritis those septic stumps were removed by a dentist; that same evening the temperature dropped, the pain disappeared, and next morning the swelling of the knees began to subside; she had never since had the slightest sign of any arthritis. He thought there must be a large number of members present able to instance cases where arthritis had disappeared after treatment of the mouth.

Mr. G. THOMSON said he thought there should be some injury or other cause to account for an eye becoming infected. It seemed to him that when there had been "oral sepsis" and pus showed itself in other parts of the body there had been an injury, such as a blow on the part affected—e.g., on the arm when pus was found between the periosteum and the bone. There seemed to be nothing of that kind in the cases related that evening.

Mr. JAMES said he absolutely agreed with what the author had said and also with what Mr. Densham had said. He thought dental experience was considerable with regard to the association of arthritis with oral sepsis. He had asked the author a question which he feared had been interpreted as expressing an opposite view.

Mr. RAYNER BATTEN supported the author with regard to the relation of eye diseases to oral sepsis. In most cases of serous iritis it was necessary to seek some constitutional cause. More often than not it could not be traced either to syphilis or to tubercle, and the constitutional cause was very often not found. But in a large number of cases there was a septic condition of the mouth and these became distinctly better after the removal of the septic cause. He thought the connexion was due to absorption and not to direct infection. In some cases, where there were bad corneal ulcers, it was possible there might be some direct infection by the fingers or otherwise, but in the cases of serous iritis and choroiditis such cases must be constitutional in origin and the septic condition in the mouth was very likely to give rise to it.

Mr. J. F. COLYER emphasized Mr. Paterson's view. He would like to know in what way the author associated the sepsis with the eye condition, how he thought the infection was brought about, whether direct or via the intestinal canal; also how far the cases were associated with caries pure and simple, and how many were associated with periodontal disease. His own feeling was that as far as these conditions were concerned there was very little absorption through the tissues of the mouth.

Mr. GOULDEN, in reply, said that with regard to Mr. James's point as to the connexion of the case of panophthalmitis with the extraction, there was no other cause that could be discovered, and the clinical history of the case, a wound in the mouth followed in a few hours by septic condition, seemed to him to be sufficient evidence that the septicæmia was probably due to something injected into the gum, perhaps with a dirty needle. Certainly the organism found in the blood was the organism that caused the panophthalmitis. He did not take a culture from the mouth as he thought it was hopeless to do so. He understood that bacteriology as applied to the mouth was unsatisfactory, there being so many organisms that it was difficult to decide which organism was the cause. Knowing what he did now, if he had a similar case he would certainly take a culture from the mouth. He was not very sanguine of finding anything in the blood, but in very few cases of panophthalmitis had the blood been examined and therefore he took a culture. With regard to direct infection of the eyes by the fingers, &c., he had seen cases, two quite recently, that he believed were due to direct infection from the mouth, but he had only lately paid attention to such cases and it would be a little premature to give his opinion on the subject. He had had two cases of corneal ulcer where both patients had very objectionable conditions of their mouths and both had abscesses connected with bad teeth. With regard to the roots of eyelashes becoming infected, he was afraid he could not say whether it was due to direct infection or not; it was nearly always a staphylococcic infection, but whether it arose from oral sepsis he could not say. He was pleased to hear Mr. Paterson's remarks. Mr. Paterson evidently thought that the case of synovitis and eye infection was a case of venereal infection, but the man had never had any antisyphilitic remedies at all. It was considered that the condition of the knees was due to oral sepsis and the patient was always treated on that line. The septic condition of the knees was diagnosed also by Mr. Jones, of Liverpool. He was sorry Mr. Henderson was not present, because when he first started work at Moorfields it was Mr. Henderson who first pointed out to him the likely connexion between diseases of the teeth and diseases of the eye. With regard to the question as to how the infection was caused, in the particular cases he had mentioned he thought the connexion would be by the blood-stream. There was no external injury. The cases which he noted as causing eye disease were not, as far as he could judge, cases of ordinary dental caries, but cases in which there was pus formation around and stumps of teeth the crowns of which had disappeared.

Odontological Section.

February 27, 1911.

Mr. A. Clayton Woodhouse, Vice-President of the Section, in the Chair.

A Case of Absorption of a Permanent Molar due to an Unerupted Premolar.

By T. Stordy, M.R.C.S., L.D.S.

This case is a very good example of the absorption of a permanent molar due to an unerupted premolar. The patient was a girl aged 10. The four six-year-old molars were in a bad state, and it was decided to remove them under nitrous oxide. The tooth in question was the left upper molar. On the tooth being extracted I saw that the anterior buccal root was missing, and, thinking it had been broken off, I sought to remove it, when to my surprise a premolar was removed. On further examination of the extracted tooth I saw that the anterior buccal root had been almost completely absorbed, and there was a large excavation on the buccal side of the palatine root. The second temporary molar was in position and quite firm. The first temporary molar had been shed and the first bicuspid had erupted. I think there are two points to be noticed in this case. The first is the amount of absorption that has taken place. One sees absorption of this kind much more frequently in the twelve-year-old molar caused by an unerupted wisdom tooth. The second point of interest is the amount of backward displacement of the premolar, which I am presuming is the second premolar. This tooth normally develops and erupts on the palatine side of the second temporary molar, but in this case the second premolar had moved back to a very considerable extent, and, judging from the manner in which the absorption had been produced, it looked as if the premolar was erupting along the line of the anterior buccal root of the first permanent molar tooth, and for a great portion of its existence had occupied a position much posterior to that normally occupied.

DISCUSSION.

Mr. JAMES asked whether there were any symptoms previous to the extraction, or any previous history.

Mr. JOHNSON had had a similar case of a twelve-year-old molar, of which the posterior-buccal root was entirely absorbed, due to a wisdom tooth. The patient came with pain, and at first nothing was seen, but the probe slipped in where the posterior-buccal root should be. He opened up on the buccal side to some extent, but could find nothing very satisfactory. He thought at first he had found the cervical edge of the cavity, and decided to drill into the top of the crown and put in an arsenical dressing, but ultimately decided to remove the tooth. There was no posterior-buccal root at all, and the pulp-chamber had been opened from underneath. The wisdom tooth was standing right in the socket of the tooth. There had been no symptoms of any pain previously.

The CHAIRMAN (Mr. Clayton Woodhouse) considered the absence of pain was very remarkable.

Mr. STORDY said the four molars were all in a very bad state. No definite history of pain was obtained. The tooth was taken out about five years ago, and unfortunately the X-rays were not used, as they might have given some information as to the condition of the roots of the second temporary molar.

(1) Three Cases of Torus Palatinus. (2) Skull from a Barrow showing somewhat Symmetrical Mandibular Exostoses.

By W. DE C. PRIDEAUX, L.D.S.

I HAVE recently seen three cases of torus palatinus, and of these I bring models of two. One, from a woman aged 50, is associated with a somewhat narrow palate (fig. 1); in this respect differing from those illustrated in Mr. Rickman Godlee's paper[1] of February, 1909 (Surgical Section of this Society) and reproduced in Mr. Colyer's recently published "Dental Surgery and Pathology," although in its circumscribed character and roundness it is somewhat similar to an example shown therein, taken from a model lent by Mr. William Hern.

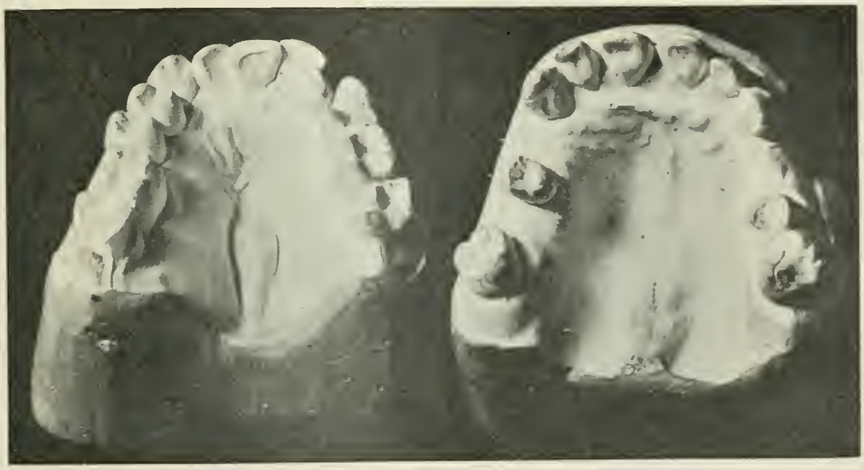


FIG. 1.

Narrow torus and palate, woman, aged 50; English.

FIG. 2.

Long and wide torus, woman, aged 60; wears denture; English.

The two others, situate in flat and massive palates, and extending in the median line fairly uniformly the length of palate, are similar and from women aged respectively 27 and 60. I show the latter (fig. 2); it was brought to my notice by a patient complaining that her plate (of narrow gold) caused a soreness. This, at first, I thought showed present growth, but I am inclined to believe that the plate, worn for many years, had shifted back somewhat, because my slight easing some six months ago has been sufficient. Judging from its margin there is no tendency to encroachment, and my patient remains perfectly comfortable.

---

[1] *Proceedings*, 1909, ii (Surg. Sect.), p. 175.

The mandible I show (fig. 3), with somewhat symmetrical exostoses, from a round Dorset barrow of perhaps *c.* 1500 B.C. These growths are on the lingual aspect of the alveolus and in the premolar region; if, as Mr. F. Coleman hints in his paper on "Exostoses of the Mandible" (Odontological Section, November, 1909),[1] they may usually be found associated with well-developed and massive jaws, this specimen, showing such evidences both in bone and teeth, would certainly favour the view that torus mandibularis is in a measure an overgrowth.

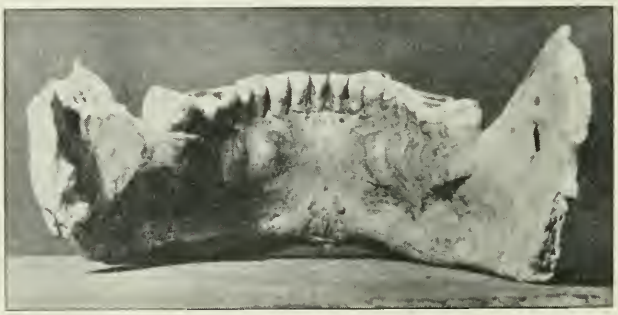


FIG. 3.

Prehistoric mandible having symmetrical exostoses.

Mr. HOWARD MUMMERY said that one of the models shown by Mr. Godlee was that of a lady who had been under observation for about ten years; during this period no change in the growth had been noticed. A model, however, taken a few years later, showed a distinct change in shape and size. This would indicate that it was an exostosis and not a torus palatinus.

---

[1] *Proceedings*, 1910, iii, p. 22.

Model of a Maxilla with Exostoses.

By P. J. PROUD, L.D.S.

THE man was a patient between 60 and 70 years of age, a very gouty subject. He could give very little history of the condition except that it had been slowly getting larger and gave him no trouble.

DISCUSSION.

The CHAIRMAN supposed every dental surgeon had seen cases of the kind in practice. He could remember an old gentleman of 92 whose mouth had the most extraordinary exostoses both in the upper and lower jaw, the ridges standing out to the thickness of a quarter to half an inch all round.

Mr. PROUD said the mandible in his patient was quite normal.

Mr. J. F. COLYER said that, looking at the model shown by Mr. Proud, he personally would think the thickening of the external alveolar process was nothing more nor less than a normal reaction of the tissues to toxins in the periodontal membrane.

Mr. PROUD said he could not say it was a true pyorrhœa case, although there might be some periodontal trouble. There was no excessive inflammation round the gum; in fact, anyone seeing the patient would say he had an excellent set of teeth, especially in the lower jaw. All the teeth, including the third molars, were standing perfectly firm, and there was only one cavity in the mouth.

Mr. COLYER said that possibly he differed from Mr. Proud as to what he called a case of periodontal disease. It was not necessary for pus to accompany periodontal disease. Cases in which there was a reactionary condition in the alveolar process were cases of a chronic character which one was inclined to consider gouty. He was firmly of opinion that Mr. Proud's patient had periodontal disease.

Mr. JAMES agreed with Mr. Colyer. When he saw the model he thought it looked like a case of periodontal infection, and rather typical of a condition seen frequently when the case was one of a very chronic character.

A Case of Multiple Supernumerary Teeth preceding a Central Incisor.

By A. A. H. JOHNSON, L.D.S.

THE case I wish to bring before you to-night is one which I think you will agree with me is a most unusual and interesting one. In dealing with it I propose first to give you the clinical history; (2) to read the report that Mr. Hopewell-Smith has kindly sent me of the examination he has made of the specimens; (3) similar cases previously reported; (4) to suggest some points on which I should like to hear your opinion.

CLINICAL HISTORY.

In July, 1908, the patient, a boy aged 8½, was brought to me because the right upper central incisor had not erupted, although the corresponding left tooth had been in place for a long time. There was nothing in the history to suggest violence or any other cause for the delay. The temporary tooth was present and loose, and I could feel a thickening of the alveolus, which I took to be the permanent tooth. I removed the temporary tooth.

In January, 1909, when I next saw the boy, I was able to feel the same fullness in the position one would expect the unerupted permanent tooth to occupy, but the condition did not appear to have materially altered since the previous visit. I advised patience.

September, 1909: The boy's parents were getting much worried as the boy was nearly 10 years old and the tooth had not put in an appearance. The condition showed no alteration and felt as before. I advised that radiographs should be taken. The radiographs showed the permanent central incisor unerupted, but in good position and surrounded by alveolus, the only differences between it and the left upper central being: (*a*) Its not having erupted; (*b*) its apex was not complete, while the apex of the left central was fully formed; (*c*) there was an irregular mass of something of a similar character to tooth-substance lying in contact with the cutting edge and also on its lingual and labial aspects. As it was very doubtful with what we were dealing, I advised opening down and seeing exactly what it was (fig. 1).

The boy was anæsthetized with chloroform on October 9, 1909, and I made a vertical incision down to the bone from high up in the sulcus to the ridge of the alveolus, and then a similar transverse incision along the edge of the gum. With a director I was now able to reflect the two flaps so formed, together with a piece of alveolus, thus exposing the mass. I then found the six denticles I now show arranged so as to form a cap over the cutting edge of the permanent tooth, and quite separate from the sac of that tooth. They were lying on the lingual, labial, and distal side of the tooth, but not the mesial side. After all had been removed the flaps were replaced and ligatured with horsehair sutures. After this I advised waiting for the tooth to erupt.

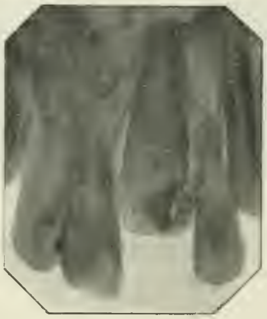


Fig. 1.

The unerupted incisor is seen imbedded in the maxilla, and at the cutting edge a dark, irregular mass which constituted the six denticles.

I did not see the patient again until September, 1910, when I found the condition apparently unchanged, and so had more radiographs taken. If you compare these two sets of radiographs I think you will agree that the tooth has moved down slightly, but the apex is in the same condition as before.

I have seen the boy again this last week, but there is no further change yet.

Mr. Hopewell-Smith's Report.

"Recently Mr. A. H. Johnson submitted to me for examination and report six denticles of varying size and shape, which he has removed from the incisor region of the right maxilla of a boy aged nearly 10 years. The size and weight of each have been carefully ascertained and, as is seen in the photograph, collectively they exhibit singular gradations in their dimensions, and, individually, they show a tendency to the possession of a common pattern, namely, that of a truncated cone (fig. 2). These particulars are as follows:—

"It is difficult to describe the gross anatomy of each denticle; roughly they remind one of the partially developed crowns of hypoplastic incisors or canines of the deciduous series. In the two largest a constricting ring occupies the equatorial region. In the four largest, at the bases of the crowns, cup-shaped depressions exist and appear to have been filled in the fresh state with soft tissue, probably the dentinal papilla of each tooth-germ.

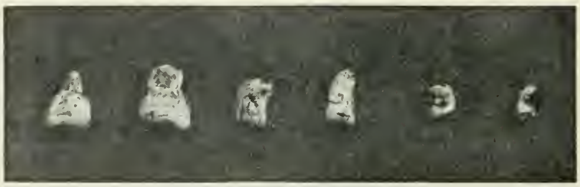


Fig. 2.

The six denticles mentioned and described in the text.

		Weight.	Measures.
(1)	Largest	0·152 grm.	6·7 mm. in length 5·5 ,, in width at base
(2)	Next	0·117 ,,	6·0 ,, in length 5·0 ,, at base
(3)	,,	0·07 ,,	5·0 ,, in length 3·5 ,, at widest part
(4)	,,	0·05 ,,	5·5 ,, in length 3·0 ,, at widest part
(5)	,,	0·02 ,,	2·8 ,, in length 2·5 ,, at widest part
(6)	Smallest	0·015 ,,	4·0 ,, in extreme length 2·25 ,, extreme width

"Sections have not been made because of breaking the morphological sequence of the specimens and because the microscopical examination would throw no light on the method of their formation and pathological significance. There is no doubt, however, that enamel, and also dentine, are present, and cementum is absent.

"It is most likely they represent six aborted tooth-germs, and they may be either the contents of a compound follicular odontome, a congeries of supernumerary tooth-germs or an imperfect attempt to give evidence of the rare occurrence of polyphyodontism in man. Their several positions with regard to the ' Zahnleiste ' in the original instance would be the only guide as to determining the latter condition.

" Similar cases have been reported from time to time by Tellander, Windle and Humphreys, and Mathias, and this is of exceptional interest on account of the clinical history which Mr. Johnson is able to supply."

SIMILAR CASES REPORTED.

There have been, as far as I can find, only three cases of an exactly similar nature reported previously.

The first by Dr. E. C. Kirk, the report of which case appears in the *Dental Cosmos* for 1898.[1] The chief points in the report are as follows : The specimen was from the jaw of an adult and was procured from the post-mortem room, and consequently there is no previous clinical history. An examination of the reproductions of the specimen shows that the tooth, a left upper central incisor, had remained unerupted through life, as had also the 13 satellites grouped round the cutting edge of it. Other points of interest that have struck me with regard to it are that judging from the photographs the apex of the tooth remained incomplete and thickened, and also that there was no great swelling or cystic collection of fluid present.

The other cases are those of Mr. Huey and Mr. Clack, of neither of which have I been able to get any detailed report.

Judging from these cases it seems to me that they may form a distinct class of compound follicular odontomes differing in that they are separated from the involved unerupted tooth, and also in that none have had any marked swelling in connection with them, whereas swelling is a marked feature in a typical compound follicular odontome.

The following are some points of interest on which particularly I should like to hear the opinion of members :—

(*a*) *The Apex of the Tooth.*—In the radiographs the apex has a peculiar appearance ; instead of having a clear-cut edge as is usual it looks blurred and thickened almost as though there was a woolly ring around the apex. What is the significance of this ?

(*b*) What is the reason of the delayed eruption ? Was the tooth held back by the denticles ? Is it due to the faulty apex or to a general disorganized condition of the whole mass.

(*c*) Is the whole mass, tooth and denticles, to be looked on as a compound follicular odontome ?

---

[1] *Dental Cosmos*, Philad., 1898, xl, p. 281.

(*d*) What germ or germs do these denticles represent? And in this connexion I might remind you of the positions occupied by the denticles. They were lying lingually, labially and distally to the permanent tooth, and meeting to form a cap over its cutting edge.

(*e*) I should like to know what the opinion of members is as to the likelihood of the tooth erupting by itself and becoming a useful member.

(*f*) Also, should it not put in an appearance in a reasonable time, what course would members suggest for treatment?

DISCUSSION.

Mr. HOPEWELL-SMITH said Mr. Johnson had been very fortunate in meeting with such an interesting case, and he was happy to be associated with him in putting it on record, although his share of the work had been extremely small. Several remarkable things struck him—the gradation in size of the tooth rudiments, the close resemblance that the larger ones bore to the normal cusps of teeth during development, and the fact that one denticle was situated on the labial surface of the unerupted incisor. They were not the ill-developed masses of hard tissue which had been described by some authors in similar cases, and there was no evidence of dichotomy of tooth-germs or gemination. The condition, in his opinion, might be either a compound follicular odontome, or an example of atavism, or an involution of the "Zahnleiste," in the mid-line of the maxillæ. Against its being a follicular odontome it might be argued that an odontome connected with a first maxillary incisor was very rare indeed, and that usually the denticles found in compound specimens of the species were shapeless masses of enamel and dentine, as seen in specimens in the Royal College of Surgeons and other Museums. Moreover, the usual signs did not point to its being a tumour of that kind.

The condition might be regarded as an example of atavism, and a recurrence to a form of polyphyodontism. It was curious that one rudimentary body should have been found on the labial side of the unerupted incisor, and that the others were found on the lingual side. The deciduous tooth was shed. Some observers, like Kükenthal, Röse, and Marett Tims, would, he believed, consider that there were present, or before the deciduous tooth was shed, evidences of the existence of a pre-milk, milk, permanent, and post-permanent dentition, the pre-milk tooth-germ being that on the labial side, the other denticles belonging to the post-permanent group. Supernumerary teeth in the incisor region occurred usually on the lingual side of the permanent series, in the molar region on the buccal side. If that theory of polyphyodontism could be applied to the present case it was an example of atavism, a reminder of the supposed reptilian origin of man. He knew of no very great argument against it, except that, perhaps, it would be an extreme instance; and that as there were six denticles, five of them were merely vestigial remains of the tooth-band extending deeply inwards beyond the post-permanent tooth-germ. Or there might have been an involution of the tooth-bearing portion of the embryological nasofrontal process, the central part of which went to form the endognathion, when as a result of a rather elongated "Zahnleiste" several rudimentary teeth were formed. Against that view there was evidence that it was unilateral, whereas, if it were true, it would be expected to be symmetrical.

Bearing upon Mr. Johnson's communication was a case of his own which he had recently seen. A man, aged 28, had, in the superior intercanine region, implanted in the premaxillary bones seven teeth—all fully erupted and the majority well formed. The plaster casts and photographs showed the actual condition. From an examination of the jaw it was obvious that the intercanine region was exceedingly broad; it measured no less than 34 mm., while the mandibular intercanine region measured 18 mm., about normal. The normal adult measurements were about 28 mm. in the upper and 18 mm. in the lower jaw. It was obvious that both the endo- and the exo-gnathion were broader on the right than on the left side, accommodating four teeth to the other three. It was very difficult to interpret the condition and correctly name the teeth. Morphologically they would appear to be the right first and second incisors, a third incisor (the precanine of Sir William Turner) and a supernumerary first incisor, all in the right premaxilla; and in the left, the left first and second incisors, and a supernumerary first incisor. It would seem as if Nature having a wider space to fill than usual had filled up the gap by supplying the patient with an extraordinary number of teeth. Whether it was correct to call them supernumerary teeth he would not care to say. Rosenberg, who at the end of the last century collected an enormous amount of literature on the subject, and inspected large collections of skulls, plaster casts of the mouth, &c., considered, in spite of the opinions of Virchow and Busch, that every supernumerary tooth covered with enamel was atavistic. Rosenberg's reading of the condition presented by his (the speaker's) patient would be Incisor 1, having on its distal surface Incisor 2, and on its mesial surface Incisor *alpha*, and Incisor *gamma* on the distal surface of Incisor 2, on the right side; and on the left, Incisor 1 and Incisor 2, with Incisor *beta* in between. The formula would read:—

$$ {}^{\gamma}\mathrm{I},\ {}^{2}\mathrm{I},\ {}^{1}\mathrm{I},\ {}^{\alpha}\mathrm{I},\ \mathrm{I}^{1},\ \mathrm{I}^{\beta},\ \mathrm{I}^{2}. $$

The difficulties of interpretation surrounding the whole matter were, however, very great.

The CHAIRMAN said that if the central in Mr. Johnson's case did not grow down very soon it had much better be removed, because the mouth was distinctly crowded.

Mr. C. ROBBINS said there was a practical everyday way of looking at the matter, whether there was an explanation of the intrafollicles or not. The great point with dentists and parents was whether the retarded tooth would come into position, and in what way could it be assisted to do so. He had two cases of marked retarded eruption of the right central, one in a boy and one in a girl. After using the X-rays to localize and verify the diagnosis, he had felt it incumbent upon him to make a little temporary piece. In one case the opening was filled with an imitation white vulcanite tooth because there was not thickness enough for a porcelain tooth, and in the other he used an ordinary temporary plate with a central incisor. He did that for three reasons: First, because he thought it better to keep the space well apart to prevent the teeth crowding together; secondly, because it was well to have as good an appearance as possible during the period of waiting; and, thirdly, he thought the amount of pressure of any apparatus on the gum helped slightly towards the eruption of the retarded tooth. The age of the girl was 12, and the age of the boy 14. The boy's case was complicated by the non-eruption of the four bicuspids. He had extended the plate further back, and as the bicuspids came down he made room for them in the plate.

Mr. STANLEY MUMMERY had had a somewhat similar case in his own practice, a girl who had the left upper central unerupted, in its place being two teeth lying side by side, the labial aspect of the one and the lingual aspect of the other in contact. It was quite impossible to decide which of the teeth was a supernumerary tooth, and Mr. Clark took some excellent skiagrams which eventually showed which was a supernumerary and which the permanent central. The supernumerary was extracted, and the edge of the other tooth caught by a band. There was about $\frac{1}{16}$ in. projecting below the gum, and that was caught by two hook wires and twisted until it was straight. A narrow band of gold was then made and pressed beneath the gum so as to embrace the tooth, with a little tag at the back. A vulcanite plate was then made with a strong pianoforte wire which bore on the tag, and in a fortnight the tooth was in place. After wearing a retaining plate for a few months, it looked as though the tooth had erupted in the normal manner with the other teeth.

Mr. BOYTON showed two models of a case of supernumerary teeth in a patient aged about 18. In answer to a question by the Chairman, he said that no teeth had erupted since.

Mr. BALDING had been going through a quantity of old models, with the idea of throwing some away, and had come across some he thought might be of interest; although they would not be of great value scientifically, owing to the want of history. The first was a model of a maxilla, showing two supernumerary central incisors. The supernumeraries were standing inwards on the palatal surface, and the two normal incisors had their lingual surfaces facing one another. The case occurred in his father's practice about twenty-five years ago, and there was no history that could be given. He also showed a series of models of another maxilla which, though imperfect, were of some interest because the case seemed very much like the one Mr. Mummery had just mentioned, and also had certain points of similarity to Mr. Hopewell-Smith's case. The models were from a boy, the first being taken at about the age of 9. On the right side there was apparently a supernumerary central incisor geminated, and on the left side there were two central incisors separate. It was before the days of the X-rays, and the geminated teeth were extracted, and after some time the other two central incisors erupted, with the labial surfaces facing one another. Each tooth had a quarter turn, so that the mesio-distal axes of the teeth were in the antero-posterior line. After they had erupted, they were regulated as shown by the models.

Mr. STURRIDGE said it was almost impossible to advocate treatment in Mr. Johnson's case without seeing models of the lower jaw. One of the models seemed to show quite distinctly a lack of space where the central incisor had come down, but in a former model there was plenty of room. If the articulation would allow for expansion of the side teeth, and slightly pushing the other central incisor, he thought there would be enough room for the central to come down. It might erupt almost immediately in consequence of the irritation set up by moving the adjoining teeth, and perhaps eruption might be further aided by lancing the gum over it.

Mr. LEWIN PAYNE said it was perfectly obvious that the occurrence of supernumerary tooth-germs in the front of the mouth was very common, and their presence might be accounted for by the condition Mr. Johnson had suggested, but the formation and maldevelopment of supernumerary tooth germs had been usually attributed to mechanical or other interference with the normal growth of the tooth-band, or the tooth-germs. He thought the case in question was one of a compound follicular odontome, though the relationship between a compound follicular odontome and multiple supernumerary teeth might be a close one. In the case mentioned in the paper, the teeth were malformed, grouped together, and probably encapsuled, and therefore should be classified rather as a compound follicular odontome than as a group of ordinary simple supernumerary teeth.

Mr. JAMES asked whether there was any cystic condition present. Compound follicular odontomes had been described as being of a cystic character, but it seemed very difficult to get a history that they were really cystic. The history of recorded cases in the old days was very imperfect. There was a specimen in the Middlesex Hospital Museum where a large number of denticles were fused together, many of them situated in the tooth mass, but as this specimen had never been cut it was not possible to say whether it was chiefly composed of bone or cementum. The denticles, however, could be clearly seen. One interesting point in this case was that these supernumerary teeth or compound follicular odontomes preceded one of the normal teeth. Mr. Gabell, Mr. Lewin Payne and himself had been working for a long time at odontomes in connexion with the British Dental Association Committee, and they found that a large number of odontomes preceded a normal tooth. An odontome was removed, and a tooth was found situated underneath. Supernumerary teeth did not always precede fully developed teeth. With regard to the model shown by Mr. Boyton, he would like to know if there was any history of syphilis.

Mr. BOYTON said the patient's general appearance suggested syphilis, but he did not care to put the question.

Mr. DOWSETT wished to know why the form of growth, supernumerary tooth or odontome, should be classified as a compound follicular odontome. In the old nomenclature such an odontome as the one under discussion that evening would have been called a composite odontome. According to Mr. Bland Sutton's definition a compound follicular odontome was one where the follicle had overgrown and calcified in a sporadic manner, calcifying into cementum and osseous tissue, not dentine and still less enamel.

Mr. JOHNSON asked whether Mr. Robbins had any knowledge of what the apices of the delayed teeth were like previous to their eruption. If the apices of the teeth were unformed, as was the apex of the tooth in the case brought forward in the paper, it would bring the cases more into line than they otherwise would be. With regard to Mr. Mummery's case he would like to know whether the tooth was tested to see whether the pulp was alive or dead after it had been forcibly erupted. It struck him as possible that the pulp might have suffered rather severely by being erupted in so short a time. If the pulp had died and there was no apex it was well known what that meant. With regard to Mr. Sturridge's remark as to the lack of room, he did not see the boy for twelve months after the denticles had been removed, and therefore was unable to put in an apparatus to keep the space open as he had intended. He was now making a fixed apparatus that the boy could not take out, and he hoped in that way to get the space back again. He did not think the central was wedged, the radiograph showing that the central and lateral were overlapping. That did not alter the fact that there was not sufficient room in the arch at present, but he was hoping to obtain it. With regard to the question of cystic fluid, he believed there was a small amount of fluid round the denticles. In removing one of the denticles he had a feeling of the puncturing of a sac and the escape of fluid, but the amount was so small that it was difficult to say anything as to its character.

Odontological Section.

March 27, 1911.

Mr. A. Clayton Woodhouse, Vice-President of the Section, in the Chair.

A short Preliminary Note on the Distribution of the Nerve-fibres of the Dental Pulp.

By J. Howard Mummery, M.R.C.S., L.D.S.

I come before you to-night somewhat in fear and trembling, not that I wish to make any dogmatic statements, but to take you into my confidence and invite your criticism before proceeding further with my work. The communication which I propose to make this evening is on the much vexed question of the distribution of the ultimate nerve-fibrils of the dental pulp. It is more in the nature of a preliminary note than a paper, as I am now working on some fresh material, and hope to be able to place the subject before you in a more complete form at a later date.

I have lately resumed some work which I commenced nearly twenty years ago, and in examining some slides found one or two which seem to show much more distinctly than they did the passage of the nerve-fibrillæ to the dentine. These specimens were prepared by the iron and tannin process, and, unlike many microscopical preparations, had improved by time. I can only suppose that, this being an impregnation process and not a stain, the action of the tannin on the iron in the tissues had become more complete.

To show how far I had carried the investigation in former years, I may perhaps be allowed to quote from the 1904 edition of Tomes's "Dental Anatomy." The author says (p. 63) :—

> "Mr. Mummery's iron preparations clearly show that fine fibres, traceable into connexion with indisputable nerve-bundles deeper into the pulp, do run out straight towards the dentine and right up to it as though they entered it."

Again (p. 98) :—

> "Mr. Mummery, using as a stain an iron salt followed by tannin, has succeeded in showing that from the nerve-bundles which course along more or less parallel to the surface of the pulp very fine fibres are given off, which run towards the dentine, and I have gone over these preparations with him. Whilst there are appearances suggestive of their entering it, we could arrive at no certain conclusion; such is the difficulty of the investigation where very delicate structures are in relation with a calcified material."

Since this was written the late Herr Morgenstern and Dr. Römer have carried out further investigations on the pulp nerves, and both, although differing in some details in the conclusions they arrived at, describe the ultimate nerve-fibrils as penetrating the dentine from the pulp. I believe that neither of these observers have published any photographs, but have illustrated their views by drawings, and their observations do not appear to have been corroborated by others using the same methods.

I have confined myself entirely to photographs, and, allowing that the fibres I shall show in the lantern slides *are* the nerve-fibrillæ (and certainly their connexion with the nerve plexus beneath the odontoblasts seems fairly well shown), these fibrillæ pass into the tubules of the dentine, and appear to accompany the fibril. On their ultimate terminations in the hard tissue my specimens shed no light, although the German observers describe them as terminating in the spindle-shaped enlargements at the junction of the enamel and dentine. Unless, however, the nerve-fibrils can be actually traced up the tubes into these enlargements, this must, I think, be looked upon as purely theoretical.

As is well known, the medullated nerve-fibres enter the tooth pulp at the apical foramen, and in most cases accompany the blood-vessels in their passage up the pulp. At their terminations they break up into a fine brush, losing their sheath, and are distributed in the pulp tissue as delicate ultimate fibrils, which, at all events under the action of reagents, are dotted or beaded. They form an intimate plexus beneath the odontoblasts, called the plexus of Raschkow, and fine fibres pass from this plexus around and between the odontoblasts to the dentine, where, as I shall hope to show you immediately, they enter the tubes with the dentinal fibril. They seem to be very unevenly distributed on the surface of the pulp, being much more abundant, as pointed out by Morgenstern, towards the cornua of the pulp. I have not been able to trace any of these fibrils directly into the odontoblasts; nor, I believe, has Mr. Hopewell-Smith, although I agree with him that they closely envelope and embrace these cells.

I had a considerable difficulty in obtaining satisfactory photographs, as a small diaphragm has to be used in photographing these impregnated specimens, and the colour, which is blackish, would not, I found somewhat to my surprise, give me a well-defined photograph. It is really a purplish-blue, and by interposing a filter of copper acetate solution and an optically worked picric acid glass screen, I procured very sharp definition on the plates. Direct light was used from an acetylene burner, using the edge of the flame.

I have made use of various processes in staining sections for this investigation, but have found none so satisfactory as the iron and tannin. The chromic acid method employed originally for this purpose by Boll showed an immense number of fibres passing towards the dentine, but I could not trace them further, and the method is open to the objection that the deposits formed in the dentine are very misleading. The same objection applies to the Golgi method. Those in which I used the Weigert stain gave the most perfect definition of the structure of the medullated fibres, but the fine fibrillæ were far less perfectly defined. I do not think the iron and tannin process is open to these objections, at all events to the same extent; the fine fibres saturated with the iron salt are blackened by the tannin, and any dirt or deposit from imperfect washing or clearing can be easily detected.

I will now show the slides upon the screen, that you may be able to form an opinion as to whether or no I am justified in my conclusions.

DISCUSSION.

Mr. GABELL recognized the extreme difficulty of producing photographic work under such a high power of magnification, and asked whether Mr. Mummery, with his finger on the fine adjustment, was able to trace any short, fine dotted filaments from the dentine back to the plexus of Raschkow. It was a thing that was almost impossible to show in a photograph.

Mr. SIDNEY SPOKES asked whether some of the photographs shown that evening were from specimens prepared by Mr. Mummery twenty years ago, or new specimens made for the present purpose. He remembered having the pleasure of seeing Mr. Mummery's former specimens and trying to focus the nerves with the fine adjustment and trace them, as Mr. Gabell had suggested, and Mr. Mummery was good enough to allow him to examine them carefully at the time. Although he was able to focus right up to the dentine it was never possible to see the appearance Mr. Mummery had shown in the specimens that evening. Therefore, if they were the old specimens, the very interesting question was raised as to what change had taken place owing to the reagents that were used in preparing the specimens.

Mr. F. J. BENNETT congratulated Mr. Mummery on his excellent photographs and also upon his perseverance on taking up the subject again after twenty years. There was no doubt that in some microscopical specimens the reagents seemed to have a preservative effect upon the tissues and also the power of selection. We knew very little about the chemistry of stains and dyes used in microscopical work, but apparently Professor McCullum, of Toronto, and others were finding that a great deal could be learned both about histological structures and chemical action by studying the stains in specimens. Mr. Mummery wisely declined to enter into any discussion of the functions of the nerves in those cases. He hoped when Mr. Mummery said he was continuing his investigations he did not mean that another twenty years would elapse before the information was brought before the Section.

Mr. HOPEWELL-SMITH thought it was somewhat of an impertinence for him to criticize the paper, as he was not present at the beginning of the meeting, although he saw most of the lantern slides. He had some little difficulty in accepting Mr. Howard Mummery's beliefs, partly on anatomical grounds—he could not quite understand a non-medullated nerve-fibril running in a calcified tube by itself, as such a condition did not obtain in the bones—and partly on clinical grounds. He thought that if the dentine was enervated caries would always be an extremely painful process, even at the commencement, which it was not. In preparing a cavity the deeper the excavation was made very often the less sensation was found. If there were non-medullated fibrils in the dentinal tubules he thought there would be more pain as the cavity became deeper and approached the pulp. From the pathological point of view he would expect that the formation of adventitious dentine or secondary dentine in the pulp would be associated with pain if there were nerves in the tubes. The subject was of peculiar interest to him, because the very first paper he ever wrote on dental subjects twenty-two years ago was associated with the nervous system of the pulp, and he had followed with much interest the writings on the subject both in France and Germany. He thought the old ideas of Waldeyer as to the odontoblasts forming the dentine matrix were quite obsolete, and he was pleased to find that Walkhoff and Korff and Studnicka had agreed lately that the odontoblasts did not form the dentine matrix, but probably the sheaths of Neumann. He still believed that the odontoblasts were not nerve-fibrils but sensation transmitters, and that the dentinal tubules contained nothing but their peripheral protoplasmic processes bathed in serum, and that the non-medullated nerve-fibres arborized around the odontoblasts, and did not penetrate the hard parts of the teeth. It was not so in fishes and reptiles, and could not be in mammals. The great objection to that idea was that the odontoblasts were mesoblastic in origin; but he might say that doubts were beginning to be expressed that the three germ layer theory might not be correct. Von Klaatsch had raised that point, and believed that it was not necessary for one to hold rigidly to the idea that the nervous system emanated entirely from the epiblast. The botanists had, two years ago, relinquished that theory with regard to the structure and growth of the cells of plants. He was sure the Section was much indebted to Mr. Mummery, whose work on dentine was so well known, for giving the Section his latest researches on the subject, and he was very sorry he could not altogether agree with his findings.

Mr. H. BALDWIN said that, as Mr. Hopewell-Smith had based two of his objections on physiological grounds, he was bound to confess himself that he had always considered the mere fact that the dentine was a highly sensitive structure was a proof that nerve-fibres of some sort must enter the dentine, and, of course, the only way for them to enter the dentine would be for them to travel in the dentinal tubes. He did not think there was any instance in such a highly developed structure as the human organism of inherent sensation being imparted to any structure without the presence of nerve-fibrils. Clinical experience showed positively that dentine was inherently a highly sensitive structure, and it would naturally be expected that the outer layers of the dentine would be the most sensitive if they were the actual termination of the nerve-fibrils. Mr. Mummery's photographs certainly showed very fine black fibres running along the tubules, and he would like to ask Mr. Mummery whether it was certain that those black fibres were necessarily nerve-fibres. If by the method of staining only nerve-fibres were stained, then he thought the photographs proved conclusively and absolutely that the fibres entered the dentinal tubules. In his opinion, Mr. Mummery's researches had brought forward the proof he had been always looking for, that the nerve-fibres did actually penetrate the dentine; without admitting the presence of nerve-fibrils, the only way of explaining the high sensitiveness of the dentine would be by saying that the dentinal fibres contained nerve material undifferentiated, or something of that sort, which he thought would be absurd.

Mr. LEWIN PAYNE asked whether the tubes were penetrated by these nerve-fibres from all parts of the pulp cavity or only in certain limited areas: this was a point of some importance in regard to the sensitiveness of the dentine. The great value of ascertaining the exact distribution of the nerves of the pulp was apparent, for one could not properly allocate the functions of the odontoblast cells, or other cells of the pulp, until the distribution of the nerves had been definitely determined.

Mr. J. HOWARD MUMMERY, in reply, said, with regard to tracing the fibres from the plexus of Raschkow to the dentine, he had examined a number of specimens, and certainly considered he had done that in many of them. It was exceedingly difficult with high powers to trace a single fibre for any great distance, because of the difficulty of focussing. He showed them to Mr. Tomes, who examined them very carefully, and was of opinion that the fibres did go from the plexus in between the odontoblasts to the dentine. What he was trying to prove was that these fibres, that did go from the plexus of Raschkow to the surface of the dentine, actually entered the tubes. With regard to the specimens, they were the same as those he had twenty years ago. On going through them again, he found distinct changes had taken place—changes he could not understand, except that they might be due to the action of the tannin upon the iron, which action had continued. As to the method employed; after cutting the sections, subjecting them to a solution of 2 per cent. of perchloride of iron, washing them thoroughly, and then using a 2 per cent. solution of tannin, by watching the action of the tannin upon the iron in the specimens under the microscope one could see when they were sufficiently blackened. If they were allowed to remain in the tannin solution too long, the whole specimen was blackened and made difficult of recognition. The weaker the solution the better the result. A long action of weak solutions would produce a much better effect than using too strong a solution at the beginning. He had been disappointed with Mr. Hopewell-Smith's remarks, and was sorry he could not agree with his view that the odontoblast cells were nerve-endings. He thought there were very many reasons against it, but he had purposely avoided all theoretical considerations that evening. He had simply shown on the screen certain photographs of certain preparations, and asked whether they were nerve-fibres or not. Mr. Hopewell-Smith did not answer that question, but simply said he thought the odontoblasts were nerve-end organs, and clinical experience showed there could be no nerves in the tubes of the dentine. Mr. Baldwin had shown from clinical experience that they ought to be there. Really Mr. Hopewell-Smith's objection was more on theoretical grounds than practical, and he should like still to know what was his opinion on the fibres. In reply to Mr. Lewin Payne's remark, he thought in the paper he had stated they were much more frequently seen, and in much greater abundance, at the cornua of the pulp. He had prepared a number of specimens with iron and tannin, and ground them down by the Weil process, but this method was of no use for fine detail, because it was impossible to get the sections thin enough to follow up the fine nerve-fibres. That led him to continue his investigations on the microtome sections, because he was quite certain that those fibres which were seen in such abundance, and which had been described by Ball as seen in his chromic acid preparations, could not be the fibrils of the odontoblasts. He did not think the nerve-fibres were given off at all equally; they were given off much more abundantly towards the cornua than anywhere else, and spread out in the dentine. It was a difficult point to decide, because he could not see these fibres in sections of the dentine away from the pulp, but could only trace them for a short distance into the tubules. All he put forward on this occasion was that these were appearances he had never seen before, and they seemed to him to agree with the views of other observers, who said that the nerve-fibres did enter the dentine.

The Comparative Elasticity and Internal Structure of Cast and Rolled Gold Plate.

By H. J. Morris, L.D.S., and Andrew McWilliam, A.R.S.M., M.Met.[1]

The introduction of the casting process for dental plates, and the enthusiastic assurances of the makers of casting machines for that purpose, caused us some time ago to ask the question, "Is a cast plate really better than a swaged plate, and what resistance will cast and swaged plates show respectively when subjected to equal stresses?"

Thanks to the generosity of this Section, it was possible to make a small research on this question with the remains of a grant made for a research on the strength of diatoric teeth. After the plates had been selected and prepared—namely, the rolled, the soldered, and the cast plate—we started work on devising and carrying out tests on these several plates with the object of having the two points of view of the dentist and the metallurgist properly represented.

The work done is not commensurate in its scope with the importance of the subject, and the results obtained can only be considered as opening up a new field and clearly showing that there are certain differences in the mechanical properties of these plates which, in connexion with other metals, are considered of great importance. Three pieces of 18-ct. gold were prepared and treated as regards annealing, &c., as they would be in practice; they were reduced to the same superficial dimensions, and at one place, 5 cm. from one end, they were all reduced to the same thickness, to $\frac{1}{1000}$ in. The dimensions were: Length, 6·5 cm.; breadth, 2 cm.; thickness, 0·019 in. No. 1 was ordinary rolled plate, No. 7 Birmingham metal gauge, such as is used for making plates; No. 2 consisted of two laminæ of No. 3 B.M.G. soldered together with Ash's No. 3 solder; No. 3 was cast by Mr. Sims, of Manchester, in a Solbrig press; thus representing three different styles of denture.

Although these plates were all of 18 ct., their exact chemical composition and mode of manufacture are unknown, as is also the composition of Ash's solder. They were studied in three ways:—

---

[1] From the University of Sheffield.

(1) To compare their limits of elasticity.

(2) To discover their comparative resistance to alternating shocks.

(3) To compare their internal structures under the microscope.

It was felt that it would be well to devise a test that would show the comparative behaviour of the plates under some kind of static stress, and also to find out how they stood one of the types of alternating stress test now so much used in connexion with researches on materials intended to occupy important positions in moving structures. The behaviour under bending moments was the static test chosen, owing to the small amount of each material available, and also because it seemed to bear some near relation to the kind of property brought into play when the plates are in actual use.

THE TEST FOR ELASTICITY.

This test was carried out by fixing one end of the plate firmly to a bench, leaving 5 cm. projecting in each case, and then attaching weights in equal increments to the other end and noting the deflection of a recording pointer. This is a simple device which was considered suitable for obtaining preliminary results, and these results, when obtained and plotted, proved to be quite of sufficient importance to be laid before the members. Strictly, the stress should be applied so that the bending moment increases in direct proportion to the weight added, and this is not exactly the case with this simple arrangement, but as the only idea was first to get comparative results these are not vitiated, and the only warning that need be given is that members familiar with stress-strain diagrams should not be anxious about the shape of the higher parts of the curves. These parts, owing to the excessive bending of the test piece, are not accurate, as there the ounces in the scale-pan mean much smaller bending moments than in the lower parts of the curves. There is, however, no abrupt change from this cause in the important parts of the curves.

In the ordinary stress-strain diagram of a tensile test of a steel there is first the region of proportionality, where equal increments of stress, or force applied, give equal increments of strain or effect produced, and the mathematicians tell us that therefore during the whole of this period the metal has been acting as a perfectly elastic body. Whilst this condition exists the stress-strain diagram is a straight line with a greater or less slope as the strain is greater or less in proportion to the stress applied. It will be noted how perfectly the rolled 18-ct. gold plate fulfils this condition up to a load of 43 oz. Allowing for a possible small error, the soldered plate seems to be elastic up to a load of 32 oz., whilst the "elastic limit," as this point is called, is reached for the cast plate at 14 oz. Not only so, but the deformation produced by a given stress, even within the elastic range, is least for the rolled gold, almost the same for the soldered, and very much greater for the cast plate.

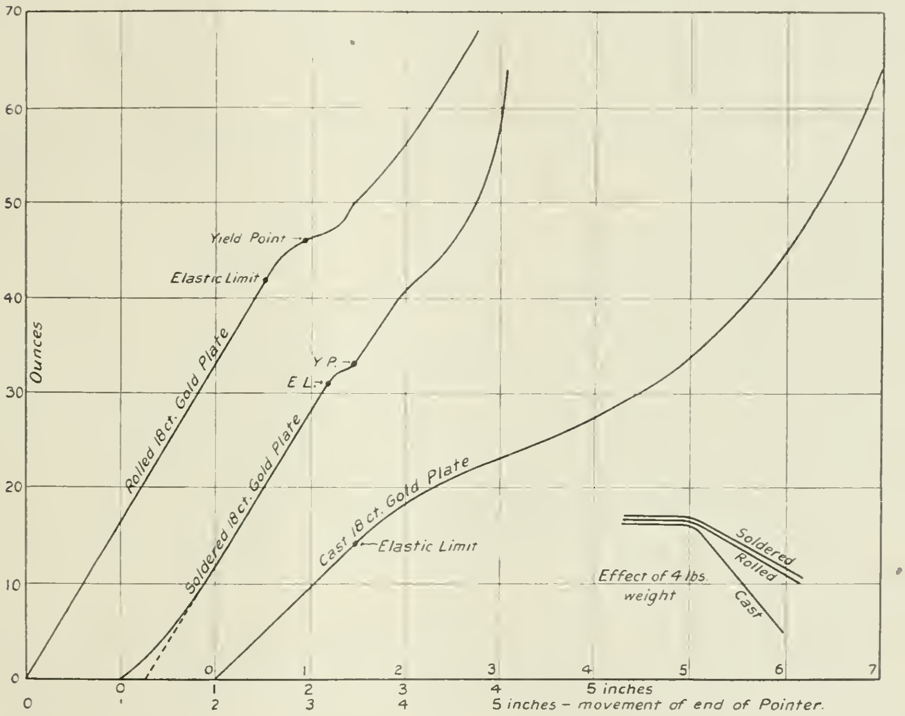


FIG. 1.

Diagram showing results of tests for elasticity.

After the elastic limit is passed there is generally a short range where the strain is slightly more than proportional to the stress and the line becomes slightly curved. Then a point is reached at which the next equal increment of stress produces a sudden increase in the corresponding increment of strain, and this is known as the yield point. The rolled plate shows this very clearly, the soldered one quite distinctly, but the cast plate seems to be giving all the time after its elastic limit is passed. For reasons already given, the portions of the curves beyond the yield points should be ignored, although they are recorded to show the actual results obtained by the rough method employed. When the yield point is passed, however, the metal has broken down so far as use is concerned, and really after the elastic limit, any further stress produces at least a slight permanent set or deformation, so that the curves are good within the limits that it is worth while to consider for present purposes, as the perfection of the straight line within the elastic limit of the rolled-gold plate fairly well demonstrates.

THE DYNAMIC TEST.

The dynamic test chosen was Dr. Arnold's alternating stress test. This test consists of knocking the test piece backwards and forwards a given distance on each side of the vertical and at a known rate in a machine specially designed for the purpose. Here, again, the test pieces ought to be prepared with great care, and the plates *were* prepared to give preliminary indications, but these results also seemed of sufficient importance to record until further, and perhaps more accurate ones, were obtained. The plates were divided longitudinally into two each, making six in all, and were then successively clamped in the alternating stress testing machine. Each strip projected 1½ in. from the top of the die in which they were clamped, to the striking point, and were bent ½ in. on each side of the centre line at each shock (fig. 2).

This test is analogous to what an artificial palate undergoes in the mouth when sunken alveoli have brought about a rearrangement of the stresses (fig. 3).

The first strip of rolled gold took rather more than 2,450 shocks and the second 5,090 before they broke. The first strip of the soldered plate took 6,860 shocks and the second 10,190 before they broke. The first of the cast strips broke after 782 shocks and the second after 120. In showing this brief and feeble resistance to alternating shocks, cast gold is only behaving in the way that one would expect such a cast metal to do, and the result occasioned no surprise. The results show clearly the inferiority of the cast gold plate with regard to this test; but it must always be remembered that the translation of the results of tests into probable behaviour under certain conditions of actual use is an ever-present difficulty in the practice of metallurgy, which demands for its successful application to the satisfying of the world's needs the exercise of the highest skill born of study and experience. The results as between the rolled and soldered plates might be misleading, as the microscope reveals the fact that the plates soldered together are not in absolute metallic contact throughout, in spite of great care in soldering. If those plates happened to be gripped in such a place that the two plates could be bent partly as two thin plates, that is to say, under conditions that would allow of even slight relative movement of the two plates, then the results from the soldered plates might partially approximate those that would be obtained from thinner plates, and the thinner the plate the higher the number of alternations it would endure, all other conditions being equal. Therefore, all that can safely be said about these results is that under this test the rolled plate and the soldered plate yield results very much superior to those given by the cast plate.

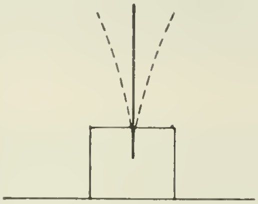


FIG. 2.

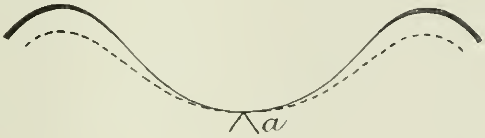


FIG. 3.

Diagram to show how the alternating stress operates in actual practice after alveolar ridges are absorbed. (*a*) represents the central ridge of palate.

The appearances of the fractures of these plates were of much interest. The rolled plate had been heated up as a whole before being cut into test strips, but not to any determined temperature; the other plates had the heat treatment necessary for their manufacture. The plates all broke with a crystalline fracture—quite a common experience under the test, even with plates which would be bent double without a sign of a flaw and would show a fibrous fracture after breaking under a tensile test. The rolled plate showed the finest fracture and the cast plate the coarsest, but the soldered plate was very nearly as coarse in the fracture as the cast plate. As a general rule, if a plate of metal made fine in the structure by cold or hot work is heated to higher and higher temperatures, the tendency is to increase the size of the grain. This does not necessarily mean that when the grain of the rolled plate reaches the same size as that of the cast one it will have the same properties, as some of the beneficial effects of rolling may still remain. It would be wrong to attempt to make final or dogmatic statements from such a small number of experiments, but what has been done is in very general agreement with results obtained in connection with the

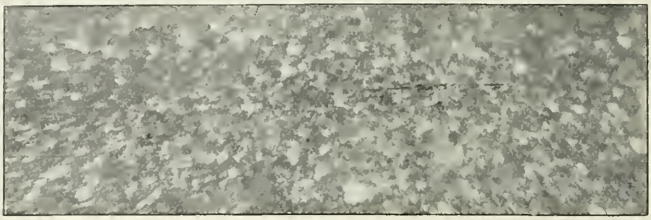


Fig. 4.

Rolled 18-ct. gold plate, transverse section. (× 50.)

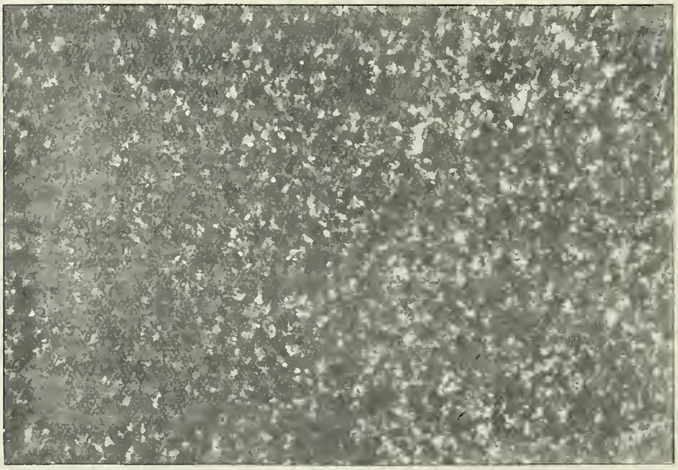


Fig. 5.

Rolled 18-ct. gold plate, parallel to face of plate. (× 50.)

ferrous metals, where steel castings, after being cast with the greatest care to insure practical freedom from flaws, are subjected to an elaborate heat treatment before being sent out for use.

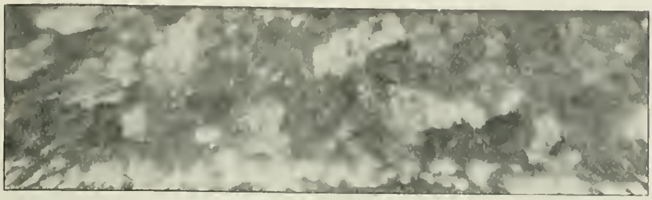


FIG. 6.

Cast 18-ct. gold plate, transverse section. (× 50.)

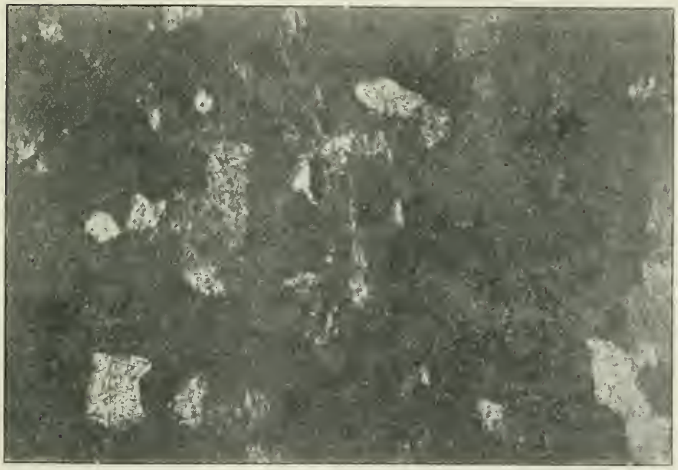


FIG. 7.

Cast 18-ct. gold plate, parallel to face of plate. (× 50.)

THE MICROSTRUCTURES.

For microscopic examination of their structures a small piece was sawn out of each plate and each piece was polished on one face and on one edge. The polishing was done by rubbing the pieces on specially graded emery papers, first 0, then 000, and finally by holding them on a rapidly revolving block covered with Selvyt and dressed with water containing alumina (sold as diamantine) in suspension. The sections were then etched with aqua regia, washed with water, the silver chloride dissolved off with ammonia, washed again with distilled water, and dried either in the air or with alcohol, when they were ready for examination.

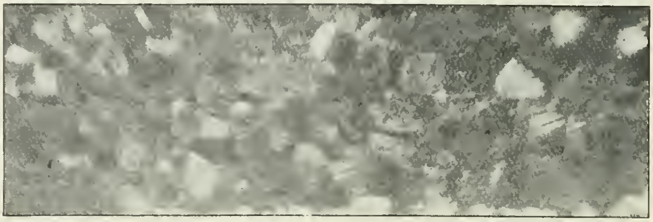


Fig. 8.

Soldered 18-ct. gold plate, transverse section. (× 50.)

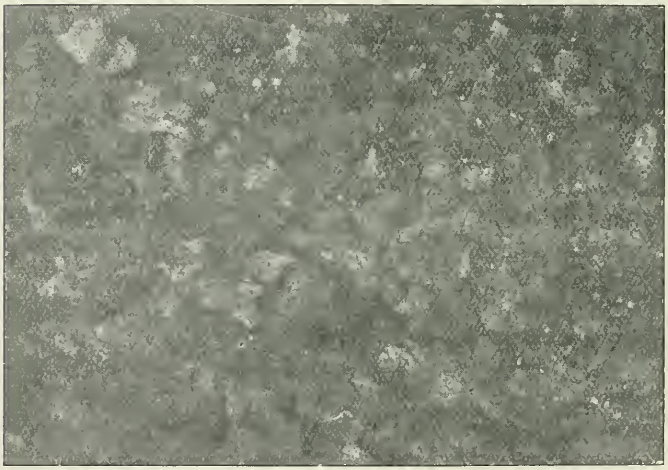


Fig. 9.

Soldered 18-ct. gold plate, parallel to face of plate. (× 50.)

Lantern slides have been prepared merely to illustrate the nature of the microstructures of the various plates, as the sections are available for examination under the microscope. It will be seen both from the transverse sections and from those taken parallel to the surface of the plates, that the rolled plate (figs. 4 and 5) is finely crystalline, the cast one (figs. 6 and 7) very coarsely crystalline, and the soldered plate (figs. 8 and 9) a little finer in the grain than the cast one.

Cast gold shows no exception to the general tendency of cast metals to be porous, and hence a cast plate would probably harbour more germs and rubbish than a swaged one. Whether this porosity can be avoided, and how, we are unable to say, but speaking generally it may be said that the thinner a casting is the greater will be the tendency for it to be porous.

If these experiments were checked and found to be, as we believe they are, in harmony with results obtained in other fields of metallurgical research, we would venture to hint at a lesson to be learned from them. It is this: When you cast a plate, cast it thick enough to be rigid, because if once the alternating stress begins to operate, the life of that plate will be brief and troublesome.

In conclusion, we wish to thank you for your encouragement in this research and your attention to our paper.

DISCUSSION.

Mr. F. J. BENNETT thought the authors were to be congratulated upon their valuable experiments. He was glad to see that the grants in aid of research which were instituted by the Royal Society of Medicine had been taken advantage of. The subject was a highly practical one to dentists. It should not be forgotten that gold was an alloy to begin with, and it might have been interesting to hear what the behaviour of a pure metal was. In the soldered specimen one was practically dealing with a rolled example and a cast example, because when the solder had been melted and had cooled down it was in a crystalline form, somewhat like cast metal, and therefore would share in the evils of cast gold plate in the soldered region. He believed that cast metal was always of a crystalline structure, and when a strain was applied there was a movement of one crystal on the other, a sort of dislocation or fault as it would be termed in geology. He thought the figures and demonstrations the authors had given that evening were sufficiently convincing to show that a cast structure was always, from its very nature, weaker than that of rolled gold.

Mr. PEYTON BALY asked what steps were taken to ensure that the surfaces of the pieces of gold tested were absolutely parallel. In the Royal Mint the gold used for making coins was drawn between two parallel plates because it was said to be impossible to get two parallel surfaces if the gold was run through rollers.

Mr. C. N. PEACOCK had made a good many experiments with cast crown work, but had not had very much experience with plate work. From the paper it would seem that cast plate work was condemned, but he thought there were cases—for instance, a high palate with one or two incisors missing—where the casting device was of much use.

Mr. PERCIVAL CAMPKIN thought the results were what would naturally be expected. For the last eighteen months he had been using the vacuum casting process in a large number of cases, and he had not had a single fracture in the mouth. It was exceptionally useful in very deep plates where it was practically impossible to strike up a plate with any accuracy, or if struck up it had to be chased into very deep crevices and was naturally weakened. Naturally a cast plate must be weaker, but the question was whether it was not strong enough for the purpose, having regard to the greater accuracy of fit obtained—an accuracy which would compensate perhaps for some weakness. Testing sections cut from completed plates, the one made by swaging the other by casting, appeared to him a more reasonable method of arriving at relative strength.

Mr. BALDWIN agreed with Mr. Campkin there were many other considerations to be taken into account in estimating the value of the two methods of making gold plates. His colleagues and himself had been using the casting process a great deal for plates, and it had given great satisfaction on the whole, although he had known of one bar lower that came back broken, showing a coarse crystalline fracture. The great merit of the method seemed to be the accuracy of the fit which could be obtained, and the ease with which the plate could be strengthened along the lines of greatest stress, and when the casting was made directly to the porcelain of the teeth, the absolute cleanliness of the work; the metal was a sound metal, and not a piece of metal which had been subjected to unequal strains, such as was a stuck-up plate in the process of striking. The bar lower which he had referred to was mended by soldering the two halves together, and gave no trouble afterwards.

Mr. GABELL thought the question of porosity was a very important one, not only in connexion with strength, but in connexion with cleanliness. The difference between a cast plate and a rolled plate was the finer surface. He did not agree with the necessity of a cast plate for a high palate, his experience being that if cases of high palate and an overlap of the alveolus were treated with a cast plate there was a very bad fit. The shrinkage of the gold was quite appreciable, and if there were places where it could bind over the alveolus it would shrink away from the palate. Such cases were only satisfactory if the plate was not carried over the alveolus. The question of strength was of importance, because if very thin plates of gold could be made he thought it would be of advantage. Of late he had been making plates of very much thinner gold than the average, and for a palate where there was practically no strain at all the plate could be made exceedingly thin and held up by suction.

He had been using No. 1 plate in making full dentures for the whole of the palate part and a rim to give the requisite amount of strength. He did not make springy plates, believing a rigid plate absolutely necessary. Cast plates were often found very successful, and a comparison of the cost of a cast plate with a rolled plate would show that the cast plate had been made considerably thicker and heavier than the rolled; therefore, if thicker, it was stronger and stood the strains of practice. The second test with regard to the alternating strains was rather disquieting, because in making plates they were exposed to heat, and if heat lowered the strength it would be necessary to discover some means of doing the work without subjecting the plates to heat afterwards. He would like to know whether soldering bands to a plate appreciably affected their strength.

Professor McWilliam said it was quite true that the gold was an alloy, though 18-ct. gold was not such a complicated alloy as 9-ct. gold, and behaved somewhat after the fashion of pure gold, although not quite so. It was a very important point to remember in connexion with the experiments. It had also to be remembered that although such materials might not break with a crystalline fracture, practically all metals, unless cold-worked, were crystalline. Really the perfection of the intercrystalline cohesion rather than the fact that the metal was made up of crystals, small or large, seemed the important factor in determining the properties of the gold. If the crystals, as in the case of pure gold, held together thoroughly and were also individually ductile, they would pull out and give fibrous fractures in the breaking. With regard to the steps that had been taken to get parallel surfaces, the plates were made as nearly parallel as possible by mechanical means—polishing, and so on; they were not assumed to be parallel. Judging from experience, with steel more particularly, he thought crown work would be a very suitable thing for castings. In many large industries in connexion with iron and steel it was necessary to find out those things for which castings had to be avoided, because of possible breakdowns after many years' service. In other cases castings were eminently suitable. Crown work seemed to him to be something that would be suitable for the use of castings, but he could not speak from the dental point of view. He agreed with the remark as to strain, for he supposed that dentists would not be satisfied if the gold plate lasted only eighteen months or two years. The movements, owing to changes in the mouth, were, he understood, only small, and their effect upon the plate would be such that it would probably take a very considerable time to produce fracture, even if the plate were destined ultimately to break in use. With regard to the lowering of the strength by heating in connexion with soldering, the soldered plate had only a slightly lower elastic limit than the rolled one, and retained all its other good properties. There was a slight deterioration, as everybody expected, in a plate subjected to soldering, but the difference was only small. It was the cast plate that showed the very low limit of elasticity. As to porosity, he was sorry that the specimens did not show under these microscopes as well as under their own, that were specially fitted for examining metals, but this cast plate did show a kind of porosity—i.e., a very distinct and decided imperfection of surface, although the main body of the plate was fairly solid. It did not show, however, what were known as blow-holes.

Mr. H. J. MORRIS said personally he had not very much information as to the durability of the plates, but yesterday a friend of his had told him of his first casualty, and that was the first of which he had heard. No doubt failures would not be heard of for some years, until through absorption the alternating shock test which he had endeavoured to illustrate in the paper came into operation. With regard to the parallelism of the plates, and how pure gold would compete with other alloys, it had not been possible to go into such matters from an ideal point of view. What they had endeavoured to do was just to take specimens representing what was actually used by a dentist in his daily work, and see how they stood the tests, without endeavouring to experiment from the point of view of pure science. Perhaps after the researches had been continued there might be something more to tell the Section.

Odontological Section.

April 24, 1911.

Mr. W. B. Bacon, President of the Section, in the Chair.

(1) An Erosion Cavity on the Lingual Aspect of a Lower Incisor. (2) Dislocation of the Pulp of a Temporary Incisor, the Result of a Fall, with subsequent Removal of the Pulp and Apex intact.

By C. F. Peyton Baly, M.R.C.S., L.D.S.

I must apologize for the triviality of these communications. I do not wish it to be understood that the cavity in this first tooth is an erosion cavity: rather, I ask the question whether it is an erosion cavity? It is a case of a lower incisor from a patient, aged 65, who had been instructed to use floss silk, but who had apparently not been taught how to use it, and the result of his endeavours to keep his teeth clean with floss silk is that he has cut a clean little nick in the tooth.

The other case is one from hospital practice. The patient was a boy, aged 1 year and 10 months, who fell downstairs and knocked out an upper incisor. He was brought to the hospital with the tooth and what was apparently a black tooth in situ. On investigation this turned out to be the pulp of the tooth which had been left intact in its place in the mouth, and as the result of the irritation, had swollen up and was greatly congested. The pulp with the remaining apex of the tooth was extracted by means of a small straight spoon excavator, which was passed carefully up the socket, twisted round, and cut off the pulp, bringing the pulp and the ring of dentine intact.

DISCUSSION.

Mr. GABELL said with regard to the first communication he would like to know whether floss silk was used by itself or in conjunction with any tooth powder, and how long the treatment was kept up. He noticed there was a carious cavity on the mesial or distal surface of the tooth, in spite of the use of the silk. Was there any possibility of the matter being explained by the wearing of a plate?

Mr. BALY said no powder was used and no plate worn in contact with that tooth. It appeared to him that the patient, instead of passing the floss silk between the teeth, had looped it behind the tooth and pulled the two ends. The use of the silk had been going on for years. The tooth was extracted because it was loose. The other incisors were free from caries.

Mr. RUSHTON said the case seemed to bear out a contention of his that erosion cavities were nothing more nor less than friction cavities. Some years ago he did his best to try and find a number of erosion cavities for a certain subject he was working upon, and he asked the porter at the National Dental Hospital to save him teeth that were extracted; of the many thousands extracted at that Institution the number of erosion cavities was absurdly small. The teeth came from a class of people that did not use a tooth-brush. In private practice, amongst people who used a tooth-brush, such cavities were extremely common. He came to the conclusion, then, that erosion cavities were friction cavities, no matter what the friction came from, aided by the quality of the teeth or their environment. Mr. Peyton Baly had called the case a trivial one, but they never knew what important bearings a so-called trivial case might have.

Mr. STANLEY MUMMERY said the late Dr. Miller made an extensive series of investigations into mechanical erosion and came to the conclusion that a large number of so-called erosion grooves were due to the tooth-brush, but at the same time an equally large number were not due to any explainable cause. He had himself two patients in whom the lower incisors were worn or destroyed by erosion nearly down to the gum level: the bite was not edge to edge, and there was no apparent cause for the condition.

Mr. PEYTON BALY said from his general experience he rather agreed with Mr. Rushton that erosion cavities were mostly due to friction.

The Relation between the Teeth, and certain Diseases of the Skin and Mucous Membrane.

By W. Knowsley Sibley, M.D.

For many years I have often noted a connexion between eruptions on the face, scalp, and neck, and an unhealthy condition of the teeth or gums. Two explanations are apparent. The one, that the skin complaint is the result of an irritation, of one or more branches, of the fifth nerve supplying the teeth and oral cavity. This gives rise, by reflex action, to a lesion of the skin, over an area supplied by another branch, of the same, or an associated nerve, distributed to some area of the face, or scalp. The second, that more complex one, of a toxic condition of the blood produced by absorption of micro-organisms, or their products, from unhealthy gums, as in the common complaint pyorrhœa alveolaris.

It has long been recognized that various forms of stomatitis, ulcers, and herpetic eruptions of the buccal mucous membrane, are often the reflex result of dental troubles, and the seat of the eruption is by no means necessarily contingent to the tooth giving rise to the complaint.

The following skin diseases may be, and I believe often are, produced locally about the head and neck, and certainly in many cases are kept up by dental causes, and very frequently owe their origin to a neglected state of the mouth: Acne (comedones), acne rosacea, alopecia areata, eczema, erythema, herpes, œdema, seborrhœa and seborrhœic eczema. In this list I have not included those very obvious external lesions, produced as the direct result of inflammatory mischief inside the mouth, such as adenitis, lymphangitis, abscess, sinus and resulting scars, acute œdema, erysipelas, and other acute inflammatory dermatoses.

A study of skin eruptions on one part of the body, as the result of an irritation of a neighbouring area, may in some cases be assisted, and in others complicated by, the following dermatological problems. In some subjects, a quite local eruption produced by a traumatism will be followed by a symmetrical lesion on the opposite side, or on some distant region of the body, which has not been exposed to a traumatism or irritation. For example, a dermatitis produced by a trade or trauma on one hand, may be, and often is, quickly followed by a similar lesion on the opposite side. So again, a traumatic dermatitis on one part will frequently rapidly spread for a considerable area to the adjacent skin regions, far beyond the seat of irritation. Further, a local irritation such as is produced, for instance, by pediculi or scabies on one part of the body, is frequently rapidly followed by an irritable skin eruption over regions far removed from the offending organisms, and long before the parasites have had an opportunity of spreading. *Pediculi capitis* may be the cause of a general irritable skin eruption over the whole body. Eruptions on the face of children are frequently caused by pediculi on the occipital region of the hairy scalp.

I wish to insist upon the peculiar reflex, or perhaps sympathetic, nervous skin phenomena illustrated by the above remarks, as showing what an extraordinary sensitive trophic condition of skin is present in many people, and how this complicates still more any attempt at making dogmatic statements, or drawing definite mathematical conclusions, as to the origin of a given skin lesion.

Before studying some of the diseases individually, and attempting to prove a connexion between them and the condition of the teeth, it is necessary to make some observations on the cutaneous nerve distribution of the region under discussion. The areas of nerve distribution to the skin of the head and face, however, are much more complicated than is ordinarily supposed. It is well known that tender areas of the skin, after the destruction of definite branches of the fifth nerve, often by no means correspond to the acknowledged distribution of the same branch of this nerve. So again, the position of headache in people suffering from errors of refraction may be on almost any part of the scalp. Some of the dental branches of the fifth nerve, however, according to Dr. Head, have a very definite reflex area of pain distribution, when they are irritated or inflamed. For example, pain from caries of the upper incisors is referred to the frontonasal area, and that from the two upper bicuspids, in some cases, to the maxillary area, in others to the temporal, but never to both regions in the same subject. Again, in irritation of the nerves on the side of the tongue, pain is referred to the occipital region. Head also observes that there is an association between the tenth dorsal area and the occipital region of the scalp. There are numerous communications between the filaments of the cranial and those of the upper cervical nerves, which again are associated with the cervical sympathetic. There is also a well-known communication between the orbital branch of the superior maxillary division of the fifth nerve, and the lachrymal branch of the first division, which supplies the skin of the upper eyelid. Many areas of the scalp are supplied from two separate sources. Part of the temporal region, immediately above the ear, for instance, being supplied, both from the auriculo-temporal branch from the third division of the fifth nerve, and by the small occipital from the anterior branch of the second cervical nerve. There is also the association of distribution between the second division of the fifth and branches of the cervical plexus, and of the third division of the fifth, and by the primary divisions of the cervical plexus. The great auricular nerve which is formed by branches from the second and third cervical nerves has three divisions, each supplying a skin area of the face and scalp—the first, known as the mastoid branch, supplies the scalp behind the auricle; the second, or auricular, the external ear; and the third, or facial division, supplies the skin region over the parotid gland. So these areas are chiefly supplied from the cervical, and not from one of the cranial nerves at all. The exact areas of distribution are, however, very difficult to define, and there is no doubt that part, if not all of these areas are supplied by sensation from more than one nerve branch.

From these preliminary observations, it is seen that the regions of nerve distribution of the skin of the head and face are far more complicated than the superficial areas on most other parts of the body. It is thus seen how very difficult it is to localize with any very great scientific exactitude, the nerve distribution, or the probable result of reflex irritation of the same, on the skin areas of the head and neck. From this it follows, that to endeavour to determine the correlation between morbid conditions inside the mouth, with an unhealthy state of the skin, is one which meets with considerable difficulty. To state definitely that a given condition of a particular area inside the mouth will produce a sure and certain lesion of a definite skin area, is impossible in the present state of our knowledge. At the same time much may be gathered by a study of general principles, and this is one of the objects of this paper.

It is much easier to demonstrate the effect of irritation of certain filaments of the dental nerve, by the frequently observed reflex fibrillar contractions of some muscles supplied by the seventh or facial nerve, than it is to show the result of the irritation of the same filaments of the fifth nerve, on the trophic condition of the skin supplied by other branches of the same nerve. A toothache, or even nerve irritation, not sufficiently strong to amount to pain, will, in certain highly sensitive patients, produce a twitching of an eyelid, which will continue until the cause of the irritation is removed.

After these preliminary general remarks, I pass on to consider, under different headings, some of the skin diseases for discussion, commencing with the commonest disease met with by dermatologists at the present day—*Seborrhœa*, in all its various expressions of distribution, and variety of degrees of severity, from a slight dryness or scurfiness (dandruff) of the scalp, to a disfiguring eczematous eruption, spreading over the whole face and neck. A large number of these cases are associated with neglected dental caries, which I believe by reflex action sets up the irregular processes of the sebaceous and perhaps also of the sweat glands, and as long as the condition of the nerve irritation persists, all the more difficult will it be to deal with the seborrhœa satisfactorily. Chronic seborrhœa is by far the commonest direct cause of alopecia, both general and local. In some cases the hair of the scalp falls out fairly evenly all over, and a general thinning of the same results, which if unchecked ultimately leaves the patient more or less bald. Moreover, a seborrhœa of the scalp is frequently obstinate to treat, and shows a decided tendency to recur unless treatment be continued for weeks, or even months, after the region is apparently well. Seborrhœa of the face is more amenable to treatment. When, however, the condition has once become general the prognosis is most unfavourable for a complete cure, and hence the immense importance of seeing these cases in the earlier stages of the disease.

But the cases of *Alopecia Areata*, where patches of baldness occur in definite areas, are of greater interest. Dermatologists are divided into two schools as to the ætiology of this condition—those who believe in its parasitic origin, and those who hold it is entirely a neuropathic one. I have often considered that in some of these well-defined patches, both of alteration of the colour of the hair and of baldness, there was a connexion between the irritation of some branch of the dental nerve inside the mouth, and the nerve distribution of the patch of baldness on the face or scalp. In going into the history of cases of alopecia areata both of the scalp and also of the beard, moustache or eyebrow, it is by no means an unusual thing to learn, that from a few days to a few weeks prior to the commencement of baldness, the patient had a severe attack of facial neuralgia due to a carious tooth. One frequently finds that an attack of neuralgia on the right side of the face, for example, is followed by a patch of baldness on this side, and so pain from a dental cause on the left side is followed by an area of alopecia of this side. The association is easily demonstrated when the bald patch appears on the face, especially when in the same region as where the pain was felt. The connexion, however, is more difficult to determine when the hair comes out on the scalp, and a region of circular or oval baldness reveals itself in this region. The commonest areas of the scalp for alopecia areata are the parietal and occipital regions, that is to say, just above and behind the external ear, and on the back of the scalp, usually near the median line. The former area is supplied chiefly by the posterior auricular or auriculo-temporal branch of the second division of the fifth nerve, and the latter area chiefly by the great occipital nerve, and also to a lesser extent by the smaller occipital nerve, both from the cervical region.

Jacquet, of Paris, has made a most convincing attempt to prove the association of dental troubles with baldness, and has almost succeeded in demonstrating the relation by mathematical exactness. He collected 273 cases of alopecia, and found that in 185 of these the condition manifested itself in the first twenty-five years of life, that is to say, corresponding to the period of the eruption of the teeth. Moreover, that very few of these cases occurred between the ages of 15 and 19, or that period when there is an *entr'acte* in the dental evolution. He sums up his observations by stating that in his opinion "alopecia is a trophic trouble, of which one of the most frequent pathological conditions consists in an irritation of one part of the buccal trigeminus, which is reflected to an area of minimum cutaneous nerve distribution, in anatomical correspondence with the spot irritated."

In discussing the relation between the condition of the teeth and the hair, there is always the interesting embryological connexion, which often throws light on certain unusual conditions. Developmental abnormalities of the teeth are sometimes associated with abnormalities in the formation or structure of the hair. There is also probably a degenerative relation between the two as well. Premature caries may be associated with premature changes in the hair, either in the loss of colour and commencing greyness, or in baldness showing itself earlier in life than is usually the case.

Acne and comedones, eruptions very common on the faces of adolescents, of both sexes, are often associated with dental troubles. Young people are more apt to neglect their teeth than those of more mature experience, and so it frequently happens that many of the worst cases of acne seen occur in subjects of severe dental derangements and neglected caries, producing a general state of oral sepsis. This condition probably acts both reflexly, in the irritation of the skin distribution of the fifth nerve, and also assisting the morbid process, not only by the absorption of septic bodies, but also from the resulting dyspepsia and constipation. It is therefore most important in treating these cases to insist on having the teeth properly attended to. There is no doubt a connexion between severe and persistent facial acne, and early and troublesome dental caries.

Another very common skin complaint, *Eczema*, is, I am sure, frequently associated with dental troubles. Teething is popularly considered as the cause *par excellence* of eczema in infancy and childhood. In a large number of cases there is no doubt a close relation between the digestive organs and eczema, a condition so frequently, not only in infants but also in adults, due to defective digestive processes, both of assimilation and of excretion, and often primarily traced to the teeth.

Probably there is no skin disease more directly and indirectly dependent upon the condition of the teeth than *Acne Rosacea*, that very common condition of redness of cheeks, nose, and chin accompanied by varying degrees of pustulation—a malady more common in women towards middle life, although also occurring in men, and often giving rise to a good deal of disfigurement in both, and not at all infrequently associated with defective dental conditions. I consider the first thing in all cases of rosacea, is to have the teeth thoroughly examined and treated. It is useless applying soothing lotions and ointments to the skin of the face, if an irritation is permitted to continue inside the mouth. If, on the other hand, the morbid condition of the teeth and gums is cured, a soothing lotion or ointment applied externally as a protection to the part from the changes of temperature, and other irritating climatic influences, will bring about a very considerable improvement of the complexion. Rosacea is often entirely the result of dyspeptic troubles, the origin of which is to be found in the mouth, either in the form of dental caries, deficient masticatory apparatus, or a pyorrhœa alveolaris, or a combination of all three causes. The red nose produced by acne rosacea, popularly considered to be a sign of drinking habits in the individual, is now known to occur in a large number of most temperate people, including teetotallers themselves. In many cases the disturbance of the physiological action of the skin is the direct result of a pyorrhœa alveolaris, and after proper attention to the condition of the gums, the skin of the face gets well, and the complexion, together with the general health of the patient, rapidly improves. The dermatologist who treats the skin and leaves the condition of the mucous membrane uncared for, is certainly not doing justice to the immediate alleviation of the symptoms, and is only assisting the future troubles of the patient. As an example the following case illustrates my opinion:—

A single woman, aged 27, had suffered for some years from an acne rosacea, having, she stated, had a red nose from childhood. She had tried several treatments, with little or no improvement. On examination, I found she had several carious molars and bicuspids in the upper jaw, and she had lost many of the teeth from the lower. I tried local treatment without success, and then sent her to a dental surgeon, who removed all the carious stumps and inserted new teeth in their place. The skin of the whole face soon rapidly improved and she is now free from acne, and the colour of the nose has resumed a normal appearance.

Temporary patches of *Erythema* appearing on one side of the face, on the cheek, or under the eye, are frequently due to dental causes. I saw a case recently of a middle-aged man, who for many months had had a mysterious patch of erythema suddenly appearing under his right eye, and lasting a few days and then disappearing, to reappear again a short time afterwards. This phenomena produced little or no discomfort, except the curious appearance it gave to his face, accompanied by a slight burning sensation. It quite disappeared after some carious teeth on this side had been attended to.

Most skin complaints commence as quite local conditions, and the face and scalp are certainly the commonest areas to be first affected. If the disease begins on the face, advice is usually sought; if, on the other hand, the scalp is the area primarily attacked, the condition, especially in men, may remain unobserved for a long period. A pathological state, at first purely local, will in a large majority of cases eventually spread, and may in time invade the whole body, and become a source of great annoyance to the patient, and one taxing all the skill of the dermatologist to deal with satisfactorily. A typical instance is a slight seborrhœa of the hairy scalp, at first consisting of an unnatural dryness of the area showing itself by a slight scurf or dandruff. This may remain untreated for years, until the accompanying falling out of the hair, or some discomfort, or irritation of the scalp area, draws the attention of the patient to the malady. From this a seborrhœic eczema may extend over the forehead, on to the face, and down the back of the neck, to the trunk, and hence all over the body; and perhaps the whole process was started by neglect of the teeth and gums.

Œdema (Localized).—Localized and diffused swellings of the skin of the face, neck, or of some area of the oral mucous membrane, are usually the result of some dental trouble, and occasionally give rise to difficulty in the diagnosis. The following case I saw some years ago may prove interesting:—

A girl, aged 16, had an attack of erysipelas of the head and face three months previously, and was confined to bed for three weeks. About this time she had a good deal of pain in one of the left upper molars, which was carious. About a fortnight after this, and two months before she presented herself at the hospital, her upper lip began to swell, and she had some slight pain in the left upper central incisor; this pain soon subsided, but the swelling of the lip had persisted ever since in spite of all treatment. There was at this time considerable, more or less solid œdema of the whole of the upper lip. The skin over it was shining and glossy, and the edge of the lip was considerably everted. There was some caries of the left upper central incisor, the rest of her front teeth were healthy. The carious incisor was removed on February 2, 1906, and by February 5 the œdema had nearly subsided, and a week later the lip was quite normal.

Having described some cases of skin diseases to illustrate my remarks, I pass on to make a few observations on some diseases of the mucous membrane of the mouth, also the direct result of dental troubles. These are: epithelioma, herpes, leucoplakia buccalis, lichen planus, ulcers (acute and chronic).

Ulceration of the oral mucous membrane produced by friction contact with a rough, or even only a rather prominent tooth is very common, and unless producing marked pain or considerable local discomfort, is often apt to be overlooked by the patient for a long time. Sometimes as the result of constant friction or pressure, together with slight erosions of some small area of mucous membrane, either on the side of the tongue, or of the buccal mucosa, continued for months or years, a thickening even amounting to a warty or papillomatous growth may ensue. This after a time becomes especially liable to ulceration and cancerous degeneration.

Epithelioma of the oral mucous membrane is usually the result of local irritation by a tooth, not necessarily a carious one. I describe an interesting case I saw some years ago:—

A man with an early epitheliomatous ulcer round the base of an isolated prominent lower molar tooth had also a smaller ulcer of the same nature on the inside of the cheek, where this part of the mucous membrane came in contact with the crown of the same tooth. There was thus a considerable area of apparently quite healthy mucous membrane, in the region between the two ulcers, showing clearly two separate cancerous lesions, both produced, or their site determined, by the irritation of a tooth on a mucous membrane unusually susceptible to an epitheliomatous growth.

Leucoplakia buccalis.—Sharp, rough and decaying teeth are often considered a contributory cause of this condition, sometimes popularly known as smokers' patches of the mouth. The sites of election of the lesions are the inner surface of the cheek, in a line following that traced by the junction of the teeth of the upper and lower jaw when shut, and the gums above the upper canine and lateral incisors. I have no doubt that in the case of a chronic persistent patch of leucoplakia which is either in proximity to, or is brought into contact with the teeth in the movements of mastication, the wise course is to remove the tooth or teeth in question. Many years ago I showed before the North-West London Clinical Society, a case of leucoplakia in a man aged 34. This condition affected the centre and side of the tongue, and there was also a well-marked ridge of the same complaint on the buccal mucous membrane, corresponding to the line of junction of the molars, which in this patient were all particularly well preserved. The man had been a great smoker and probably had had syphilis.

Lichen planus in the mouth usually takes the form of flat papules aggregated on one or both sides of the cheek, the striæ running along the line of the buccal mucous membrane where the molar teeth come in contact, when the jaw is shut.

I have not described the clinical features of a number of dermatological cases to illustrate my remarks, but have only related briefly a few somewhat unusual, or to my mind particularly interesting, ones, which I have abstracted from my case-books. The majority of the patients I come across are cases of various forms of acne and seborrhœa, and present no individual features of interest, although collectively they form a strong argument in favour of the marked association of many of these morbid skin eruptions with dental trouble in some form or other. As long as the origin of the complaint which is inside the mouth is overlooked, so long will it be before a satisfactory result can possibly be obtained by the dermatologist's pharmacopœia. And it is this fact that I desire to emphasize, that in every case of a skin eruption on the face, the condition of the teeth and gums should be carefully examined, and if any even suspicious places be discovered, immediate treatment for this condition should be recommended. I should not rely entirely on lotions and ointments to eradicate or cure a condition of the skin area supplied by one branch of a nerve, and leave untreated an irritated or perhaps septic condition of another branch of the same, or an associated nerve.

I feel sure my remarks will bring to the memory of many of you interesting cases of skin eruptions, which may have long puzzled the doctor in charge, but which you have been able to cure by attention to the teeth.

Having described several of the diseases of the skin affecting local areas, that is to say, eruptions on the face, scalp and neck, due or in some degree connected with the condition of the dental apparatus, I propose before concluding, to make a few remarks, on more general skin eruptions, the result of absorption of deleterious bodies from unhealthy gums into the general system. I shall only just mention those somewhat rare cases of general septicæmic conditions produced by septic absorption from the mouth, and revealed by various septic erythematous rashes, such as hæmorrhagic purpura, or general diffuse furunculosis, and pass on to say something on the subject of pyorrhœa alveolaris, and the important part this disease plays in the production of skin eruptions, on parts of the body, often far removed from the regions of the head and neck.

A great number of apparently more or less different lesions of the skin are produced and kept up by gastro-intestinal disturbances; dental caries with pyorrhœa in the mouth is one of the most frequent causes of intestinal catarrh, fermentation, and general dyspeptic conditions. The pyorrhœa produces these symptoms in two ways: in the first place, the perpetual swallowing of small quantities of pus, contaminated with large numbers of septic organisms in time produces an unhealthy state of the gastro-intestinal tract and its contents. In the second place, the absorption into the system, of the products from the septic gums, produces a general lowering of the vitality and a condition of chronic septicæmia, with all its accompanying phenomena, and including under its heading many of the common forms of chronic diseases in the human race. Many cases of troublesome eczemas at all ages are due to gastro-intestinal causes. Urticaria invariably so. Many erythemas, again, are traced directly to stomach derangements. Even psoriasis is considered by some dermatologists to be an external expression of the same internal troubles, and the most efficacious treatment is one which regulates the diet of the individual—in other words, the gastro-intestinal absorption.

This list of diseases might be considerably extended, and would of course include all those skin conditions which may occur in gouty or rheumatic patients, whose constitutional ailment is but the result of dyspeptic disturbances, the sequelæ of dental troubles, and so many of which owe their origin to unhealthy gums. Excluding skin diseases due to ecto-parasites, fungi, cocci, pediculi, and the like, a large number of people, and practically all hospital patients, except some very young subjects, are the unconscious victims of pyorrhœa in some degree or other. I am convinced that in many cases, to attempt to treat the skin complaint, and leave the gums untreated, is a very unscientific procedure. I am engaged in some research work in the Pathological Laboratory of the St. John's Hospital for Diseases of the Skin, where the Bacteriologist, Mr. W. Garner, has prepared for me some vaccines from a series of cases of skin diseases, in patients also the subjects of pyorrhœa alveolaris, and I have been treating these cases with hypodermic injections of vaccine. The method adopted for preparing the vaccine from the pyorrhœa of each case individually has been as follows: The gum in the neighbourhood of the pyorrhœa is well wiped over with a sterile swab, and all saliva and other contaminations from the mouth, as far as possible removed. Gentle pressure is then exerted on the gum, towards the root of the tooth, and a bead of pus pressed up from the socket; this pus is also removed with another swab; pressure is again exerted as before, if necessary rather deeper and firmer, until another bead of pus is forced up. A sterile platinum loop is then inserted into this fresh pus, and a culture tube of agar-agar or broth inoculated with it. As soon as a good growth has appeared on the agar, 4 c.c. of sterilized normal saline solution is poured into this test-tube, and the growth gently mixed up and stirred in this. This mixture is then poured into a fresh sterile test-tube and well shaken up with some beads, and then, if necessary, centrifugalized for a minute or so, to remove any clumps of growth which have not become finely divided up. A specimen film is then prepared with an opsonic pipette, containing two volumes of normal solution, one volume of standard blood, and one volume of the growth emulsion. These are all well mixed together, and a film made on a clean slide; after being dried and stained, a count is made, and a standard vaccine prepared, so that each cubic centimetre contains 10 million or 100 million of organisms, according to the nature of the growth; the vaccine is then ready for use. An injection is given about every ten days, the dose being usually gradually increased according to the reaction obtained. Each case must, however, be treated on its merits, and no hard and fast rule can be laid down, either as to the most suitable dose of vaccine, or the length of period between the injections—in other words, every patient has his, or her, own individual peculiarities of reaction, which must be dealt with accordingly.

I will now describe one or two examples of vaccine injections:—

A case of erythema bullosum in a single woman, aged 31, who had never been seriously ill, and always considered herself strong and healthy. She had an erythematous eruption accompanied by superficial bullæ eight years previously, affecting the neck and face only. The condition lasted for three months and she recovered. The present attack had lasted for sixteen months when she first came under my observation on February 22 of this year. The eruption commenced on the back, then on the neck, and upper part of the chest, then the forearms, and afterwards the legs. All these regions were extensively covered by *erythematous* patches in various stages of bullous formation, and many areas of pigmentation, and some superficial scarring from previous lesions. The extremities were cold and somewhat cyanosed. There was no obvious cardiac lesion. There was considerable pyorrhœa alveolaris, affecting all the lower incisors, and the left first bicuspid. The growth obtained from the pus was a mixture of streptococci, staphylococci, and slender bacilli, and from this a vaccine was prepared of such a strength that 1 c.c. contained 10 million organisms. On March 2 an injection of 5 million was given, and the next day a fresh lesion appeared in the lumbar region, there not having been a new lesion for some time previously. On March 9 another 5 million were injected, and this was followed by a fresh lesion on the dorsum of the right foot. Other injections were given on March 20 (7 million) and March 30 (10 million); fresh lesions followed each injection. I also treated the pyorrhœa directly by short exposures to X-rays, and gave her an antiseptic mouth-wash, but no internal treatment was given. On April 13, as a control experiment, she had an injection of aqua destillata. She has remained free from fresh lesions since, so to-day she had 5 million vaccine organisms injected. The condition of the pyorrhœa has already considerably improved, and she continues well in herself.

A man, aged 44, had suffered for many months from very troublesome chronic urticaria all over the body, which was very rebellious to various treatments, and he also had some acne rosacea on the face. A considerable amount of pyorrhœa was present, affecting most of the teeth on the left side of his mouth, and the lower lateral incisor, and the canine on the right side. A pure streptococcus culture was obtained from the pus and a vaccine prepared; an injection of 5 million was given on March 9, on March 16, 10 million were injected, and on April 13, 5 million more.

On making bacteriological examinations of the pus from cases of pyorrhœa under the microscope, I have been struck by the presence in practically every case examined of spirochætæ, which in specimens prepared by the Chinese ink method are morphologically indistinguishable from the *Spirochæta pallida* found in so many syphilitic lesions, and considered by most authorities to be the specific organism of that disease. This spirochæte is present in addition in most cases to the one resembling the *Spirochæta refringens.* I am not prepared at present to say if these spirochætæ are anything more than accidental organisms in pyorrhœa.

In the majority of cases streptococci were present, in some staphylococci, and in some both, in addition to numerous other less easily defined organisms, but in the majority of the cases a pure streptococcus culture was obtained.

I have placed under the microscopes specimens of the spirochætæ found in pyorrhœa and in syphilis for your examination and comparison.

My sincere thanks are due to Dr. Serrell Cooke and Dr. T. Galbraith for their valuable help, and kind assistance with the vaccine treatments.

BIBLIOGRAPHY.

DAUZATS. "Recherches sur la contagiosité de la Pelade," *Thèse de Paris*, 1901.
DE LAVARENNE. *Presse méd.*, Par., 1902, x, p. 171.
EYRE and PAYNE. *Proc. Roy. Soc. Med.*, 1910, iii (Odont. Sect.), pp. 29, 61.
GOADBY. *Brit. Med. Journ.*, 1908, ii, p. 477.
Idem. *Lancet*, 1911, i, p. 639.
HEAD. *Brain*, 1894, xvii, p. 406.
HUNTER, W. "Oral Sepsis," *Practitioner*, 1900, lxv, p. 611.
JACQUET. *Ann. de Derm. et Syph.*, Par., 1902, 4me sér., iii, p. 97.
PEDLEY, R. D. *Trans. Odont. Soc.*, 1905, N.S., xxxviii, p. 67.
SIBLEY, *Trans. Path. Soc. Lond.*, 1891, xlii, p. 381.
Idem. "Stomatitis Neurotica Chronica," *Brit. Med. Journ.*, 1899, i, p. 900.
TRÉMOLIÈRE. *Presse méd.*, Par., 1902, x, p. 567.
TURNER, J. G. *Practitioner*, 1910, lxxxiv, p. 115.

DISCUSSION.

Mr. JAMES asked Dr. Sibley in what way he definitely associated diseases of the skin with conditions arising in the mouth. The paper covered a very large area and it was rather difficult to follow at the moment exactly what Dr. Sibley was endeavouring to prove. There appeared to be three classes of cases—those which occurred by direct extension from the mouth, those due to reflex disturbance, and those due to gastro-intestinal disturbances. With regard to the first group, the speaker had seen cases in children which undoubtedly appeared to be of this nature. Of the second group it seemed possible that vascular changes and possibly trophic changes could occur which might favour a skin lesion, although this was hardly borne out by clinical experiences. The chief affections seemed to be those due to gastro-intestinal trouble, which might be the result of oral sepsis.

Mr. GABELL was very much in the same difficulty as Mr. James. It appeared to him that most of the cases described were nervous reflexes and not direct infections. He had seen acne rosacea as a mere extension in people who had pyorrhœa. Dr. Sibley described a great many cases of affections of the skin, found that the patients had pyorrhœa, and inferred that the pyorrhœa was the cause. But did Dr. Sibley realize the enormous number of people who had pyorrhœa—how very few had not got it? If he did, why did he attribute the skin affections to pyorrhœa? It did not seem to him a very sound argument. The treatment by vaccine was very interesting, but as Dr. Sibley appeared to leave the local condition untouched, he could not see very much hope of success. The first patient shown had violent pyorrhœa, and copious infection was still going on; it seemed hopeless to treat the case by mere vaccination.

Dr. KNOWSLEY SIBLEY, in reply, said that if, as the result of a dermatitis on some part of the body, produced by a local irritation after a short time, owing to a reflex or sympathetic irritation of the nerves of the skin, a similar lesion appeared on the opposite side, or even on some other region of the body, so he maintained, that when there was a violent nerve irritation, such as was produced in dentition, a nerve irritated by caries or other causes, it was explicable to get a skin lesion due to trophic changes of the nerves in the skin area, especially in the superficial areas supplied by the same or an associated nerve. The whole of the areas of the skin supplied by the other branches of the fifth nerve must be equally irritated. He had seen so many cases in which local treatment of the skin failed to cure, but which had been rapidly healed by treating the condition in the mouth. One case was that of a hospital nurse, who had for years been under treatment for the condition of the skin of her face, but until she had her teeth attended to there was no success from the treatment, and then she got perfectly well. Even the colour of her nose, which had been red since she was a girl, became normal. The other cause was septic absorption. A large number of skin diseases were proved to be gastro-intestinal in origin, due to disturbances such as indigestion, rheumatism, the absorption of deleterious bodies from the intestines, &c.; that is to say, the skin lesion was an external expression of an internal condition. The cause of indigestion in a large number of such patients was dental trouble, as caries, the absence of proper masticatory surfaces, or pyorrhœa. With regard to the hospital cases not having been treated locally, each patient had been told to see the dentist, and have his or her teeth attended to, but they simply would not go. Such people thought that if they did not have toothache they had nothing the matter with their teeth. The man he had shown with urticaria had been under treatment for a year, and although advised on several occasions to see the dentist he had never done so. Undoubtedly such cases should be treated locally, the teeth extracted or the tartar scraped, the pockets swabbed out, in addition to vaccines, but of course it would be very interesting to obtain improvement through vaccines without having the teeth treated at all. If the teeth were attended to locally, vaccines would very soon clear up whatever pyorrhœa happened to remain. The more cases he saw the more he was convinced that a large number of skin diseases were due to troubles in the mouth in one form or other. It was well known how sensitive the skin was, and a dermatitis in one spot might be repeated all over the body. Pediculi on a child's head might produce an eruption over the whole body. Exactly in the same way, irritation inside the mouth might produce trophic or sympathetic changes in the skin areas far beyond the region of the face.

Odontological Section.

May 22, 1911.

Mr. W. B. Bacon, President of the Section, in the Chair.

A Dentigerous Cyst with Cellular Contents.

By W. W. James, F.R.C.S., L.D.S.

A youth, aged about 20, presented himself with a large cystic swelling in the right maxillary region. The upper temporary canine was present, with absence of the corresponding permanent tooth. A skiagram showed the tooth to be lying above and behind its normal position and placed horizontally, the crown being directed forwards (fig. 1). At the operation, on incising the cyst the fluid contents were not normal in character, but thicker than usual and showed yellowish streaks. The idea that pus was present seemed very improbable from the clinical history. Some smears were prepared and stained. The first slides demonstrated the presence of cells, but the nuclei were not stained. The cells were in such numbers as to account for the appearance of the fluid. By placing a slide for twelve hours in a very strong solution of hæmatoxylin some nuclei were successfully stained. The slides show numerous cells which vary in size and shape; they are epithelial in type. There is a large quantity of granular material and the cells show varying degrees of degeneration with nuclei undergoing division (figs. 3 and 4). The cyst wall was removed in one piece except in the neighbourhood of the tooth, which was lying on the inner aspect of the cavity. At first the tooth could not be seen, although the cavity, which was quite as large as a hen's egg, was dried without difficulty; the cavity wall was not smooth, but irregular on its inner

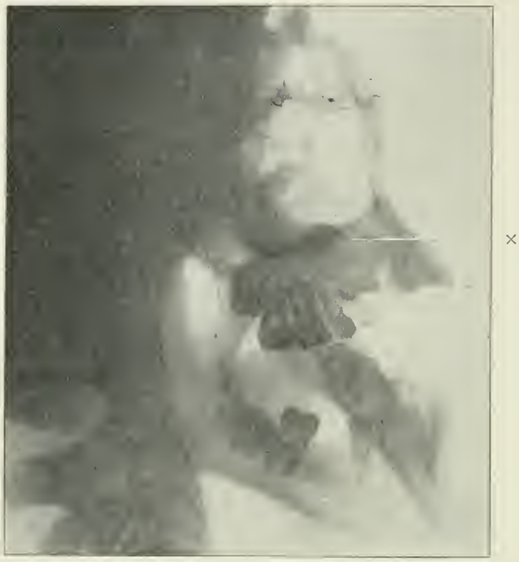


FIG. 1.

Radiograph of a dentigerous cyst. The right upper canine tooth is lying horizontally, with the crown directed forwards, ×.

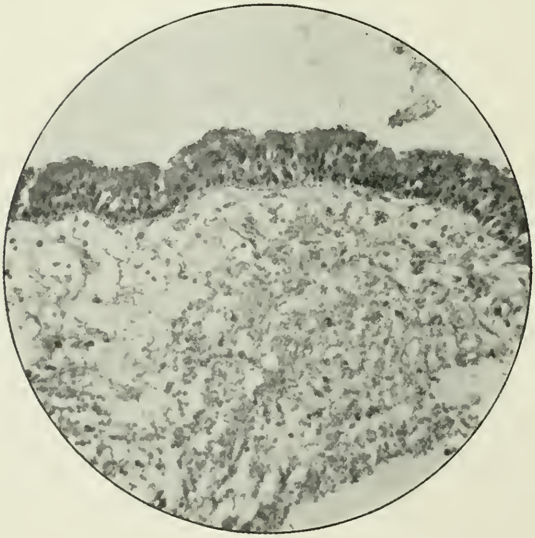


FIG. 2.

Section of the cyst wall showing the epithelial lining.

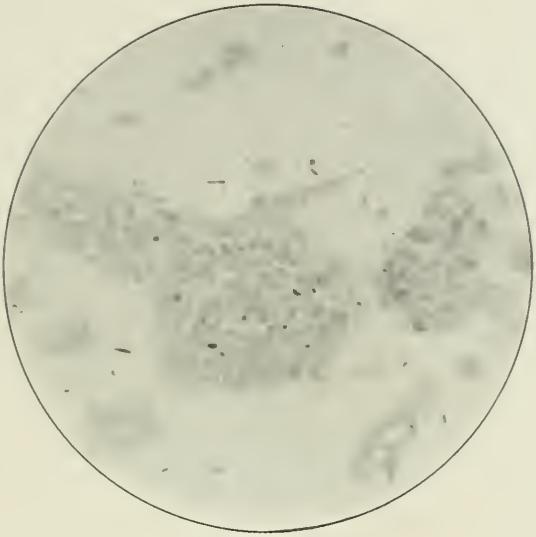


FIG. 3.

Cells undergoing degeneration from the fluid of a dentigerous cyst.

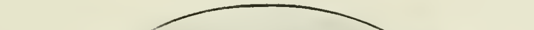


FIG. 4.

Cells undergoing degeneration from the fluid of a dentigerous cyst. (Higher magnification.)

aspect. A whitish patch proved to be the crown of the tooth; the root projected backwards and was certainly not directly connected with the cavity. The relationship of the tooth to the cyst is of great interest in these cases. Some have now been described in which the whole tooth has been within the cyst, and from this condition, through all gradations, to where the cyst was situated over the tooth, which, although associated, was definitely outside its walls. These various types are of importance from the ætiological point of view. Some advance has been made by the general recognition of the presence of an epithelial lining, although there appears to be some difficulty in always demonstrating its presence. That it occurred has long been recognized, but that it is always present has only been established recently. As there does not appear to be an illustration in the literature upon these cysts showing the epithelial character of the lining, a slide has been prepared from this case (fig. 2). The origin of this epithelium has been attributed to the stellate reticulum of the enamel organ. The amount of this tissue is so small that although it is conceivable that by proliferation and degeneration a cyst could be produced, the more probable explanation would seem to be that the epithelial lining is derived from the external epithelium of the enamel organ or from the more superficial epithelial cells of the tooth band. The crown of a normal tooth until its complete eruption is always capped by a layer of cells derived from the external epithelium, whilst the cells of the stellate reticulum are lost at a very early stage in the development of the tooth. The enamel is usually well developed and to such a degree that one would hardly expect the cyst to have commenced previous to the obliteration of the stellate reticulum. Cases with imperfect teeth are described and might therefore have arisen at a sufficiently early stage. There is one other comment which ought to be made: These cysts are undoubtedly due to a degeneration of epithelial cells, and under Mr. Bland-Sutton's classification must be placed as "aberrations of the enamel organ." The term "follicular odontome," therefore, cannot be properly applied to them.

Mr. Clark has kindly lent me the skiagrams which have been shown.

DISCUSSION.

Mr. HOPEWELL-SMITH said that until more was definitely known regarding the functions of the stellate reticulum it could not be determined what was its relationship to these cysts. He, with Dr. Marett Tims, had lately been cutting some sections of the developing teeth of the kangaroo and had found very large enamel organs, many having a large stellate reticulum, containing blood-vessels. It appeared to him that the stellate reticulum was incorrectly looked upon as a "packing material" and that it was now necessary to consider it almost in the light of a filter of serum. He thought that fact might have some bearing on the formation of fluid in dentigerous cysts.

Mr. PITTS asked whether the tooth was examined to see whether Nasmyth's membrane was present. In some of the cases that membrane had been found to be absent, a thing one might expect if the cyst was caused by a collection of fluid between the external and internal epithelium.

Mr. GABELL thought the analogy of the specimen shown by Mr. James to those shown by Mr. Turner in dental cysts some time ago was very instructive. Mr. Turner showed degenerating epithelium precisely similar to that described by Mr. James. In the early stages of the dental cyst there was an epithelial mass but not a stellate reticulum, and therefore he did not think there was any reason to suppose a stellate reticulum could be concerned in the formation of a dentigerous cyst.

Mr. JAMES, in reply, in answer to Mr. Hopewell-Smith, said that dentigerous cysts had been described far more frequently in human beings than in animals, and in human beings the stellate reticulum was very small in amount and also became lost at a very early stage. He thought the argument used by Mr. Gabell was a very strong one indeed. There was a great resemblance between the cysts, and a cyst might be regarded as a dentigerous cyst which was really and truly dental, the tooth lying just outside the wall, as in the case described recently before the Section.[1] With reference to Nasmyth's membrane, an examination was being made, but he did not know yet what the result would be.

---

[1] *Proceedings*, p. 14.

Experiments and Observations on Bread, with Special Reference to the Causation and Prevention of Dental Caries.

By J. Sim Wallace, M.D., L.D.S.

I never was quite satisfied with some experiments carried out by Mr. H. Candy under the direction of Mr. T. G. Read, and for many years hoped to see them corroborated or refuted.

You will remember that these experiments were said to show that during mastication ordinary steel-milled white bread gave rise to 43 per cent. more acid than did bread made from stone-milled flour. These experiments appear to have given rise to the impression or assumption that white bread fermented more rapidly in the mouth that did bread made from stone-milled flour. It was impossible for me to gather from the published reports the significance of Mr. Read's experiments. I am now willing, however, to accept the statement about the extra 43 per cent. of acid being produced from the white bread, but only on the supposition that, to all intents and purposes, no acid is produced during the mastication of the stone-milled bread, and that 0 + 43 per cent. of 0 is produced during the mastication of the white bread. The reason for my saying this is, that when stone-milled bread and when white bread are masticated and then ejected into a suitable vessel, it will be found that both are distinctly alkaline. Moreover, if equal amounts of bread made from stone-milled flour and from white bread are masticated, and each put into an incubator and left to ferment for a number of hours, it will be found that they ferment fairly exactly at the same rate. (Dr. Leonard Hill has shown the same result with regard to standard bread.) In fact, as it happened, at least in my experiments, the stone-milled bread actually showed a slightly greater amount of acid than did the white bread. Here I may indicate the kind of experiment which I made to illustrate this point, and others which I shall refer to subsequently: 200 gr. of white bread were thoroughly masticated, and then instead of being swallowed they were put aside in an incubator and left there for twelve hours. Water and litmus solution were then added, and neutralized with a 0·5 per cent. solution of caustic potash. The number of cubic centimetres of this solution which were required to neutralize the acid was then noted. Similarly, 200 gr. of stone-milled bread were subjected to like treatment. These experiments were repeated several times, with the slight variation that in some cases the stone-milled bread was masticated first and the white bread immediately after; in the other cases the white bread was masticated first and the stone-milled bread immediately after. The reason for trying the amount of fermentation in both of these ways will be apparent later. Now the exact figures showing the average amount of acid formed in twelve hours from 200 gr. of bread treated in this way are: Stone-milled bread (crust and crumb) required 14·7 c.c. of the K.H.O. solution (average of twelve experiments); white bread (crust and crumb) required 14·0 c.c. of the K.H.O. solution (average of twelve experiments).

Before referring to my next set of experiments I would like to refer to the late Dr. Miller's famous experiments, which, together with the enormous influence for good in regard to the solution of the pathology of caries, seem to me to have misdirected attention, to a certain extent at least, with regard to its ætiology. Miller's experiments, you will remember, were conducted as follows: 5 gr. of the foodstuff were mixed with 4 c.c. of fresh-drawn saliva, left to ferment, and the degree of acidity tested in a similar way to that I have already indicated. Here, however, you observe the experiment does not in any way represent a state of affairs such as is liable to exist at the exact sites where caries commences. If one looks into the crevices of the teeth, which are the usual sites of caries, after a meal has been taken ending with bread, it will be seen that covering over and extending beyond the incipient carious spot a visible quantity of bread may be seen. It is not, then, less than 1 per cent. of bread in saliva that we have to deal with, we have rather to deal with the foodstuff and the saliva in more like equal proportions. In fact, when some foods are eaten, chocolate for example, for a considerable time the chocolate lies in the crevices almost without admixture with saliva at all. One cannot really object to Miller's experiments or methods, however, but only to the erroneous assumptions which have been made from them. One of them I must refer to. It has been assumed that the harmfulness of any particular foodstuff might be judged from its fermentability. To a certain extent this is no doubt true, but it is of still greater importance to know first: (1) Whether the particular foodstuff is apt to lodge about the teeth for a considerable time, and (2) whether the foodstuff may itself have been effectual in removing the bacterial plaques or masses from the masticating surfaces of the teeth. Then, again, it has often been assumed that the mouth is more or less constantly supplied with something approaching 1 per cent. of carbohydrate, remaining behind in the mouth after practically all meals. It was assumed that there was very generally some little carbohydrate and, consequently, some acid being formed in the mouth. The whole theory that the *locale* for the initiation of dental caries depends on points of least resistance in the tooth seems to me to have been largely imagined as the result of the assumption that there was in the mouth a diffuse production and presence of acid in a medium—i.e., the saliva—which might only contain a fraction of 1 per cent. of carbohydrate. Similarly the theory that acids in foods, acid saliva, and acids eructated from the stomach gave rise to caries, seems to have gained currency on the assumption that the food and acid, which tended to initiate caries, was not located in the mouth at a particular spot, and that the acid formed was not, as it were, fixed or located, but wandered about till it found points of least resistance. It is not, however, points of least resistance which are important. But it is most important to recognize the existence of points which favour the periodic stagnation of carbohydrate foodstuffs in appreciable quantity, for it is at such points that caries is invariably initiated, and whether the tooth-substance in the neighbourhood of these spots is more or less resistant to solution by acid does not in any way influence the intensity of the carious process, although it *may* influence the rapidity of the destruction of the tooth-substance. It appeared to me, therefore, that it would be more satisfactory to try, to a certain extent, to imitate the degree of concentration of the foodstuffs in the saliva at the exact seat of the incipient carious spot. At first, in order to try to ascertain in what form bread might give rise to the most rapid fermentation, but subsequently to ascertain in what form bread was most detergent, I masticated equal quantities (200 gr.) of the crust and crumb, each separately, and noted the resultant amount of acid produced after incubation for twelve hours. The average amount of acid produced in this way was as follows:—

200 gr. crust of bread (white stone-milled and standard) produced on the average an amount of acid which required 16·8 c.c. K.H.O. solution to neutralize it (average of eighteen experiments).

200 gr. bread without crust produced on the average an amount of acid which required 13 c.c. of K.H.O. solution to neutralize (average of nineteen experiments).

Notwithstanding these results, it must not be imagined for one moment that crust of bread ferments more rapidly than crumb, or that crusts are more liable to induce caries than crumb ; on the contrary, the amount of fermentation indicates rather how effectually each or any of these varieties of bread cleans the masticating surfaces of the teeth, and removes the plaques or masses of bacteria which, by lodging unduly and protected from the beneficent action of the saliva by superimposed bread, would most effectually induce caries.

That the greater amount of fermentation from the crust results from it incorporating greater numbers of bacteria is indicated by the fact that 200 gr. of crust masticated before a meal give rise to much more acid than when the same amount is chewed after a meal, or even after the first 200 gr. have been masticated and ejected from the mouth. Thus :—

Crust chewed before breakfast required (on the average of nine experiments) 18·6 c.c. of K.H.O. solution to neutralize.

Crust chewed after breakfast required (on the average of eight experiments) 13·3 c.c. of the K.H.O. solution to neutralize.

Crust chewed before breakfast required 19·5 c.c. of K.H.O. solution (average of four experiments).

Crusts chewed immediately after the previous crusts had been chewed required 16·2 c.c. of the K.H.O. solution to neutralize.

While doing these experiments I kept a sound tooth, with the enamel exposed, in the fermenting bread ; it being passed on after washing from one experiment, testing the amount of fermentation in twelve hours, to another similar experiment. I only thought of doing this towards the end of my experiments, and, consequently, it was only in the fermenting medium for seventy-two hours. It was not in the slightest way influenced by the acid fermentation around it. It was my expectation to get this result, to indicate that when bread with crust, or toast, is masticated, the plaques of micro-organisms are generally sufficiently completely removed from the masticating surfaces of the teeth to prevent a rapid production of acid at any one spot on the tooth, even when it lodges for hours at any particular spot. This experiment should have been continued much longer ; still, I am of the opinion that the small amount of acid formed, not from plaques located at a particular spot on the tooth but from micro-organisms distributed relatively sparsely in the fermentable foodstuff, would not induce caries, even were such fermentation repeated over and over again for several hours, as the saliva under such circumstances would always counteract the decalcifying tendency of the acid formed.

As far, then, as the masticating surfaces are concerned, I think we may say that the lodging of bread would tend but little, if at all, to induce dental caries, provided the adherent masses or plaques of bacteria could be and were frequently removed, or rather were not allowed to form. And here it should be noted that the nature of the bacterial flora of the mouth in general is not necessarily of much importance. It is quite possible, indeed most probable, that the bacteria in a crevice which is regularly supplied with carbohydrates are of an active acid-forming type, flourishing most rapidly in a carbohydrate medium. Dr. Black, you may remember, said: "As I have cultivated micro-organisms a great deal, and especially in relation to this matter, I wish to say distinctly that the same micro-organisms are found growing in the mouths of persons who are immune to decay, as are found in the mouths of persons who are susceptible to decay and those whose teeth are decaying rapidly, and the same micro-organisms will grow in the saliva of either when used as a cultivating medium." Nevertheless we are justified in saying that the fact of the same micro-organisms being present in different mouths at points where decay is not taking place is not an important point. What importance, indeed, can we attach to the general bacterial flora, or the nature of the saliva in general, when at one place we may have decay becoming arrested and at another decay commencing. Surely the exact local conditions and the exact localization of particular kinds of micro-organisms are the points of paramount importance in this matter.

It may be said that, no matter what is eaten, masses of bacteria do adhere about the teeth even after the thorough mastication of crusts or anything else. This is quite true, and between and at the necks of teeth, masses, layers or plaques of bacteria cling to the teeth after masticating anything. But those situations are kept free from caries on a somewhat different principle. There is a coating of mucus at these situations in which the bacteria seem to flourish fairly well, but which, however tenaciously it adheres to the teeth is sufficiently greasy and slippery to permit of the easy and rapid removal of carbohydrate food particles. If the teeth are observed some short time after eating bread, it will be found that this slippery mucoid film is present in situ, but not the bread. Food particles occasionally are loosely adherent to this mucoid mass for a time, but they are more or less freely exposed to the action of the saliva, and are superficial and not impacted when there is a normal arrangement of the teeth. The protection of the tooth from caries at the neck should be more closely studied than it usually is.

The deepest layers are the most alkaline and most infiltrated with *liquefying* micro-organisms. This is a necessary corollary from Miller's experiments with saliva and mucus. The superficial parts of this mucus coating are removed and deposited more frequently than the deepest layer next to the tooth. Consequently the deepest part must, generally speaking, be alkaline, even though the superficial and more recently deposited layer be acid, because if mucus is left to ferment for a time it rapidly becomes alkaline. The micro-organisms, however, keep up a slow disintegration of this mucus, so that a constant change or continual slow flux keeps the neighbourhood of the necks of the teeth alkaline, and at the same time free from any foodstuffs which otherwise might lodge. Towards the bulge of the tooth and above it of course the layer of mucus is very thin, and being renewed after each meal it tends to become acid when newly deposited; but, inasmuch as it is thin, mixed with and bathed in saliva, it would be impossible for it to become sufficiently acid to decalcify the enamel. Saliva, as you know from Head's experiments, effectually prevents the action of weak acids upon the teeth.

It is true, of course, that caries often attacks the necks of the teeth. Here, again, however, in order to get this result, it is necessary to get food to lodge more or less continuously over or in the layers of mucus and bacteria in these situations. Certain viscous foods—e.g., bread and marmalade, or cake—may certainly lodge upon or partly impregnate the mucous layer over the necks of teeth, or this layer may become fairly thoroughly soaked in sugar when sweets are more or less continuously sucked; or, again, foods even though not of a particularly sticky nature, may be liable to lodge when the mucous membrane forms a sort of pocket near the necks of the teeth on the buccal side of the wisdom teeth; but, except in such situations, there is no distinct tendency for food particles to lodge about the necks of the teeth when they are covered with the mucous coating and the gums have not receded. When, however, this mucous coating is frequently removed and a small groove cut with a tooth-brush, then the inclined and slippery plane is broken, and food tends to stagnate at the necks of the eroded teeth. Caries may then take place, for fermentable carbohydrates may lodge in such situations in more or less immediate contact with the teeth, as the beneficent protection of the mucous coating has been removed, and instead of facilitating the removal of fermentable carbohydrates they are induced to lodge under conditions which favour the initiation of caries.

The next and last set of experiments which I would refer to are with regard to bread and marmalade: 200 gr. of this mixture (160 gr. of bread and 40 gr. of marmalade) were masticated and tested as in the previous experiments, the result being that the bread and marmalade gave rise to acid requiring for neutralization 11·6 c.c. of K.H.O. solution, while the white bread alone required 12·2 c.c. of the K.H.O. solution to neutralize the acid formed; that is to say, as far as fermentation was concerned, my experiments might be supposed—if only fermentability were considered—to indicate that bread and marmalade was rather less harmful than bread. To anyone who has made statistical comparisons of the effect of bread, and of bread and marmalade on the teeth, the idea that bread and marmalade is no more harmful than bread alone is ridiculous. But we must not consider the fermentation alone. Marmalade stimulates a rapid flow of saliva, and, consequently, the bread and marmalade do not require or receive the amount of mastication that bread alone demands. Consequently, bread and marmalade is less effectual in cleaning up the bacteria from the masticating surfaces of the teeth. Secondly, marmalade is viscous and clammy in the mouth, and therefore may stick about and between the teeth, and saturate everything about the teeth, and by its viscosity it may prevent the free action of the saliva cleaning between the teeth or penetrating into the mucous or bacterial coating at the necks of the teeth. The viscosity of sugar is well known, but to indicate how sugar is not removed from the mouth by the saliva, or that it is not easy for the saliva to exert a beneficent effect on anything through the viscous sugar, I masticated some sugar and put it into a test-tube and then left it to ferment. Before fermenting it was of course viscous; twelve hours after fermenting it was still viscous. Similarly, saliva was put into a test-tube and left to ferment; it was of course slightly viscous, but compared with the sugary solution it was relatively limpid. I put litmus solution into each, and then shook the test-tube round to mix up the colouring matter, but so viscid was the sugar solution, especially towards the bottom, that I could not get the colouring matter to reach the end of the tube in this way. The litmus solution mixed readily with the saliva, but this saliva could not penetrate into the viscous sugary solution.

Before going further it may be desirable to define the cause of caries, for, although to a certain extent I am going to make some observations based more or less on the experiments, it is difficult not to be influenced by what knowledge we already have of the cause of dental caries derived from different evidence and sources. To me, in a general way, *the cause of dental caries is the undue lodgment of fermentable but non-detergent carbohydrates in more or less immediate contact with the teeth, and undisturbed by the free access of saliva.* Bearing this in mind, and the experiments already briefly detailed, what can we say about bread in relation to dental caries? We can very safely say that :—

(1) No farinaceous foodstuff in general use in this country is less harmful to the teeth than bread when eaten with butter, and no farinaceous foodstuff is more beneficial from the point of view of oral hygiene; especially is this the case when the bread is eaten with a goodly proportion of crust.

(2) The different varieties of bread (i.e., white, standard, and stone-milled) make no appreciable difference in inducing dental caries, beyond the difference which the physical differences of the bread make in their detergent effects on the teeth. That is to say, the less finely ground may be presumed at least to have the more detergent effect. Whether standard bread may be considered as being less finely ground than white I know not, but as far as my experiments on this point go, the fermentation of standard bread and white bread gave identical results in this respect, both for crust and crumb.

(3) The crust is always preferable to the crumb in all varieties of bread. Similarly, toasted bread of any variety is preferable to untoasted bread of any variety.

The amount of phosphates or proteid absorbed from one kind of bread may be greater than from another kind. It is said that more phosphates are absorbed from white than from coarse wholemeal bread. From the point of view of dental caries this does not seem to be of any consequence whatever. It is certainly of no consequence as regards the temporary teeth, which have their enamel completely formed before bread is ever tasted.

As regards lime salts in bread, and their effect on the calcification of enamel, here, for similar reasons, we may conclude that this has nothing to do with the production of caries. A certain amount of lime salts is necessary, both for the teeth and for other purposes, but whether it is too much that is in the different varieties of bread or too little, seems to me to be of remarkably little consequence. What evidence we have seems to show that the teeth are as excellent in form and constitution whether a diet notably rich in, or notably free from, lime salts is indulged in. Judging from Professor Moore's and Professor Winckler's observation, possibly the breads having the smaller amounts of assimilable lime may be regarded as best from other points of view, especially if supplemented by cow's milk. But we need not speculate on this; we know, of course, that the chemical analysis of the teeth shows that it is not lack of lime salts at all that predisposes teeth to decay. But since the impression may exist that in a certain variety of bread there is a deficiency of lime salts, then there is all the more opportunity for insisting that a more or less exclusively bread diet should be supplemented with vegetables, in which lime salts are present in ample quantity, and in which the cellulose is present in a more agreeable and less irritating form than in wholemeal bread, besides being in a more detergent and correspondingly hygienic form. Again, if the amount of carbohydrate in white bread is proportionately great, this will give us all the better reason for insisting that jam and marmalade should not be spread upon it, but that it should be supplemented by butter, fish, flesh, or fowl, or other proteid and fatty food. To talk about sugar being a great "proteid sparer," when already too much carbohydrate and too little proteid have been consumed, is on a par with sending coal to Newcastle in order to save the inhabitants of that city from using something else which they could not possibly do without.

Though bread has but little to do with the induction of caries, how it is taken and what it is taken with is of great importance. Thus, the wicked practice of cutting off the crust, or soaking good detergent bread in milk till it has thoroughly lost its power of stimulating mastication and keeping the mouth clean, should be noted. We surely do not want to see the mouth in a continual state of fermentation, teeming with bacterial plaques, and contaminating all the food which has possibly been carefully selected and sterilized for a child's welfare. To ruin the detergent effect of the bread directly is bad enough, but, unfortunately, this is not all, for cow's milk not only contains an excess of lime salts, it contains also excess of proteids, and thus instead of a child having its bread supplemented by albuminous food of a cleansing nature, it is generally actually deprived of this because it has already got it in its most expensive and least detergent form—i.e., milk. Bread and marmalade is another of those unphysiological combinations which tend to ruin the cleansing effect of bread, as we have already indicated, and the habit which many families have of finishing breakfast with bread and marmalade makes it particularly objectionable from the point of view of oral hygiene. Bread and jam to finish off the children's "tea" is, of course, equally harmful.

But now, although the proportion of salts in white or in wholemeal bread has nothing whatever to do with the chemical composition of the tooth, I am not at all sure that the elimination of the earthy salts or a goodly proportion of the inert and innutritious cellulose is an advantage, especially for those who are found to live very largely on bread alone. These earthy salts, and the cellulose which is present in wholemeal bread, may be conducive to the hygiene of the alimentary canal, and whether more proteid is absorbed from the white bread or whether more earthy salts are absorbed from it does not seem to me to be of any appreciable consequence except for those who are on the borders of starvation. What leaves the mouth and alimentary canal in the most hygienic or healthiest state is what is of importance. It is always disease and never starvation which affects the development of the teeth. That the wholemeal bread should not be very coarse, lest it should give rise to intestinal irritation, should perhaps be remembered at the present time. Possibly, probably indeed, coarse wholemeal bread is quite suitable for those who masticate their food very thoroughly, but so many do not, and many more cannot. Thus, therefore, although wholemeal bread is to be preferred, especially because of the innutritious matter which it contains, it may be safer to advocate rather finely ground wholemeal bread. And for anyone who wants to try the silly experiment of living on one foodstuff *alone,* standard bread may be recommended. Certain experiments at least seem to indicate that such a course would be advisable under such circumstances. But before assuming that this results from "superior nutritive qualities" let us be sure that it is not due to something quite different. The craze for nutritive foods seem to blind some people to other often more important considerations. In the advocacy of standard bread the fact that pigeons fared better on this than on white bread has been brought forward. For long years pigeon fanciers have known that old mortar, little stones and pigeon grit in addition to their grain food was good for their pigeons' health. Similarly experimentalists have shown, or think they have shown, that chickens brought up on sterilized food were liable to become weak and die, unless or until they were supplied with some dirt of a rather unsavoury description. It is not my intention to indicate that standard bread derives its virtues in similar ways, but rather to indicate that we should exercise our common-sense before we extol the nutritive value of any bread; and as regards the teeth, the last thing that should be brought forward as a cause of the prevalence of dental caries is the lack of nutrition in bread. If there was more innutritious matter in our vegetable foodstuffs the teeth would fare much better; if the sugars were less concentrated than they are in sweets, if they were diluted with water and weak vegetable acids and incorporated with cellulose as they are in their natural state in fruits, we should hear less about the increase of caries. Man, more than any other animal, eats food which is particularly rich in nutritious matter and particularly free from innutritious matter. At the same time he has decayed teeth to an extent which makes him, as regards teeth, the ridicule of the animal kingdom. We can understand wholemeal bread being advocated because of the innutritious cellulose or for other innutritious constituents which it contains, but it is rather a whimsical argument to advocate bread for the teeth, necessarily containing a large amount of carbohydrate and proteid only of a non-detergent kind, because of its "superior nutritive qualities."

Moreover, we should remember the law of evolution, both retrospectively and prospectively, and instead of worrying over the problem as to what single food would *per se* be the most suitable, we should rather recognize that in matters of diet there has been, and is still further bound to be, an evolution from simple, indefinitely satisfactory, single or few food diets to the varied, definitely co-ordinated and complex dietetic regime which best subserves the needs of the highly evolved and complex physiological unit, man.

DISCUSSION.

Mr. F. J. BENNETT said he felt more grateful to the author at the beginning of his paper than he did at the end. At the first he had hoped that Dr. Sim Wallace's scientific investigations had reached a point which would lead him to abandon philosophical discussion. But Dr. Wallace did not continue his investigation of scientific fact to the end. For fifty years people had speculated as to the cause of caries, but the speculations had led to no advance beyond that which was known at the time when Robertson, of Birmingham, announced his theory of caries. Facts arising from experiments were of the utmost importance, and it was in that respect he considered the work of Miller to stand head and shoulders above the work of any other man. Some of Dr. Sim Wallace's speculations were problematical; for instance, the condition of the bacteria lying deep down in a fissure, covered by a mucous layer. It did not appear that Dr. Wallace submitted that to the crucial test of experiments. The drawing shown by Dr. Sim Wallace was obviously incorrect, both as a representation of the crown of the tooth and of the neck. He wished it to be distinctly understood that he was not carping at Dr. Sim Wallace's work, but only asking him to leave that form of investigation so common north of the Tweed, namely, philosophical speculation, for more definite scientific experiments.

Mr. CAUSH, while thanking Dr. Sim Wallace for the experiments which he had so carefully carried out, agreed with Mr. Bennett that the drawings of the teeth might have been a little more accurate. He did not think he had ever seen the sulci in a bicuspid quite in the position shown. With regard to the food question, he thought its value had been much overstated, that any investigation into the cause of caries should go beyond that of food, and take into consideration the structure of the enamel itself. During the last quarter of a century he had been devoting some time to the study of the structure of enamel, and had carried out a series of experiments in staining that tissue, with the object of discovering whether there was any difference in the structure of the enamel in different portions of that tissue. His microscopic slides and photomicrographs showed that there was a difference in the structure of the enamel when comparing the tissue surrounding the sulci, and the thin layer of enamel at the necks of the teeth, with the rest of the enamel. The question of bacteria plaques might or might not be of importance. He had a number of slides in which there were bacteria plaques and no decay, and a number of sections of decay in which there were no bacteria plaques. He believed the question of caries was more a question of the structure of the enamel at any given point, and that Dr. Sim Wallace's paper would have been of much more value if in conjunction with the experiments an endeavour had been made to find out if the enamel was the fossilized tissue suggested by Dr. Leon Williams, or the semi-calcified tissue suggested by himself (Mr. Caush), as that would possibly have helped in the solution of that great question, "The cause of dental caries."

Mr. COXON said years ago it was his lot to look after the case of three children who were fed on wheat which corresponded to some extent to the standard bread of to-day. The children had grown up very strong and healthy, but he did not see that their teeth were anything like as good as those of their parents. The children were fed on the finest red and white wheat flour, from which nothing was extracted except that it was very carefully prepared and cleansed, ground in a stone mill, and it was the only bread the children were allowed to have. Now at the ages of from 25 and 22, the teeth were decidedly worse than the teeth of their parents. But the children themselves were very fine specimens of healthy manhood.

Mr. J. F. COLYER had been very pleased to hear Dr. Sim Wallace deal with the interesting question of stone-mill and roller-mill flour. He had always thought that Mr. T. G. Read's experiments required confirmation. He had no doubt Dr. Sim Wallace's results were correct, but he would have been more satisfied had he experimented with the saliva of immunes and non-immunes in a similar way to Dr. Miller—in other words, carried out a greater range of experiments. Somehow or other he thought the stone-mill flour bread was better for the teeth than the roller-mill flour bread, and he attributed it to the fact that stone-mill flour bread required more mastication and hung about the teeth less than the roller-mill flour bread, and left the mouth in a cleaner state. With regard to caries, he was definitely of opinion that it was not on the increase, that amongst children who were well looked after it was on the decrease. It was quite easy to bring up children without decay of the teeth by carrying out the ordinary rules of hygiene. He saw hundreds of children whose mouths were absolutely free from decay, and he did not believe for one moment that they had stronger enamel than their parents; their teeth were free from caries because they were carrying out the simple ordinary rules of oral hygiene. With regard to sugar being probably the main factor in the prevalence of caries, it seemed to him that caries had developed in ratio with the imports of sugar into different countries. It was not right to say that sugar had always entered into our dietary, because sugar had only entered into the diet of the British race since the discovery of tropical countries. With regard to the whole question of foodstuffs, it should be remembered that whatever foodstuffs were used, if a child's mouth was to be functional two things were necessary, first that the child must not be a mouth-breather, and secondly, that it must be able to utilize every single tooth in its mouth. Therefore the child had to be made a nose-breather and every tender tooth removed. The child should be prevented from eating between meals, so as to give the saliva a chance of doing its cleansing work. Certainly sugar should not be the last thing in a child's diet. In that way, and by keeping the child from constant eating of sweets, 75 to 80 per cent. of the caries in this country would disappear.

Dr. J. SIM WALLACE, in reply, said that if the philosophical method which was supposed to be characteristic of the Scotch was to be opposed to the Baconian method he would plump for the Scotch. Of all the stupid things he had ever heard it was to suggest that experimental work should be commenced before the experimentalist had thought out his subject. After a man had exercised his thinking faculties it was quite possible to arrange very simple experiments to bring home the truth to people who could not think for themselves, but until thought had been exercised experiments were merely obstacles in the way. It was not very long ago since we were overloaded with undigested facts about dental caries, while at the same time there was not the faintest idea of how to bring up children without carious teeth. The only grudge he had against his countryman John Hunter was that he came to England and gave currency to the saying, "Don't think, try." Hunter probably knew that it was rather a foolish thing to say, but no doubt he wanted to get the better of the English. The experiments he himself had made had wasted most of his Sundays for the last six months, but he did not ask anyone to attach much importance to them, or to assign much importance to the diagram which had served such a useful purpose for his friend Mr. Caush. He had only meant it to serve the useful purpose of bringing home the relation of bacteria to carbohydrates under certain circumstances. With regard to the mucus, Mr. Bennett seemed to think he had been making philosophical speculations, but that was not the case. It was Miller's laboratory experiments he was speaking of. With regard to submitting the mucus to microscopical examination, there was no need whatever to use the microscope, because the facts could be seen with the naked eye. Anyone looking into the mouth would generally be able to see, unless the teeth had been very carefully brushed, the slight mucous coating, or if it could not be seen it could be scraped off, and if that was not satisfactory to microscopists it could be placed under the microscope. With reference to the question of special attention being paid to the kind of flour, he would only say that the more care taken of children's teeth *before the proper theory of dietetics came into existence* the worse they generally became. The method generally adopted was to follow the doctor's advice, and the doctor's children generally had worse teeth than other children. He would have been pleased to have tried his experiments with the saliva of immune and susceptible people as suggested by Mr. Colyer, but he had never seen any person he really thought was immune or susceptible except because of irregularities or other deviations from the normal shape and arrangement of teeth. As to Mr. Colyer's remark about his preference for stone-mill bread, it would be noticed from the paper that there was rather more fermentation from the mastication of stone-mill bread than white bread, and when he obtained those results he wondered whether they might have been due to the fact that a little more muscular effort was required to masticate the crusts thoroughly. It was rather a difficult point to settle and the amount of difference was very small. Still, if the idea was that the greater the fermentation caused in this way the less harm, then perhaps the stone-mill bread should be preferred to the roller-mill bread.

He had never been able to get a grip of the "capillary theory." If the mouth was open and there was a liquid lying in the floor of the mouth, capillary attraction would no doubt cause that liquid to come up between the teeth, but it would also bring up the saliva, and saliva was antagonistic to a certain extent to the decalcification of the teeth by acid. Consequently he did not see that the capillary theory was of very much value. But the viscous theory, or the theory that sugar penetrated into the mucus and clung about the teeth, could hardly be called capillary attraction. If he understood it rightly it was not capillary attraction that was meant but the viscosity of the sugar which got between the teeth and remained there.

Odontological Section.

June 26, 1911.

Mr. W. B. Bacon, President of the Section, in the Chair.

A possible Cause of Facial Asymmetry.

By W. Rushton, L.D.S.

It is a commonplace observation that, in many of our patients, the two halves of the face are not symmetrical; a fact sometimes prominently brought to our notice when we are engaged in fitting artificial dentures. It is also a well-known fact that artists and photographers choose the "best side" of their sitters when taking their portraits, for the same reason; and the fact that Gustavus III of Sweden had a face in which one side differed markedly from the other has served as a theme for one of our most gifted novelists of to-day, Maud Coleridge, in her romance "The King with Two Faces."

Through the kindness of Professor Rushton Parker, of Liverpool, there has been brought to my notice a paper by Dr. Richard Liebreich,[1] in which the author brings forward a theory to account for this condition, which I shall briefly lay before you. The author's attention was first drawn to the subject when engaged in researches on the origin of myopia, the difference between the two eyes, and its effect on vision. He subsequently investigated many thousand skulls of different periods and nationalities, and came to the conclusion that asymmetry of the face is a characteristic sign of the human race and is found amongst all races and has always existed. This conclusion brings him into direct conflict with Lombroso and Max Nordau and that school, who class asymmetry as one of the stigmata of degeneracy, and who hold that asymmetry of the cranium and face is the outward and visible sign of an ill-balanced intellect. Dr. Liebreich replies that if it is a sign of degeneracy, the human race has been degenerate for many thousands of years, far into prehistoric times, and he cites ancient skulls and mummies (including the famous Rameses II) in support of his contention. The usual form of asymmetry, which he says occurs in 97 per cent. of cases, is, briefly, that the left side of the face is flatter than the right, chiefly noticeable in the malar bones. The maxilla comes next in distortion and appears displaced to the right; the left surface being flatter and the left canine fossa being less marked. In a small number of cases the opposite takes place. In the dry specimen the differences are more marked than in the living, as some of the asymmetry is effaced by the skin, flesh, and fat. The most marked difference is noted in the malar bones; the right being prominent, while the left with a wider curve is deflected backwards and slightly upwards. This causes a difference in the form and position of the borders of the orbital cavities. That on the right is practically in the same plane as the face, while the left is in a plane inclined backwards. The superior external angle on the right is more advanced than that of the left, which is also a little higher. His observations on dwellers in town and country might have tempted him to conclude that asymmetry

---

[1] "L'Asymétrie de la Figure et son origine," by Dr. Richard Liebreich (Masson et Cie, Paris).

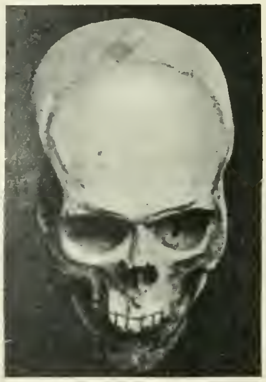


FIG. 1.

Flattening of left side of face, with displacement of median line to the right.

increases with culture, and therefore bears some relation to intellectual development; but he rejects this theory and concludes that the cause must be found elsewhere, and claims that he has discovered it in the domain of embryology. He states his conviction that it is the result of a purely mechanical cause—namely, the pressure of the bones of the pelvis on the head of the fœtus. In the usual fœtal position the weight rests on the left cheek, which explains the usual form of asymmetry, resulting in a pressure from left to right. The opposite position, which is much more rare, forms the opposite form of asymmetry, pressure from right to left. Pressure shows itself also in the maxilla and the teeth, the irregularity being more marked in cases of cleft palate, where the imperfect dental arch offers less resistance to pressure. One would naturally expect this pressure to be greater in the case of the European woman surrounded by a tight corset than in the case of, say, the Negress, and this is so. Heredity, too, may play a part. The author states, in conclusion, that after having found the origin and wide distribution of facial asymmetry, he set himself to discover the reason of this irregularity and found it as follows: "One knows," says he, "that the head of the embryo, and with it the rest of the body, is displaced from the median position to a lateral position, because the antero-posterior diameter of the pelvis is smaller than its other diameters. This difference depends on the vertebral column and on the curve of its inferior part. It is this curve, as well as the two other curves of the vertebral column, which is indispensable for the upright position,

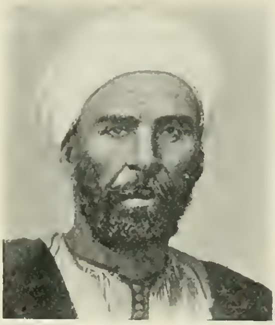


FIG. 2.

Facial asymmetry; general displacement to the right.

walking, and other human movements." He thus arrives at the conclusion that facial asymmetry is a consequence, a necessary accompaniment of the erect position of the genus *Homo*, and becomes one of the distinctive signs of it.

I have it on the authority of Professor Herbert Spencer, of University College Hospital, that the theory propounded by Dr. Liebreich is a reasonable one, and that he agrees with it. In cases of extra-uterine gestation the deformity is sometimes very marked. He considers, however, that Dr. Liebreich's figures are incorrect, and that, instead of 97 per cent. and 3 per cent., they should be 70 per cent. and 30 per cent. respectively.

I have brought this communication before the Section firstly to place it on record, and, secondly, to inquire whether it has any practical bearing on our work. Dr. Liebreich says there is a tendency to flattening of the alveolar arch on the affected side, but as regards my own collection of models of irregularity I must confess to having failed to see any such phenomenon, and I should be glad to know if any member present has done so. The other point which strikes one is that it may have some relation to deflection of the nasal septum and disease of the antrum, but Mr. Herbert Tilley has not noticed any such relation, though it may exist. It is a subject probably more of academic than practical import, but I thought it of sufficient interest to bring it before your notice.

In conclusion, it might be pertinent to ask what rule is to be observed as to obtaining correct alignment when fitting artificial dentures when the face is asymmetrical?

I am indebted to Mr. Doherty for preparing the lantern slides, which are taken from Dr. Liebreich's paper.

DISCUSSION.

Mr. THOMSON asked whether it was assumed that the human fœtus was asymmetrical from birth? If so, it would involve its being asymmetrical before birth. He did not know whether human skulls of children immediately after birth had been examined and found to be asymmetrical, but if it was only a question of the adult skull being asymmetrical, then he thought nothing was proved with regard to inheritance, as it might be due to environment. It was well known that the face of a child might be affected by its being carried on the one arm of the mother or suckled more on one side than the other.

Mr. STANLEY MUMMERY said the same thought had also occurred to him. He had frequently noticed in looking down on the top of a baby's head that there was a considerable difference in the two sides, and on inquiry he always found that the baby lay on the flattened side. He had noticed it particularly in children a few weeks or months old. When a child lay on one side the head was pushed across and the face was thrown a little to one side. Asymmetry was not confined entirely to the face and skull, but often extended to the whole body. Every man had a slight difference in the length of his legs and arms, and the ribs also were sometimes higher on one side than on the other. It would be interesting to know whether Mr. Rushton had examined people with a view to discover whether asymmetry of the head corresponded with asymmetry of the body, or whether there was no relation between them.

Mr. W. RUSHTON said he was not an authority on the subject himself. All he could say was, that the writer he had mentioned had studied the subject, and that the view put forward by him was upheld by so high an authority as Professor Herbert Spencer. As to how much was due to heredity and how much to environment, that was a matter which really nobody knew much about. One theory was that it was due to the method of holding or suckling the child, which might be as worthy of belief as the theory that a deflected septum in certain cases was caused by persons blowing their nose always with the right hand, and that by blowing the nose with the right hand and left alternately a deflected septum was avoided. With regard to the connexion between asymmetry of the body and asymmetry of the head, he had no information upon the point, but he thought asymmetry of the body was chiefly caused by disease, such as rickets, and by occupation. A little time ago Professor Arthur Keith, in the course of a most interesting series of lectures at the Royal College of Surgeons, exhibited the bones of a man found in a stone cyst near Walton-on-the-Naze. The right humerus and scapula were very much larger than the left, and the asymmetry was probably caused by the man's occupation—he might have been a right-handed flint chipper or blacksmith. It was an interesting subject, but the only practical point to the members of the Section was its bearing on the articulation of teeth or the fitting of artificial dentures, and he had hoped for something helpful to be said on that subject.

Cases of Mal-occlusion of Permanent Molars.

By F. St. J. Steadman, M.R.C.S., L.D.S.

I have the models of two cases showing mal-occlusion. In the first case the three upper molars on the left side bite completely outside the lower molars (figs. 1 and 2). The patient is a girl aged 25. I could get no history whatever of any bad habits such as tongue-sucking or anything else that could account for the irregularity. The upper arch appears to bulge outwards on the affected side, and the lower arch is flattened.

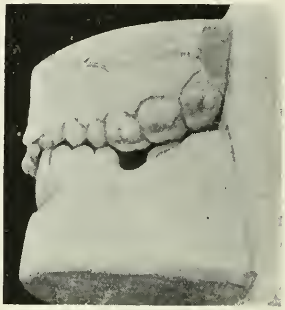


Fig. 1. (Case I.)

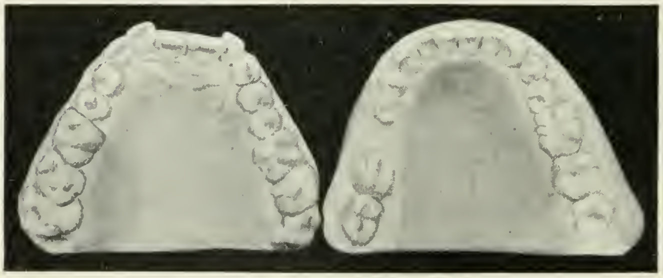


Fig. 2. (Case I.)

The second case is that of a man aged 30. The second left lower molar is the only one affected; it bites inside the corresponding molar (figs. 3 and 4). In this case the upper arch appears to be perfectly normal in character, but the lower arch has a very marked inward curve on the affected side.

The right side of the first case is normal. In the second case the right lower teeth had been removed before I saw the patient. Judging from the straightness of the alveolar ridge, this side also was normal. The upper teeth I removed myself owing to advanced caries. The interesting point, upon which I should like to hear opinions, is the cause of the condition, as I could get no history of anything that would account for it.

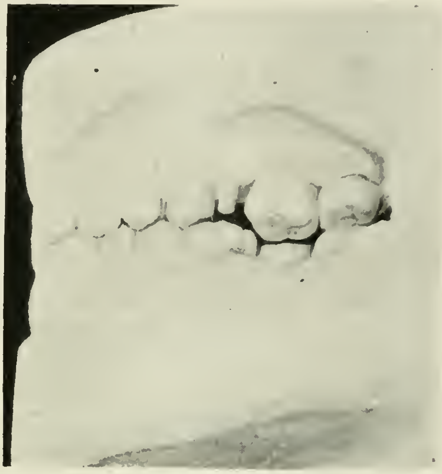


FIG. 3. (Case II.)

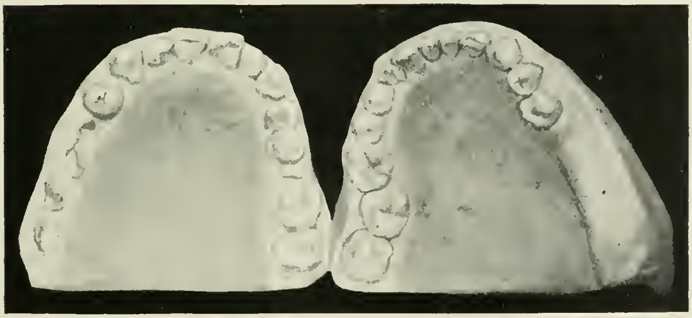


FIG. 4. (Case II.)

DISCUSSION.

Mr. J. F. Colyer was much interested in Mr. Steadman's cases, and referred to one patient under his care in whom both the premolars and the first and second molars were in a similar condition. He happened to have models of the girl before she cut her first permanent molars, and her deciduous teeth were in perfect occlusion, showing that whatever had brought about the outward throw of the premolars and molars had occurred since the age of 6. What it was due to he did not know. He could put his hands on half a dozen cases, but that was the worst one. The interesting feature was that the condition occurred subsequently to the eruption of the deciduous teeth. The first permanent molar came down definitely outside the lower teeth, and then the first premolars, when they came down, had the curious thrust.

Mr. Thomson thought the explanation of the two models was that some of the molars had been extracted on one side. In the case where the permanent molar was inside the arch, the six-year molars had been extracted or had decayed, and the twelve-year-old molars had come forward and crowded the teeth inside. In the other case the six-year molars had been extracted also, and had allowed the bunching up of the three molars, a thing which often took place, and the twelve-year-old molars were crowded out. Sometimes they were crowded into a triangular form. There did not seem to be the usual signs of sucking of the tongue across the molar teeth. He had seen a number of cases of that kind. In one case the finger on each side had been used, and the whole of the upper teeth had been thrown outside the arch. The present cases, however, were due to nothing of that kind, but simply due to the extraction or loss by decay of the molar teeth.

Mr. J. F. Colyer could not agree with Mr. Thomson's explanation, because if Mr. Thomson were right one would expect to find thousands of cases, taking into consideration the number of people for whom extractions had been performed. He thought the explanation was much more likely to be found in a slight developmental defect. As illustrating the light comparative pathology threw on such things, there was sometimes a very rare abnormality in which the premolar sometimes erupted externally and sometimes internally to the arch, with all the deciduous molars absolutely in place. It was very difficult to account for that irregularity when it was remembered that the premolars, as a rule, were developed, as it were, within the roots of the deciduous teeth. In the anthropoid apes the premolars were developed well above the roots of the corresponding deciduous teeth. It was therefore quite likely in the type of case under consideration there was an aberration in tooth development; instead of the premolars being developed in the roots of the deciduous molars they were developed above the roots. It was easy to see how such a tooth could be developed right or left. Displacement of a premolar externally or internally was not a very uncommon thing amongst animals, and there were quite half a dozen cases in the Royal College of Surgeons Museum.

Mr. STEADMAN said that he had listened with great interest to Mr. Colyer's explanation. It had occurred to him that the teeth might have simply erupted outside the arch owing to some developmental defect. The exact nature of this defect he did not understand. He would have considered it impossible with premolars, owing to their being locked between the roots of the deciduous teeth, but Mr. Colyer's explanation seemed to account for this. He did not quite follow Mr. Thomson's remarks. He did not see how the extraction of a lower tooth could possibly affect the upper in this way: moreover, he had himself removed the lower tooth a long while after the condition had become established.

A Case of Infection of the Cavernous Sinus due to Oral Sepsis.

By GORDON TAYLOR, M.S.

CAVERNOUS sinus thrombosis is by no means a common lesion, and although thrombosis of this sinus has an infective source much more frequently than a traumatic or marasmic origin, yet the mouth does not appear to be one of the more frequent channels of invasion, even in cases of the infective type of thrombosis; indeed, in only 7·7 per cent. of a series of 182 cases collected by Dwight and Germain in 1902,[1] was the oral cavity the starting point. This case would therefore appear worthy of a note as a contribution to the literature, and my object in bringing it before your notice to-night is to remind you of one of the more uncommon results of oral sepsis.

H. H., a male, aged 30, was admitted in the early morning of January 8, 1911, into the Middlesex Hospital, under the care of Sir Alfred Pearce Gould, for whom I operated and to whose courtesy I am indebted for permission to publish the notes of the case. The man was in an unconscious condition on admission, and totally unable to furnish any account of himself; the following details, therefore, were obtained from his doctor and his relatives. On December 26, 1910, that is, about a fortnight before his arrival in hospital, the patient had consulted his medical man for neuralgia, from which he had been suffering for some weeks. The neuralgia, which was most severe on the right side of the head and face, was attributed to the unhealthy condition of his mouth by the doctor, who discovered that he had been previously fitted by some dentist with an artificial dental plate over a number of filthy stumps in the upper jaw, which had never been extracted. The patient was accordingly instructed to consult another dentist, and on December 31 and January 2 some of the stumps are said to have been removed under gas anæsthesia. The neuralgia, however, improved but little under this treatment, and he began to suffer from violent headache, which was almost constantly present. There was but little fever, but he was unable to sleep. On January 4, that is, four days before admission, the right eyelids became swollen and œdematous; the left eyelids showed no signs of œdema till January 7. On January 5, 6 and 7, he was so ill that he remained in bed in the morning, but about midday his condition always seemed to improve a little, and he would get up and was able to talk to his friends. On January 7 he had much more severe pain in the head, and the slightest attempt to move the latter occasioned great agony; during the afternoon, some ten hours before being brought up to the hospital, he became suddenly unconscious, and as the evening advanced the stupor deepened and his general condition appeared to his friends to be becoming worse every hour. He was admitted about 1 a.m. on January 8.

---

[1] *Boston Med. and Surg. Journ.*, 1902, cxlvi, p. 456.

The patient was a well-nourished man, and it was noticed that as he lay in bed his head was drawn down and back towards the right shoulder, and any attempt to straighten it appeared to cause him much pain. The temperature and the pulse-rate showed little alteration from the normal; there was marked œdema of both right and left conjunctivæ, and some proptosis of both eyeballs. The globes did not move, and the corneal reflex was absent; the pupils, which were midway between contraction and dilatation, reacted very sluggishly to the stimulus of light. An ophthalmoscopic examination revealed no change in the optic disks, but the retinal vessels were tortuous and wiry. The limbs were not paralysed, the knee-jerks were brisk and equal on both sides, and a flexor response was elicited on plantar stimulation. There was an absence of any discharge from the nose or ear, and no tenderness over the frontal sinus or mastoid antrum could be found. The mouth was in a filthy condition, marked pyorrhœa being present, and there were several filthy stumps in the upper jaw, especially on the right side, and it seemed as if the carious teeth, which were supposed to have been extracted, must simply have been broken across. There was no obvious bare bone exposed beneath the periosteum, but pus could be squeezed from round the dental sockets. The cardiovascular and the respiratory systems seemed healthy, and there was no abnormality in the specimen of urine withdrawn by catheter from his bladder.

The diagnosis of bilateral cavernous sinus thrombosis was sufficiently obvious, but unfortunately I did not connect in my own mind the oral sepsis with the intracranial lesion. Had I recognized the causal relationship between the two conditions, I should have refrained from any operative interference; but finding no obvious lesion in face, lip, nose, or ear to account for the thrombosis, I took the view that the latter might be due to the pressure on the sinus of some tumour in the middle fossa of the skull in the region of the Gasserian ganglion and the divisions of the fifth cranial nerve connected with it, and that the involvement of these structures by the supposed neoplasm had produced the neuralgia. The pulse and temperature showed little alteration from the normal at the time when the patient was first seen, and appeared to me to rather negative the diagnosis of an infective thrombosis. These data, and the faulty conclusions which I had drawn from them, together with the entreaty of the relatives that I would do something for the man, since he was getting rapidly worse every hour, induced me to perform a decompressing operation with a view to diminishing the supposed increase of intracranial pressure.

Under very light chloroform anæsthesia a sub-temporal decompressing operation was performed, a large area of bone being removed on one side. The only point of interest about the operation was the exceedingly free hæmorrhage which took place from the meninges and diploic tissue of the cranial bones, which proved somewhat troublesome to arrest. A few hours after the operation the œdema of the eyelids appeared to be less marked, but the temperature rose to 102° F., and then assumed an intermittent type; the pulse increased to 112, and then varied in its frequency between 96 and 200; the temperature just before death, which occurred some thirty-six hours after the trephining, reached 107·6° F. At no time was consciousness recovered.

Post-mortem examination: On examination of the brain a purulent basal meningitis was found to be present; the exudate, which was of a yellowish-green colour, was situated over the inferior aspect of the anterior pole of the right temporo-sphenoidal lobe, and extended into the Sylvian fissure and across the anterior perforated spot towards the left cerebral hemisphere. The exudate had also extended backwards towards the posterior part of the base of the brain, but had not spread on to the convexity of the hemisphere, and no tubercles were visible.

Both cavernous sinuses were found to be thrombosed, and purulent grumous material could be squeezed from them, and from one to the other along the two intercavernous channels, which together constitute the circular sinus. Each orbit was examined, but no suppuration discovered there, and an investigation of the tympanic cavity, the nasal fossa, ethmoidal cells, antrum of Highmore and sphenoidal sinus on either side, failed to reveal any focus of suppuration. On removing the right great wing of the sphenoid from the cranial aspect, and exposing the right zygomatic fossa, an abscess was found containing about 2 dr. of greenish-brown pus, and just at a junction of the great wing and the sphenoid body the bone appeared to contain pus between the osseous cancelli. No sign of suppuration was present in the left zygomatic fossa. The mouth was in an extremely foul condition; several dirty stumps of teeth were present in both jaws, especially in the upper molar region on the right side. Several small pyæmic abscesses were found on examining the lungs; these numbered about six in all, and were found lying in the visceral pleura. They were fairly evenly distributed, no lobe being free from an abscess.

Bacteriological examination: A Gram-positive staphylococcus was obtained in films from the teeth. From the brain, pterygoid abscess and cavernous sinus Gram-positive and Gram-negative bacilli were obtained in films, and on culture the *Bacillus pyocyaneus* was identified. A *Staphylococcus pyogenes aureus* grew from cultures from the teeth.

The post-mortem findings would therefore seem to point to the infection taking place in the right upper molar region, and spreading by the posterior superior dental veins to the right pterygoid plexus; and that from the latter the infection took place by way of the emissary veins to the right cavernous sinus, as evidenced by the history that the right eyelids were the first to suffer, and that in three days it spread to the left side by way of the circular sinus. The absence of any suppurative focus in the left zygomatic fossa also supports the view that the left cavernous sinus was only secondarily involved. The termination of the case in a meningitis, and in the development of pyæmic abscesses in the lungs, is a very characteristic feature of the condition.

In some of the surgical text-books no mention at all is made of oral sepsis as a possible cause of infective thrombosis of the cavernous sinus; nevertheless, a fair number of cases are now on record in which this state of complication has followed infective conditions of the oral cavity. Dwight and Germain, in 1902, collected 182 cases of infective thrombosis, in thirty-four of which the channel of infection appeared to be the mouth, and in 1904 Roth added another case, in which the cause was a carious tooth in the left molar region of the upper jaw. Your own Secretary, Mr. Warwick James, collected several cases, which he published in the *Royal Dental Hospital Gazette* of December, 1910, and believes that in many of these cases the infection arises in connexion with the third mandibular molar. In the *Lancet* of November 12, 1910,[1] Mr. S. W. Milner reports a case of thrombosis of the cavernous and circular sinuses, in which the infection commenced in an alveolar abscess round a decayed tooth in the left lower jaw.

As regards the bacteriology of this particular case, it is a matter of regret that the same organism was not found to be present in all the lesions—teeth, pterygoid region, cavernous sinus, and membranes. It is well known that the *Bacillus pyocyaneus* is frequently present in cases of intracranial sinus thrombosis, but whether it was the primary infective organism in this case is not proved, since it was not recovered from either films or cultures taken from the teeth.

Other organisms found in cases of cavernous sinus thrombosis have been a *Staphylococcus aureus*, found in cases due to the spread of infection from a furuncle (Nonnes, Cabarnes, and Axenfeld) ; Villard found streptococci in a case in which the portal of entry was a facial carbuncle. Again, a *Staphylococcus aureus* was found by Mitvalski in a case due to extension of erysipelas, when a streptococcus would rather have been expected. Pneumococci and pneumobacilli have been found in cases due to extension of the infection via the pterygoid plexus from an inflamed tonsil.

DISCUSSION.

Mr. JAMES was particularly interested in the paper because he had for some time tried to follow up some of the cases of cavernous sinus thrombosis. It seemed to him that a large number that had arisen from the mouth had not been recognized, and that it would probably be found that a greater proportion of cases arose in connexion with the mouth. In a post-graduate lecture he gave at the Royal Dental Hospital, referred to by the writer of the paper, he tried to point out that the difference in the character of the tissues behind the third mandibular molar, or associated with the third molar, and that of the other part of the mouth where teeth were situated, was such that infection was much more likely to spread into the pharynx than it was where the infection had occurred further forward. The laxer tissue, and therefore the veins in that region, were much more likely to become involved and a septic thrombosis set up, with extension to the cavernous sinus. In the recorded cases it was very difficult to select those due to infection from the mouth. The history given was very imperfect, and in some cases they had been regarded as idiopathic. Occasionally reference had been made to some trouble in the mouth, sometimes in the region of the third mandibular molar. It was possible such cases might be much greater in number than was generally supposed and from the figures given.

---

[1] *Lancet*, 1910, ii, p. 1416.

Mr. STANLEY MUMMERY asked whether it was possible to get phlebitis of the pterygoid plexus and cavernous sinus which did not go on to thrombosis? He remembered a patient whose lower wisdom teeth were erupting on each side; it was an exceedingly difficult eruption, there was very considerable œdema and the patient was seriously ill, with a temperature of 104° F., being delirious for more than twenty-four hours. The mouth was in a very septic state, although there were no septic teeth present. After repeated lancing and spraying out the mouth continuously with antiseptic solutions, the temperature went down and the case ended quite satisfactorily. He would like to know whether such a case could be accounted for by phlebitis spreading from an erupting tooth.

Mr. GORDON TAYLOR thought Mr. James was right in believing the cases were more common than would appear. Until he had occasion to deal with the case described, he regretted his ignorance of the connexion between an infected cavernous sinus and oral sepsis. With regard to the possibility of recovery of a case of infection of the pterygoid plexus and cavernous sinus, he would think it was quite possible for infective thrombosis of the pterygoid plexus to take place and yet the patient recover: but once the infection made its way along the veins to the cavernous sinus, he thought the patient must certainly die. Thrombosis of the pterygoid plexus alone was quite compatible with recovery, and possibly that was the condition in the case referred to by Mr. Mummery.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FOURTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1910-11

OTOLOGICAL SECTION

1805-1834
1907

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1911

Otological Section.

OFFICERS FOR THE SESSION 1910-11.

President—

A. H. CHEATLE, F.R.C.S.

Vice-Presidents—

P. WATSON-WILLIAMS, M.D.
HUGH E. JONES.
W. JOBSON HORNE, M.D.
W. MILLIGAN, M.D.

Hon. Secretaries—

H. J. MARRIAGE, F.R.C.S. (*Senior*)
SYDNEY SCOTT, M.S. (*Junior*).

Other Members of Council—

ALBERT A. GRAY, M.D.
T. MARK HOVELL, F.R.C.S.Ed.
W. H. KELSON, M.D.
RICHARD LAKE, F.R.C.S.
EDWARD LAW, M.D.
HUNTER F. TOD, F.R.C.S.
F. H. WESTMACOTT, F.R.C.S.
A. L. WHITEHEAD, M.B.

Representative on Library Committee—

HENRY J. DAVIS, M.B.

Representative on Editorial Committee—

J. DUNDAS GRANT, M.D.

OTOLOGICAL SECTION.

CONTENTS.

October 21, 1910.

PAGE
Discussion on Syphilis in Relation to Otology. Opened by Arthur H. Cheatle, F.R.C.S. 1

November 18, 1910.

By Arthur H. Cheatle, F.R.C.S.
- (1) Congenital Malformation of the Auricle and Meatus 25
- (2) Healed Fracture of the Right Temporal Bone in which the Line of Fracture in the Meatus can be seen 26
- (3) Nerve Deafness in a Syphilitic of Thirty Years' Standing 26

By H. J. Davis, M.B.
- (1) Specimen of Temporal Bone from a Case of Thrombosis of Cavernous Sinus... 27
- (2) Extradural Cerebellar Abscess tracking through the Jugular Foramen into the Neck, Sloughing of Middle Third of Internal Jugular Vein; Operation; Recovery 28

By Sydney Scott, M.S.
- (1) Lateral Sinus Thrombosis (Streptococcal). with Early Pulmonary Metastasia; Recovery 30
- (2) Retrocerebellar Abscess; Autopsy 34

Infiltration of both Auricles (? Chronic Lymphangitis) in a Woman, aged 32. By Dan McKenzie, M.D 37

By C. E. West, F.R.C.S.
- (1) Chronic Dermatitis of the Pinna due to Infection by *Staphylococcus aureus*, under Treatment by Vaccine 38
- (2) Chronic Infective Labyrinthitis 39

The Brain of a Patient who died from Cerebellar Abscess. By W. H. Kelson, M.D. 40

PAGE

Large Tuberculous Polyp removed from the Middle Ear of an Infant, aged 7 months. By H. J. Marriage, F.R.C.S. 41

Deformity of Left Auricle. By G. Seccombe Hett, F.R.C.S. 42

January 20, 1911.

The Bacteriology of Chronic Post-nasal Catarrh: A Preliminary Study from Fifty Cases. By C. Ernest West, F.R.C.S. 43

Case in which Disease in a Large Sinus Tympani caused Persistence of Discharge after the Radical Operation; Caloric Tests applied to the exposed Inferior Crus of the Posterior Semicircular Canal. By Arthur H. Cheatle, F.R.C.S., and G. J. Jenkins, F.R.C.S. 54

Exostoses in the Middle Ear. By Arthur H. Cheatle, F.R.C.S. 56

Lateral Sinus Thrombosis following Acute Otitis Media in a Boy; Operation; Recovery. By W. J. Chichele Nourse, F.R.C.S.Ed. 56

Acute Inflammation of the Mastoid treated by Bier's Method. By G. C. Cathcart, M.D. 58

Unilateral Deafness, probably Complete, associated with Vertigo. By Dan McKenzie, M.D. 60

By W. Milligan, M.D.
  (1) Two Cases of Cerebellar Abscess 61
  (2) Suppuration of the Labyrinth; Facial Paralysis; Complete "Bridge" Operation; Recovery 63
  (3) Chronic Otitis Media Suppurativa; Necrosis of External Semicircular Canal; Fistula full of Bone Granulation Tissue; Operation; Recovery 65

February 17, 1911.

Report on the Fatal Termination of a Case of Deafness and Discomfort in the Right Ear, as Early Symptoms in a Case of Epithelioma originating near the Right Eustachian Tube. By Edward Law, M.D. 67

By H. J. Davis, M.B.
  (1) Caries and Necrosis of Temporal, Parietal, and Occipital Bones following Mastoiditis due to Scarlet Fever in a Girl, aged 5; Operation; Recovery 70
  (2) Extradural Cerebellar Abscess tracking through the Jugular Foramen into the Neck 71
  (3) Acute Vertigo, with Repeated Falls always to the Left, during the last Two Months... 71

Fistula of the Ampulla of the Superior Semicircular Canal on the Left Side. Bilateral Radical Mastoid Operation; Instrumental Exploration of the Left Vestibule; Total Vestibular Ablation-reactions (Left Side); Cochlea unaffected. By Sydney Scott, M.S. 73

New Growth of Unknown Nature in the Tympanum. By W. H. Kelson, M.D. 77

March 17, 1911.

PAGE
Injuries of the External and Middle Ear. By W. Milligan, M.D. 79

Injuries of the Middle and Internal Ear. By C. A. Ballance, M.V.O., M.S. ... 87

Fifteen Specimens of the Temporal Bone, showing Types of Fractures. By Arthur H. Cheatle, F.R.C.S. 107

Specimens of Fractured Temporal Bone. By G. J. Jenkins, F.R.C.S. 107

May 19, 1911.

Deafness due to Occlusion of Eustachian Tubes by Scar-tissue. By D. R. Paterson, M.D. 109

By Arthur H. Cheatle, F.R.C.S.

(1) Erysipelas of the Head: Invasion of the Right Auditory Meatus, where an Abscess formed with extension backwards to the Sterno-mastoid Muscle, and Invasion of the whole Sheath of that Muscle 111

(2) Three Specimens of Chronic Middle-ear Suppuration in each of which the Opposite Side was Normal, the six Bones being all of the Diploëtic Infantile Type 112

Periotic Bones of Fossil Cetacea. By W. Jobson Horne, M.D. 114

Labyrinthine Vertigo (Menière's Symptoms—Non-infective) treated by Operation. By G. J. Jenkins, F.R.C.S. 116

By G. N. Biggs, B.S.

(1) Leprosy with Involvement of the External Ears, Palate, Pharynx, &c. 120

(2) Syphilitic Disease of the Nose treated with "606" 121

Notes on a Case of Temporary Deafness and Blindness due to Intestinal Toxæmia. By A. L. Whitehead, B.S. 121

By F. H. Westmacott, F.R.C.S.

(1) Sequestration of Bony Labyrinth in a Boy, aged 4 123

(2) Eight Specimens of the Otic Element of the Arctic Whale 124

(3) Cast of Cavity in the Left Mastoid Process of a Woman, aged 75, filled with Cholesteatoma—Smooth Polished Surfaces 124

Eight Cases of Mastoid Operation. By Charles J. Heath, F.R.C.S. 125

Chronic Suppurative Mastoiditis, with Abscesses in the Left Temporo-sphenoidal and Frontal Lobes. By P. Watson-Williams, M.D. 134

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

LONDON:
JOHN BALE, SONS AND DANIELSSON, LTD.,
OXFORD HOUSE,
GREAT TITCHFIELD STREET, OXFORD STREET, W.

Otological Section.

October 21, 1910.

Mr. ARTHUR H. CHEATLE, President of the Section, in the Chair.

Syphilis in Relation to Otology.

An Address Introductory to a Discussion on the Subject.

By ARTHUR H. CHEATLE, F.R.C.S.

INTRODUCTION.

IT seemed, at first, that to open a discussion on the subject of "Syphilis in Relation to Otology" would be an easy task, but I have been pulled up short repeatedly by my ignorance of many really essential points, not only with regard to precise pathology but also as to the parts involved.

A great advance has been made of late years in our knowledge of syphilis, the discovery of the *Spirochæta pallida* as the direct cause and the Wassermann test being illuminative; but there is still a very great deal to be learned, especially with regard to the minute pathology of the later stages.

Difficulty always arises as to the demarcation between the secondary and tertiary stages; this will continue as long as the description of the disease is based on clinical and not on true pathological findings. The passage of the disease through the mother to the child seems to produce a change in the disease, for in its congenital form the disease appears to differ from the acquired in the selection of parts most frequently affected and in the way in which they are affected. The failure of mercury and iodide of potassium in the treatment of some of the eye and ear manifestations points the same way.

The subject of discussion is such a large one that I cannot enter into many details, and I have left many gaps through sheer ignorance.

The subject is considered under the two main headings—(I) Congenital and (II) Acquired. Each of these is dealt with as the disease affects

(*a*) The external ear, including the cuticular layer of the membrane.

(*b*) The middle-ear tract.

(*c*) The perceptive portion, which is subdivided as far as is possible into (i) the labyrinth, (ii) the nerve, and (iii) the centre.

In the Acquired (*a*) (*b*) and (*c*) are considered in the (*A*) Primary, (*B*) Secondary, and (*C*) Tertiary stages.

(I) CONGENITAL.

I found it impossible, to my own satisfaction, to divide the lesion into secondary and tertiary stages. These are therefore described together under the three anatomical divisions.

(*a*) *The External Ear.*

Personally I have not seen many lesions in this situation, but Mr. F. C. Madden, of Cairo, where the disease is rampant, describes in a paper, "Syphilis in Egypt," the occurrence in infants of "general desquamation and deep, punched-out ulcers on various parts of the body, especially on the ears." Condylomata are described by various authors as occurring on the auricle and in the meatus.

(*b*) *The Middle-ear Tract.*

Eustachian obstruction occurs in the "snuffling" stage in ulceration of the nose and nasopharynx, and in the resulting atrophic nose. I have never seen a case of post-ulcerative obstruction or stenosis of the Eustachian tube; indeed, ulceration is decidedly very rare in the nasopharynx—it is more common in the nose. I have seen necrosis of the bony septum in a boy aged 7½, but the ulceration did not extend to the lateral walls of the nasopharynx.

An obstinate case of tubal obstruction was present in a middle-aged lady who had lost all the internal structure of her nose, except the septum, in childhood. The nose was consequently very dry, and the Eustachian tubes were involved in the dry condition.

Inflammation of the middle ear may occur in the "snuffling" stage in nasal ulceration and in the atrophic state, but these syphilitic conditions alone do not, as far as I know, cause suppuration, but they are a predisposing cause, the introduction of a pyogenic organism being necessary.

Very rarely the tract is involved in a gumma. Madden, in his paper, describes a case.

A child was admitted into the hospital for chronic mastoid disease. An operation had been performed in the country some time previously, and on admission there was a deep, unhealthy opening, discharging thick, caseous material, behind the ear. A further operation was performed, and a large quantity of caseous matter and carious bone removed—probably tuberculous, it was thought. Ten days after the operation some bleeding occurred, and the child died after repeated hæmorrhages from the lateral sinus, a gush of cerebrospinal fluid appearing just before death. Post mortem the condition was found to be syphilitic, the remains of large gummata involving the petrous portion of the temporal bone being quite conclusive. Various gummata scattered through the dura mater and the parietal and occipital bones were also found.

(c) *The Perceptive Portion.*

I may at once state that I do not know of any evidence to prove that there is ever a syphilitic lesion in the fœtus which causes severe or complete deafness at birth. Deafness causing deaf-mutism in congenital syphilis is due to a lesion in early childhood, and even then the disease has been greatly overrated as a cause of deaf-mutism. Kerr Love, in his book, gives a table of the causes of deaf-mutism in 1,410 children from various parts of the country; in only two is syphilis given as a cause, and they are both returned as occurring in the Glasgow Institution, where there were 173 inmates. The table may not be strictly accurate, but it serves to show how rarely congenital syphilis is a cause of deaf-mutism.

There is no proof, to my mind, that otosclerosis is due to syphilis, congenital or acquired, although I am aware that there are differences of opinion.

The Common Type of Congenital Syphilitic Deafness.

Sex.—There seems no doubt that the female is more prone to this form of deafness than the male. Sir Jonathan Hutchinson states, when discussing this part of the subject in the *Archives of Surgery*, July, 1897,[1] that "it is difficult not to suspect that some unrecognized law as regards hereditary transmission underlies these remarkable facts."

Age.—No fixed age for the onset of congenital syphilitic deafness can be given, although it is most frequent between the ages of 6 and 16. It may occur earlier than the sixth year and it may be much later than the sixteenth. Not uncommonly the trouble begins between the ages of 20 and 30, and even much later. A woman, aged 54, got very deaf in both ears in two months, having been slightly deaf in the left ear for two years; she had signs of keratitis in the left eye, which she said had been "bad" at the age of 8. Another woman, aged 64, stated that her deafness did not come on until she was aged 55; signs of keratitis, from which she had suffered in childhood, were present in both eyes.

Modes of Onset.—From the clinical point of view these may be divided into those commencing more or less insidiously, first in one ear and then in the other, with tinnitus but without giddiness, and those in which giddiness is a very marked feature and the onset of deafness often extremely rapid. Occasionally, however, the onset is without giddiness; then, after a long period, the hearing becomes much worse, with attacks of giddiness. The cases beginning without giddiness are so well known that it is superfluous for me to give a description. I shall only say that both ears are always affected sooner or later, and this is to me a curious fact and incapable of explanation.

Though very common in the congenital form, onset without giddiness is very rare in the acquired.

That giddiness may be a marked feature was pointed out by Dr. Urban Pritchard and myself in 1898 in the *Archives of Otology*, xxvii, p. 415, and some of the cases there related are worth describing again, together with others.

The giddiness with vomiting may be extremely acute and complete and lasting deafness may result in a few hours; at first the attack is usually thought to be an ordinary bilious one until the deafness is noticed.

A boy, aged 15, was brought on September 16, 1896, on acount of complete deafness in the right ear, the history being that six weeks previously he had been suddenly attacked by severe giddiness and vomiting, the former lasting a week, the latter two days; on the third day deafness was noticed in the right ear and had remained absolute ever since. The father was doubtful as to whether he had had syphilis. The patient was the eldest of three; he had suffered from "bad eyes" a short time before examination, but the trouble was stated by an eminent ophthalmic surgeon to have been more of an ordinary phlyctenular than of a syphilitic nature. For four or five years the boy had suffered from general enlargement of the lymphatic glands, especially of those in the neck, for which he had undergone various treatments without much benefit. There was no facial paralysis, loss of taste, nor headache. On January 13, 1897—that is, four months later—swelling and ulceration of the left tonsil and faucial pillar were found. Under mercury and iodide of potassium the throat condition rapidly cleared up and the glands greatly diminished. This was one of the rare cases in which only one ear was affected, but the later history is unknown.

---

[1] *Archives of Surgery*, 1897, viii, p. 283.

Mr. Fagge showed a girl, aged 21, at the Otological Society on May 7, 1900.[1] She had become quite deaf in both ears in a few days after a "bilious attack." The eyes were affected, and her mother had had four miscarriages immediately before her birth.

C. A. S., a female, aged 11, suddenly became deaf in both ears during attacks of giddiness. The eyes had been "bad" two years previously, and evidence of keratitis was present in both eyes. This case will be referred to again in dealing with treatment.

The giddy attack may be subacute in character.

C. D., a male, aged 28, presented himself on account of extreme deafness, the history being that at the age of 18 the deafness had become complete in two months during marked attacks of giddiness.

The giddiness may run a chronic course.

A. G., a male, aged 11, was first seen on December 14, 1896. He was the second of four, the other three being perfectly healthy. There was no history of miscarriages or still-births. Deafness in both ears was first noticed in the preceding August. The onset was associated with attacks of headache and giddiness, which were worse on getting up in the morning; occasionally vomiting occurred with the attacks. His mother had often seen him staggering towards the left when walking. When giddy he sometimes fell forwards, striking his nose or forehead, the ground appearing to rise up and hit him; objects about him appeared to be moving to one side or the other, more frequently from left to right. He had had two or three attacks during the preceding month. He complained of humming "like an engine at work" in the right ear. On examination he was a bright, healthy-looking boy, relating his symptoms quite clearly. He had never had bad eyes, and the only sign of congenital syphilis was very slight pegging of the left central incisor tooth. A two-foot watch was not heard on the right side, and only on touch on the left. All tests showed internal ear deafness; giddiness could be elicited by turning the head sharply to the left. On May 13, 1897, five months later, active keratitis was present for the first time, so helping to demonstrate the true nature of the case. He had been blistering behind the ears since his first visit. On April 21, 1898—that is, about a year since the last visit—he again came for examination, having been in the country for five months. Blistering had been continued until a month previously. The left ear had improved markedly, the watch being heard at 2 in. and low conversational voice at 2 ft.; on the right side shouting was heard close to the ear, but the watch was not heard. Slight occasional vertigo was still complained of. The active eye trouble had ceased.

---

[1] *Trans. Otol. Soc. U.K.*, 1900, i, p. 59.

The case is of interest, for the ear preceded the eye troubles—an unusual thing—by one year and nine months. The vertiginous attacks extended over a long period, and the blistering appeared to produce improvement, especially on the left side

A mixed mode of onset is illustrated in the following cases:—

A man, aged 31, stated that six years previously he had suddenly become deaf in both ears, but got quite well; three years previously he had had an attack of giddiness and vomiting, and had gradually got deaf since. On examination evidence of keratitis was present in the right eye; the teeth were characteristic; internal-ear deafness was present, and he could hear only fairly loud shouting on the left side and not at all on the right.

A woman aged 27, who had been very deaf for four years, stated that she had a giddy attack, in which the room went round, with vomiting after the deafness had started. She had had keratitis five years previously.

It is curious that a remnant of hearing is usually present, even in the very bad cases, and whatever their mode of onset may have been, when they appear to have reached finality. It seems that the trouble may occur early in life, leaving a certain amount of useful hearing in one ear, and then later in life, without any apparent cause, it may reappear suddenly and destroy the remnant.

Site of the Lesion.—I think that the labyrinth is the part affected in both varieties of onset. In some cases, at all events, nystagmus is present, but it must not be mistaken for the coarse irregular movements of the eyeballs due to the eye lesion. I have never seen the facial nerve implicated, nor have I seen great headache nor temperature suggesting meningitis. There was absence of vestibular reaction to cold in the few cases in which I have tried it, if the deafness was complete. In a girl who was quite deaf on the left side and had a remnant of hearing on the right, the reaction was absent on the left side and quite brisk on the right. I have not employed the other methods of testing.

Nature of the Lesion.—One cannot help thinking that the pathology of the cases without giddiness must differ from those in which giddiness is a feature. A somewhat slow onset without giddiness suggests the osteitis found by Walker Downie; the onset with giddiness suggests an exudation, but this has never been proved. It is possible that both osteitis and exudation may exist in the same case. Post-mortem examinations have been very few. The best is that of Downie, and is recorded by Kerr Love in his book on deaf-mutism. It was the case of a boy aged 17, whose hearing began to fail during recovery from eye affection, and who became deaf in six months, his last remnant of hearing leaving him suddenly. Death occurred as the result of a breaking-down gumma of the right parietal bone. (Downie makes no mention of giddiness as a symptom.) The right temporal bone was examined, and the following is the result of the examination. The base of the stapes, incorporated with or ossified to the border of the foramen ovale, was immovably fixed to the inner wall of the tympanum. The internal auditory meatus at its inner extremity was of average normal size and calibre; both the auditory and facial nerves contained therein were healthy; but, tracing the meatus outwards, at a distance of 1 cm. from the inner opening, the upper wall became suddenly thickened, encroaching on the canal; and at a further distance of 3 mm. the canal was almost obliterated. The vestibule was so greatly encroached upon as to make it doubtful whether any of the space remained. The cochlea was easily examined, and was of average size, but the modiolus and lamina spiralis ossea were so thickened as to occupy an unusually large proportion of the cochlea. Of the semicircular canals only a trace could be found, the remaining portions of this part of the labyrinth being lost in a mass of dense bone of ivory hardness. The immediate cause of the deafness was thought to be the partial destruction of the labyrinth and pressure on the trunk of the auditory nerve.

Associated Syphilitic Lesions.—The most constant are those of the eye; of these, interstitial keratitis is the most common. I think that any ulceration of the cornea should always be regarded with great suspicion. In one case recorded above, the eye trouble had been called phlyctenular by an eminent ophthalmic surgeon, and the real condition was altogether missed. One eye only may be affected. It is not sufficient to assume that congenital syphilitic eye trouble is not nor has been present because there are no signs of keratitis; the trouble may lie in the choroid, &c.; so a thorough ophthalmic examination should always be made in doubtful cases. The eye troubles usually precede those of the ear, but they may occur a long time after, or they may be absent all through life. The teeth are usually characteristic, but are not infrequently perfectly normal. Scarring at the angle of the mouth may, or may not, be present. Tertiary ulceration of the fauces or nose is occasionally seen.

A "diffuse gummatous knee" was associated in one case in which giddiness was a marked feature and the onset of deafness rapid. General enlargement of the glands was co-existent in a similar case. Walker Downie found at the post-mortem examination of his classical case a gumma of the parietal bone; indeed, it had caused death; cicatrices in the liver and highly amyloid condition of the thoracic and abdominal organs were present also.

I have no doubt that the list can be further extended.

Diagnosis.—This is in the great majority of cases quite easy, but the true nature of those with giddiness is sometimes overlooked. Very rarely all other signs may be absent, or they may be only slightly marked; but internal-ear deafness, with or without giddiness, coming on without any other ascertainable cause between the ages of five years and twenty-five years, is pretty sure to be due to congenital syphilis. Wassermann's test can be used to help us. In the few cases—about ten—of undoubted progressive congenital internal-ear deafness examined or me by Dr. Emery, it has been positive. In those cases which have reached finality, one would expect it to be negative; that is, if no other lesion is in progress.

Treatment.—In my experience mercury and iodide of potassium are powerless, even in the early and progressive stages, and when Wassermann's test clearly shows that active syphilis is present. The failure is inexplicable to me; for, granted that a slight lesion in the perceptive part may quickly produce a profound and lasting deafness, one would expect that progressive deterioration would be arrested by the exhibition of these drugs. There is certainly some mystery in the pathology of these cases. Sydney Stephenson, in dealing with the constitutional treatment of interstitial keratitis, speaks of the failure of mercury and iodide of potassium ; but he found that some cases became more tractable when the extract of thyroid gland was given with them, and the same with atoxyl. We have yet to learn if Ehrlich's "606" is of any use. It is reported that it occasionally produces blindness, which would be a terrible complication for one of these patients. Repeated blistering behind the ears, recommended by Dr. Urban Pritchard, has given the best results in my experience. The sooner it is applied the better chance there is for checking or improving the disease. It is useless if the deafness has been stationary for about two years. It should be continued for months, or even years, as long as even slight improvement takes place. Pilocarpine sometimes does good in the early stages, and I think it should always be tried for about three weeks, but blistering should be carried out at the same time.

The patient, A. S., aged 11, quoted above, was admitted into the hospital a fortnight after the rapid onset of profound deafness with giddiness. Pilocarpine was given hypodermically every day for a fortnight without any improvement whatsoever; it was then stopped and repeated blistering was adopted, with the result that at the end of another three weeks she was immensely improved, and left the hospital hearing ordinary conversation quite well, whereas on admission it was necessary to use a slate. I have had this patient under observation for twelve years; she still has fairly good hearing, but occasionally she feels slightly giddy and more deaf, and at once she comes asking for the blistering fluid, and it has so far never failed to relieve her. She was not tested with mercury and iodide of potassium. I saw her a few weeks ago on one of her periodical visits, and took the opportunity of having a Wassermann done, with a positive result.

The best treatment of all is, of course, prevention. I must not touch on the large question of the stamping out of the disease, nor even of the time at which marriage is safe; but I cannot help thinking that the onus must lie with those who have charge of the congenitally syphilitic infant. I would ask, if treatment were undertaken in infancy and carried on for two years, or, under guidance of a Wassermann until the test was negatived, whether the later lesions could not be prevented? Some years ago I carefully inquired of the mothers of congenital syphilitic children with internal-ear deafness whether treatment had been applied in infancy, and invariably the answer was in the negative.

Inflammation may spread to the labyrinth from a middle-ear which has become infected indirectly through the disease. The petrous portion may be involved in a gumma, as in Madden's case, recorded above.

(II.) ACQUIRED.

(*A*) *Primary.* (*a*) *The External Ear.*

I have never seen a case of chancre in this position, and Politzer only mentions four in his book.

(*b*) *The Middle-ear Tract.*

I have not seen or heard of a chancre involving or affecting the middle-ear tract. The greatest chances of such a thing occurring are by direct infection by instruments or the finger of the surgeon.

(*B*) *Secondary.* (*a*) *The External Ear.*

The auricle shares in the rash with the rest of the skin. A papular infiltration on the external surface of the membrane has been described by Lang. The post- and pre-auricular glands also share in the usual general glandular enlargement. Condylomata are occasionally seen. Not more than four cases have come under my observation; curiously enough, I saw two in one day at the Royal Ear Hospital.

A man, aged 41, who had had the primary sore for four months, came to the hospital complaining of pain in, and discharge from, the right ear, and a sore throat. The ear had been bad for fourteen days, beginning with pain and tenderness in the outer part. There was a great deal of most offensive discharge lying in the concha and meatus. After the discharge had been cleared away, the passage was seen to be occluded by greyish irregular masses. Improvement took place under general treatment, syringing with lotio nigra and dusting in of calomel, but the condition did not clear up as rapidly as one expected, and, perhaps for this reason, the patient ceased attendance before a cure was effected.

Dr. Dundas Grant showed a case at a meeting of the Section a short time ago, and I should like to hear of the later progress, as these cases are sometimes rebellious to treatment.

I have never seen the commencement of condylomata. Knapp describes them as beginning as "red efflorescences which gradually increase in size and are followed by a diffuse swelling of the meatal walls combined with a moderate amount of secretion. These condylomata, in the form of red or greyish red, ragged, warty excrescences, arise more or less quickly on the secreting parts and extend from the auditory orifice to the osseous meatus, rendering the lumen impermeable."

The ulceration, which is described as occurring in the early secondary stage, I think must be rare, for I have never seen a case; perhaps it is the healing stage of condylomata.

(*b*) *The Middle-ear Tract.*

The Eustachian tube may be obstructed by the specific nasopharyngitis, and the specific infection may spread up the tube to the middle-ear, causing symptoms and signs like those of an ordinary catarrhal inflammation.

I have never seen a case of acute suppuration as a result. I think that a superadded pyogenic infection is necessary for such a result. Nor have I ever seen the *Spirochæta pallida* in the discharge from the middle ear, and I should like to know if others have.

An acute inflammation of the tract, including the antrum and mastoid cells, occurring during the surgical treatment of condylomata has been described by an author, but it is questionable whether the infection was due to the direct spread of the disease or to the surgical measures employed, or to a pyogenic infection from the nasopharynx. The case was that of a boy whose ears were completely blocked with condylomata. The soft succulent masses were more than once removed with a sharp curette and local and general mercurial treatment was adopted. A fresh crop of condylomata appeared, and increased to such an extent that both passages were again entirely occluded. A severe inflammation of the left middle ear, leading to abscess in the antrum, followed. The usual operation for mastoid abscess was performed, the antrum being found full of pus and surrounded by a mass of carious bone. The diseased parts were removed, the meatus scraped out, and drainage provided. The right ear was also cleared of condylomata.

(*c*) *The Perceptive Portion.*

The slight deafness which is described as frequently occurring in the early secondary stage, and always yielding to general mercurial treatment, is probably toxic. The more serious cases are those characterized by attacks of giddiness, vomiting, tinnitus, and deafness—the last often being profound and of rapid onset. Sometimes one ear only is affected. I have seen both ears affected in a soldier in whom the symptoms were very acute, and, although mercury was rapidly and efficiently used, no recovery of hearing took place, the failure being probably due to the fact that the nerve endings were rapidly destroyed.

These cases resemble those already described as occurring in the congenital form, and suggest an exudation into the labyrinth as in the iris. In the slighter cases mercury, if used thoroughly and early, together with counter-irritation by blistering behind the ear, gives very good results. We have yet to learn the effects of Erhlich's "606" in this condition, and, indeed, in all syphilitic aural affections.

The cases of giddiness, deafness, tinnitus, and facial paralysis will be described under the tertiary lesions.

(C) Tertiary. (a) The External Ear.

The auricle and meatus may be the site of gummatous ulceration which may be mistaken for epithelioma. Occasionally marginal ulceration of the helix is seen.

J. E., a man aged 48, who had had syphilis eighteen years previously, had an indolent kind of ulceration of the helix on both sides involving the cartilage and looking as if pieces had been nipped out. Active ulceration of the soft palate and superficial glossitis were also present. The condition soon healed under mercury and iodide of potassium.

I have also seen complete destruction of the tragus.

(b) The Middle Ear.

Eustachian Obstruction.—I have seen Eustachian obstruction due to the presence and irritation of septic discharge from nasal ulceration, and to the dryness and crustation after the destructive process was over. In about three cases I have seen a gumma of the post-nasal space in the "adenoid" region, and they appear to me to form a special class. In all, deafness due to Eustachian obstruction was one of the chief complaints, together with a somewhat rapid onset of nasal obstruction with thick speech and the passing of septic discharge from the naso-pharynx to the pharynx.

A man, aged 42, had had syphilis three years previously, and had been treated for eighteen months with mercury. He came complaining of deafness in the right ear, hoarseness, nasal obstruction, and an uncomfortable throat due to the passage of discharge into the throat from the back of the nose. The deafness was found to originate in the middle-ear tract and was at once relieved by inflation. In the "adenoid" region could be seen an irregular swelling covered with mucous. On examination the mass felt quite firm, and one could imagine that it might have been mistaken for an epithelioma. The lateral walls of the nasopharynx were clear as far as could be determined. The hoarseness was due to swelling of the left ventricular band. Under mercury and iodide recovery took place rapidly. In the others a precisely similar mass was seen and felt in the "adenoid" region; and in one, who at first denied syphilis, the mass felt so hard that in order to exclude epithelioma I removed a piece under chloroform; in this case internal-ear deafness and giddiness were also associated, and will be fully described later on from that point of view.

I have never seen complete Eustachian obstruction due to past ulceration of the nasopharynx.

Complete adhesion of the soft palate to the pharyngeal wall may cause trouble in the ears. Spencer showed a case at the Laryngological Society, November 10, 1897, in which great pain in the ears was complained of.[1]

Mr. Jenkins tells me that in hemiplegia he has found deafness due apparently to paralysis of the tubal muscles.

Tertiary nasal and nasopharyngeal lesions may predispose the tract to infection, with all its attendant dangers; but I cannot be sure of ever having seen acute or chronic suppuration which could be described as syphilitic, although I have seen delayed healing of the wound after the acute mastoid operation in a syphilitic woman. Nor have I even seen a case presenting the signs and symptoms of chronic middle-ear catarrh which could be classed as syphilitic.

Very rarely the tract is involved in a gumma; such a case has not, as far as I know, come under my care.

At a meeting of German laryngologists held in Dresden, May, 1900, M. Castex and M. de Parrel showed a syphilitic of thirteen years' standing, who had rhythmical elevations, 100 per minute, of the soft palate, and, if the palate was fixed, of the base of the tongue; the contractions were accompanied by rhythmical clickings in the Eustachian tube and tympanum on the left side. Castex stated that he had seen a similar case in a syphilitic general paralytic, and it was suggested that the contraction in the patient shown was a prodromal symptom of general paralysis.

---

[1] *Proc. Laryng. Soc. Lond.*, 1897-8, v, p. 4.

(c) The Perceptive Portion.

The nature and site of the lesions in the perceptive portion are not accurately determined by post-mortem examination, but we are able to more than conjecture the latter in most cases, by comparing the symptoms and signs with those produced by suppuration in which the site has been determined by post-mortem findings. We are specially indebted to Dr. Alexander Bruce and Dr. J. S. Fraser for their classical case.

(i) *Labyrinth (probably).*—C. Q., a middle-aged man, had syphilis in 1894. The primary sore lasted six weeks. The secondary manifestations were very slight. He took pills regularly for two or three months, and then on and off, with intervals of one to three months, until 1900. During the whole treatment the gums were only slightly affected. He was quite well until August, 1909, when he had acute pain in the right shoulder for two or three days, the pain shooting down the same arm to the fingers, especially the fore and middle fingers, which also felt numb, the middle finger being the one more affected in this way, and it had remained affected on the palmar surface. Transitory tingling was also noticed over the whole face, and a prickling sensation in the soles of the feet. The right arm was weak for a month, with inability to lift it above the shoulder, but this got quite well without any permanent loss of power or wasting. On February 18, 1910, he came complaining of a high-pitched hissing noise in the left ear, like the escape of steam, with deafness and a feeling of unsteadiness, but no actual giddiness, vomiting, or nausea. These symptoms had existed for three weeks. He stated also that he had neuritis affecting the inside of the ear. On examination, the membranes, nose, nasopharynx and pharynx were normal. There was no nystagmus or inco-ordination. The pupils were equal and reacted to light. The knee-jerks were rather over-active. Numbness of the palmar surface of the right middle finger was present. No facial paralysis or herpes or any history of them.

Conversational voice : $\frac{\text{Right ear, normal.}}{\text{Left ear, 1 ft.}}$ Whisper : $\frac{\text{Right ear, normal.}}{\text{Left ear, 6 in.}}$

A medium C tuning-fork on the vertex and nose was referred to the right ear, and when placed on the left mastoid the sound was conducted across to the right ear. His own voice was heard by himself louder on the right side. He was at once put on a course of mercurial inunction and repeated blistering behind the left ear. Great improvement rapidly occurred; when seen on May 31, 1910, he said that he did not feel deaf unless tested with a watch, that the noises were very slight and occasionally absent, and that the unsteadiness had gone.

Conversational voice :	Right ear, normal. Left ear, normal.	Whisper :	Right ear, normal. Left ear, not quite normal.

The tuning-fork on the nose was still referred to the right ear. In this case the ear symptoms came on sixteen years after the primary infection. I suggest that the lesion was in the labyrinth, the cochlea being the part most affected, but I have an open mind on the subject. The so-called neuritis of the ear is interesting and suggests changes in the pars intermedia of Wrisberg.

A man, aged 47, had syphilis, which was untreated, fifteen years previously. He came complaining of varying deafness in both ears, giddy attacks in which he fell down and was sick, noises in his ears, and recent nasal obstruction with thick speech. There was no facial paralysis or herpes. Although internal ear results were given by the tuning-fork, yet he distinctly improved on inflation, but not by any means to normal. In the nasopharynx was a firm mass in the "adenoid" region, and from the space came a great deal of muco-pus. Syphilis was denied, but the mass was so suspicious that I removed a piece for microscopical examination. Later on, superficial ulceration was seen creeping down the right posterior faucial pillar, and when again taxed with having had syphilis he confessed. Under mercury and iodide the giddy attacks ceased, the post-nasal space cleared, and the hearing very greatly improved, although a certain amount of internal-ear deafness remained. This was evidently a mixed case, some of the deafness being due to the post-nasal conditions and some to the labyrinthine.

A woman, aged 62, had a gumma in the breast in May, 1909; during the previous month she had severe Ménière's attacks. In October, 1909, the attacks became extremely acute and confined her to bed for fourteen days. She also suffered from much headache and pain at the back of her head and neck. Meningitis was excluded by examination of the cerebrospinal fluid. There was no facial paralysis or optic neuritis. Great improvement occurred under mercury inunctions and iodide of potassium. The giddiness ceased, but the hearing, though improved, did not recover completely.

Politzer quotes Moos,[1] who reported the case of a syphilitic individual, aged 37, in whom marked subjective noises, attacks of dizziness and headache, suddenly appeared, which were later followed by disturbances of hearing. At the autopsy he found thickening of the periosteum of the vestibule, the foot of the stapes raised and immovable, the connective tissue between the membranous and bony labyrinth hyperplastic, infiltrated with small cells, Corti's organ and semicircular canals infiltrated with cells, and the acoustic nerve normal.

(ii) *Nerve (probably)*.—A young married woman came on May 7, 1908, complaining of deafness in both ears, the left being the worst, and left facial paralysis. The right ear had been slightly deaf since childhood. She had also necrosis of the superior and inferior maxillæ on the left side, with loss of teeth. A week before Christmas, 1909, what seemed to be a gum-boil appeared in the left upper jaw, a molar tooth became loose, and the left gum receded; then some teeth became loose in the lower jaw, one of them being removed by a dentist. About February 17 she became very ill with giddiness and sickness, the room appeared to whirl round her; the attack lasting a week and confining her to bed. On getting up at the end of the week she noticed noises, like the rushing of sea waves, and deafness in the left ear. Another loose tooth was removed, and three pieces of bone came away. Fourteen days before examination a piece of bone came away from the upper jaw on the left side; she was very weak and swayed on walking. Five days before examination she felt very sick but did not actually vomit. Four days before examination she complained of pain in and behind the left ear, with twitching of the left side of the face, followed by paralysis. For two days everything had tasted "watery." There was marked internal-ear deafness on the left side with complete facial paralysis. There was no herpetic eruption. A circular dusky spot the size of a florin was present on the right side of the chest, and an ulcer was present in the roof of the mouth quite away from the teeth troubles. Under mercury and iodide of potassium the facial paralysis and loss of taste recovered, together with all the other signs; but, although some of the hearing recovered, a great deal of nerve deafness remained. This was probably a case of meningitis involving the seventh and eighth nerves in the internal auditory meatus. The loss of taste is interesting.

A similar condition, but on both sides, occurred in a man, aged 48, who had the primary sore in 1894. He was treated intermittently with mercury for one and a half years. In 1907 he had a node on his forehead, and developed kidney trouble; and during 1908 and 1909 he was in bed for nine months with swelling of his legs. In January, 1909, he became slightly deaf in the left ear, with pain on the left side of his head and facial paralysis; vertigo was not a feature. In September, 1909, after having noticed some slight deafness in the right ear with vertigo for several months, he was seized with very great pain in the nape of the neck and in the right mastoid region, whence it spread up to the vertex. Facial paralysis developed in a few days and became complete. There was no herpes nor optic neuritis. In October, 1909, he was put on mercury and iodide of potassium; the hearing in the left ear improved, but not in the right. The facial paralysis recovered on the left side, but not completely on the right. The kidney condition improved, the swelling of the legs being only slightly present and worse on fatigue. There was no loss of taste in this case.

---

[1] *Virchows Archiv*, Berl., 1877, lxix, p. 313.

The only case of herpetic geniculate ganglion inflammation which I have seen was described by me in the *Transactions of the Otological Society*, 1901, ii, p. 6. The seventh and eighth nerves were involved also; and the pain, which was so marked in the previous case, was also experienced in the nape of the neck and behind the affected ear. In her case the ear symptoms and the facial paralysis came on after the appearance of the herpes; the temporary perversion of taste came last of all. Dr. Milligan and Mr. Ballance thought the case to be one of peripheral neuritis, probably post-influenzal, but there was no history of influenza. Although the patient was a single woman, which made the search for infection a very delicate matter, yet she had pigmented scars on both legs and a smooth ulcer the size of a threepenny-bit on the calf of the left leg, without varicose veins, and recovery occurred under mercury and iodide of potassium, and counter-irritation behind the ear.

I cannot be certain that, apart from tabes, there is a progressive sclerosis of the auditory nerve alone, although I have tried to persuade myself that some cases were of that nature.

It is not uncommon to find internal-ear deafness and tinnitus in tabes; indeed, these symptoms may be the earliest indication of the more general trouble. J. Ramsay Hunt, in the *Journal of Nervous and Mental Disease*, 1909, xxxvi, p. 342, speaks of tabetic otalgia, describing how, in the course of tabes dorsalis, sharply lancinating pains may occur in the depths of the auditory mechanism. He has observed five cases of this nature, in one of which degenerative changes were demonstrated in the pars intermedia of Wrisberg.

(iii) *Centre (probably)*.—I have seen one case of early general paralysis in which deafness and tinnitus—certainly due to changes in the perceptive portion—were complained of. The auditory hallucinations —which sometimes are present in general paralysis—are, I believe, most frequently observed in those patients who have some pre-existing auditory trouble with tinnitus. I have never seen a case in which the hearing centre was involved in a gumma.

The following is a case I do not attempt to classify, and I am not sure that syphilis was to blame. A man, aged 61½, a syphilitic of thirty years' standing, came complaining of alternating deafness in both ears for six weeks or two months, with headache at the back of the head, but without giddiness or facial paralysis. The trouble was certainly in the perceptive tract, according to all tests, and inflation made him worse. He had had two vague brain attacks in which he suffered from loss of memory with regard to recent events and failure in remembering people; from these he had recovered, but with some loss of memory; Wassermann's test was negative.

Effects of Vascular Lesions.—I have not seen a case in which I could diagnose endarteritis of the vessels of the labyrinth or centre, or thrombosis of cerebral vessels; but perhaps some of the cases related above may be due, directly or indirectly, to such conditions. Cases of thrombosis are more likely to come under the care of the neurologists. Politzer quotes the case of Baratoux and Kirchner. Unfortunately, no clinical history is given; but they found a typical syphilitic endarteritis in the blood vessels of the mucous membrane of the tympanic cavity, periosteal lenticular accumulation of bone on the promontory, and the formation of cavities in the wall of the labyrinth identical with the condition found in otosclerosis, as described by Politzer.

I think, gentlemen, I have said enough to show how much there is yet to learn, and how necessary it is that the subject should be discussed.

DISCUSSION.

Dr. URBAN PRITCHARD said the subject was an exceedingly interesting one, especially as so much had yet to be learnt about it. He was glad to hear the President's allusion to the fact that in congenital syphilis the age was not always a limited one. He had himself seen cases which came on quite as late in life as those which the President mentioned, and in one or two the age caused him at first to doubt his diagnosis. Another interesting point was that occasionally the keratitis came on after the internal-ear symptoms, and that might put one off the diagnosis. Probably every member had removed adenoids from young children in whom there were no obvious signs of syphilis, but in whom there was some nerve as well as middle-ear deafness; the removal being followed by no, or only temporary, improvement in the hearing. His view was that almost all those cases were due to congenital syphilis, and in a few of the cases this was shown by the keratitis coming on afterwards. It was many years ago that he first discovered the usefulness of blistering. Sir Jonathan Hutchinson had told him no treatment was of any good for the condition. But soon after that Sir William Bowman sent him a young man who had inherited syphilis, affecting his internal ears, and who was beginning to recover from keratitis. He then thought of trying blistering, and to his great astonishment he not only improved but ultimately became normal. He saw the patient from time to time for some years afterwards, and he occasionally had slight returns, but blistering always restored him. He was very glad to hear the President's remarks on prevention, in other words the early treatment of all the early symptoms in infants, with the idea of preventing internal ear disease. In regard to acquired syphilis, he had always been struck with the infrequency of Eustachian deafness in syphilitic pharyngitis. Another point was that when there were secondary or tertiary affections of the meatus, it was very easy to make a mistake in thinking that the condition was epithelioma. One case had so impressed him that he often quoted it. An aural surgeon brought him a case of disease of the external meatus. Thinking it might be epithelioma, he removed a piece for examination, and sent it to a pathologist. The report was epithelioma, and he therefore suggested complete removal. The patient made arrangements, but a day before, he met a medical practitioner, who disbelieved the diagnosis, and put the patient under mercurial treatment. No operation was done, and twelve months later his friend the aural surgeon met the patient, who was quite well. One did not often see ulceration at the edge of the helix, but every now and then it was seen, and he had recently seen a marked case, now healed, which had been affected on both sides. He had other reasons for diagnosing it as syphilitic, although the man attributed it to frost-bite.

Mr. WEST said that, having had occasion to go through the literature of otology recently, he could say he believed the President had embodied practically everything which was known on the subject. He wished to ask if there was any definite proof, in the late cases of labyrinthine trouble, of the patient being syphilitic; and, even if the patient was syphilitic as a child, that the deafness now was of syphilitic origin. Because a person had an old nebulous cornea and deafness at the age of 56, it was a big jump to regard his case as syphilitic labyrinthitis. He supposed everybody agreed as to the powerlessness of ordinary remedies in congenital syphilis, and a number of the cases one saw had been referred from the ophthalmic departments, where they had been under treatment on account of keratitis for weeks, or perhaps months, before the appearance of deafness. It seemed hopeless to attempt anything with mercury and iodide in those cases. He had never seen a case of chancre of the Eustachian tube, but many cases were given in the literature, over 100 being recorded. A collection made in 1900[1] gave 88 cases, 71 being of French origin. He thought their disappearance coincided with the increasing use of sterilizers, and the disuse of the practice of taking a number of patients in succession. In regard to condylomata, he had been struck with the intense pain they caused. There was a grey, ragged, heaped-up condition of the meatus, so that it was impossible to see the membrane. A case he saw a week ago illustrated tertiary acquired paralysis of the auditory and facial nerves. A woman came to the aural department of St. Bartholomew's Hospital, totally deaf on one side, and with complete facial paralysis on that side. Wassermann's reaction was positive. There was very little doubt about the patient having acquired syphilis five or six years previously. In a fortnight, under mercury and iodide, the facial paralysis had disappeared completely, but the hearing was uninfluenced. He had seen one case of tertiary ulceration of the external ear (meatus and concha) in a woman, and a very extensive scarred case in which the meatus, concha, and tragus had been involved, and there was great stenosis of the meatus. He had seen one case of ulceration of the margin of the pinna, which he believed was syphilitic, but there was no definite evidence of it. He could give one case of auditory hallucination in general paralysis, in which the patient was known to him personally, and he knew there was nothing wrong with his hearing even to a late stage in the disease.

Mr. A. L. WHITEHEAD said that he would have thought that interstitial keratitis would not be so unusual a sequel to an aural affection as the President said. He thought the President made a slip when speaking of corneal ulceration, as interstitial keratitis did not ulcerate. Mention had been made of gummatous infiltration about the knee-joint. There was a particular joint lesion, especially of the knee, which had been described as chronic painless fluid effusion of the joint, which he had been in the habit of regarding as a most valuable sign; it was only found by examination of the knees, as the patient had no discomfort. With regard to ulceration of the nose and nasopharynx in congenital conditions, he did not think that was infrequent. He had one case which died from meningitis following mastoid disease, which supervened upon middle-ear suppuration, which had been caused by nasopharyngitis following occlusion of the nasopharynx from cicatricial contraction. He thought the point was not sufficiently emphasized that in congenital syphilis there might be double lesions; he, in fact, thought that was almost the usual thing; one very rarely found a perfectly normal drum and Eustachian tube in those cases. Although the nerve deafness was the predominant feature, there was considerable middle-ear trouble as well. With regard to condylomata in the acquired condition, he agreed with Mr. West as to the pain which was present. Another point was that although such cases were very intractable, as a rule they eventually healed without causing contraction; indeed, they left a comparatively normal meatus.

---

[1] Sendziak, *Monatschr. f. Ohrenheilk.*, 1900, xxxiv, p. 419.

Mr. MACLEOD YEARSLEY said he was in agreement with the greater part of the paper. There was, however, one question upon which he would like to join issue—namely, that of deaf-mutism. It was said in the paper that the disease never caused lesions *in utero* such as to produce deaf-mutism. He did not think that statement was proved, as Baratoux had described his researches upon still-born children who were syphilitic, and he found the middle ear and internal ear involved—chiefly purulent middle-ear inflammations, with adhesions of the membrane, hæmorrhage, and accumulations of pus in the labyrinth, and destruction of the organ of Corti. He therefore thought it possible that syphilis might cause deafness *in utero*. It was quite true, however, that most cases of deaf-mutism were due to congenital syphilis occurring in early childhood. Kerr Love, Hahn, and Castex had written on the subject, and Kerr Love gave the percentage of 1·8 and Castex 2·75 of the cases in Paris institutions. He did not know whether Scotsmen were less liable to syphilis, or whether the operation of Contagious Diseases Acts in France checked the disease, but he knew that the percentage in London was much greater. He (Mr. Yearsley) recently published a paper on syphilis as a cause of deaf-mutism based upon the examination of 500 consecutive children in the London County Council deaf schools: 17 were undoubted cases of congenital syphilis, 9 boys and 8 girls, or 3·5 per cent.; or, if the acquired cases only be taken, 7·5 per cent. So that syphilis was not a cause which had been overrated. The great question of prevention must come in, and it was to be hoped that, with the growth of infant consultations, some of these terrible cases of deaf-mutism would be prevented, for they are the most terrible of all deaf-mute cases. He had been continuing his examinations of deaf-mutes since that paper was published, and he had come across other cases which, when he came to get out further statistics, he thought would keep up the percentage. He was in agreement with the statement that otosclerosis was not due to syphilis, and he was glad the remark had been made publicly. He also agreed with what Mr. Whitehead had said concerning concomitant middle-ear involvement in congenital syphilitic cases. It was comparatively uncommon to find a normal membrane, and one could generally demonstrate some middle-ear lesion in addition.

Dr. DAN MCKENZIE alluded to Mr. West's remark that syphilitic deafness might come on at a moment when the patient was under treatment for keratitis, and therefore under antisyphilitic treatment. A striking case illustrating this point came under his observation about eighteen months ago; it was that of a woman, aged 54, who was admitted into hospital for gummata of the lymphatic glands of the neck. He thought they were ordinary tuberculous glands and removed them in the ordinary way. The wound healed perfectly, but six weeks later there was recurrence. In the meantime the specimens had been submitted to Dr. Wingrave, and he suggested the possibility of the glands being syphilitic. Iodide and mercury were given, and the glands got well. Yet while in the hospital and having large doses of these drugs, which were having an effect on the glands, the patient became absolutely deaf in both ears in six weeks. This showed that, although a patient's blood might be loaded with antisyphilitic remedies, yet the spirochæte had the power of causing absolute deafness as if there had been no treatment at all. It was an acquired case. With regard to the pathology of the congenital cases, he agreed with Mr. Yearsley, and remarked that another paper by Otto Mayer had appeared in the *Archiv für Ohrenheilkunde*,[1] which dealt with cases of children dying from syphilis. Microscopical examination showed distinct lesions, probably of a meningitic character, consisting of round-celled infiltration of the auditory nerve and cochlea, and there were a number of other points showing serious pathological change; so that, had those children lived, they would probably have suffered from serious deafness. With regard to the vestibular tests, in one of those tested by him the vestibular reactions were normal, but in all the others tested the vestibular reactions were absent in proportion to the deafness. Allusion had been made to the frequent association of obvious changes in the membrane with the internal-ear trouble. It had been remarked by Gerber that when the middle-ear tract was primarily affected the disease tended to spread rapidly to the labyrinth also.

Mr. MCDONAGH did not think one could gauge the effect of treatment in congenital syphilis, because, irrespective of treatment, congenital syphilis tended to give a positive Wassermann reaction, which was not altered however much mercury was given. A positive reaction should not be taken as an indication that the "606" had no effect since the Wassermann reaction in congenital syphilis is not changed, although "clinically" the child is cured. The proof that it could not always indicate active syphilis was shown in a patient he had, aged 45, who had the scars of double interstitial keratitis, was deaf, and brought three children to St. Bartholomew's Hospital with ringworm. She gave a positive Wassermann reaction and the children a negative one. If she had active syphilis then it was very probable that her children would have been syphilitic. In all cases of late acquired syphilis a positive Wassermann could not be an indication of active syphilis. In late acquired syphilis one could more often change the reaction by giving "606." He had treated only one case of ear trouble with "606," and that was an acquired case in which the patient had a lesion of the eighth nerve on one side. He had reported the case in the *Lancet*[1] that week. Hearing was certainly improved after the injection. With regard to eye troubles after the injection of "606," he thought they could be disregarded, as Ehrlich had over 12,000 reports without any eye trouble. He (the speaker) had injected eighty-two patients and there had been no eye trouble. Two of them had optic neuritis, which cleared up after the injection.

---

[1] *Arch. f. Ohrenheilk.*, 1908, lxxvii, p. 189.

Mr. SYDNEY SCOTT said he would not mention his own clinical experience, but would like to ask Mr. McDonagh whether the reaction of Wassermann's test would be regarded as so certain that we could rely upon a single negative reaction in order to exclude congenital or acquired syphilis.

Mr. McDONAGH, in reply to Mr. Scott, reminded him that there was nothing certain in medicine, but in congenital syphilis if one got a negative Wassermann test one could be fairly certain the case was not congenital syphilis. In acquired syphilis that did not always hold good ; there was a small percentage who had syphilis but who gave a negative Wassermann.

Mr. CHICHELE NOURSE asked whether he was to understand that blistering was only of value in congenital cases, or whether it was also good in acquired syphilis.

Dr. H. J. DAVIS said it was odd that condylomata should appear on a dry surface like the inside of the ear. He had only seen two cases, and the first he mistook for polypus. The patient was a young man who had intense pain, said "he had polypus," and asked Dr. Davis to remove it. He proceeded to do so, but was surprised to find that it did not bleed. On further examination he found one or two deeper down. He saw the patient again two weeks afterwards, and he noticed he had glands in the neck and that the case was one of secondary syphilis. The position was unusual for condylomata. The patient referred to had no aural discharge or any trouble with the ear at all as far as he (the patient) was aware before the appearance of the condylomata.

Mr. WAGGETT said it was very important, when a patient had otalgia with a history of syphilis, to examine the post-nasal space. The mirror frequently revealed ulceration on the posterior wall of the nasopharynx. He did not know whether the President referred to that class in his remarks. He asked for Mr. Cheatle's experience of suppuration in tertiary syphilis in the middle ear. He had himself seen very few cases. With regard to facial paralysis, he had had a curious experience, that of two mastoid operations performed for mechanical reasons, upon the same tertiary syphilitic patient. The operations were separated by five or six years. Both operations were followed by temporary facial paralysis.

---

[1] *Lancet*, 1910, i, p. 1198.

The PRESIDENT (Mr. A. H. Cheatle), in reply, said he had come to learn, and he had learnt. He had already expressed his ignorance on some of the points about which he had been questioned. As to the proof of the two elderly "congenital" cases being due to syphilis, he had taken every care to exclude everything else. Most of the cases were seen before Wassermann's test was in vogue. The keratitis frequently preceded the ear trouble, and he thought it was more frequent than was generally supposed. The "gummatous knee" was so spoken of by a surgeon, but probably would not be described in that way now. With regard to middle-ear affections in congenital syphilis, apart from the Eustachian obstruction, &c., met with in the snuffling stage in nasal ulceration and in the post-ulcerative condition, he did not think there was such a thing as chronic catarrh of the middle ear due to congenital syphilis. It was curious that condylomata did not cause contraction. Mr. Waggett had mentioned a class of case which he (Mr. Cheatle) had specially alluded to—gummata of the adenoid region, which he regarded as a class by itself. He was very glad to hear Mr. McDonagh's remarks, and it was clear that very much more experience was necessary in order to place the matter on a sound basis. He had certainly seen a congenitally syphilitic person with a negative Wassermann.

Otological Section.

November 18, 1910.

Mr. Arthur H. Cheatle, President of the Section, in the Chair.

A Case of Congenital Malformation of the Auricle and Meatus.

By Arthur H. Cheatle, F.R.C.S.

The patient is a woman with a small but well-formed auricle without a meatal opening. There is some hearing, and the rotation test shows vestibular reaction. There is asymmetry of the face. The mastoid process is flattened as compared to the other side. An X-ray photograph shows the presence of mastoid cells, demonstrating that a middle-ear tract of some sort is present. An exploratory operation has been performed, but no meatus was found. There was no evidence of the tympanic plate, and the jaw appeared to articulate with the posterior meatal wall. A small accessory auricle above and in front of the real auricle has been removed.

DISCUSSION.

Dr. W. Milligan said the justification for operating in such cases was where one palpated the presence of an annulus tympanicus, and where one could demonstrate with the stethoscope or some such instrument the passage of air towards the middle ear. He asked whether, in this case, Mr. Cheatle investigated the nasopharynx and the condition of the Eustachian tube. If one could feel from the outside even a rudimentary annulus tympanus, and could demonstrate that air did affect the deeper part of the meatus, one was justified in making an exploratory attempt.

Mr. C. E. West asked what structure in the posterior meatal wall the jaw articulated with. He took it that it would be the anterior surface of the mastoid process.

Mr. Cheatle, in reply, said the reason he did the exploratory operation was that the patient had hearing in the ear and the vestibular reaction was normal. The mastoid cells were present, showing that a middle-ear tract was present. He was unable to detect by feeling whether there was a bony meatus or not. It might have been mere closure of the meatus at the auricle. He wished to make certain of that point, and that was the reason he operated. He examined the nasopharynx with the mirror, and also felt it, but found no difference in the Eustachian tubes. He catheterized frequently but could not hear air enter, though Mr. Jenkins thought he could. He endeavoured to apply the caloric reaction test to the ear, and went so far as to blow iced water up the Eustachian tube, but without result. The jaw apparently articulated with the anterior wall of the mastoid process.

A Case of Healed Fracture of the Right Temporal Bone in which the Line of Fracture in the Meatus can be seen.

By Arthur H. Cheatle, F.R.C.S.

A man, aged 30, sustained a fracture of the base of the skull, involving the right temporal bone, at the age of ten years. A certain amount of middle-ear deafness has resulted. On examination, cicatrices can be seen in Shrapnell's membrane and in the posterior segment of the membrana tensa. The line of fracture is visible in the posterior superior wall of the bony meatus. Small gaps in the bone, filled in by flaccid membrane, can be seen. The internal ear is not affected.

Mr. Cheatle added that the condition must be rare, as it was only the third case of the kind he had seen in his life.

Note of a Case of Nerve Deafness in a Syphilitic of Thirty Years' Standing.

By Arthur H. Cheatle, F.R.C.S.

A man, aged 51, had syphilis at the age of 21. For seven or eight years he had noticed the gradual onset of deafness, with ringing tinnitus in both ears, the right being the worse. No giddiness, headache, herpes or facial paralysis. Examination shows marked nerve implication. No signs or symptoms of tabes or of involvement of other nerves. Wassermann's test is positive. I had stated, in opening the discussion on syphilis at the last meeting, that "I cannot be certain that, apart from tabes, there is a progressive sclerosis of the auditory nerve alone." This case, however, appears to be of that nature.

Specimen of Temporal Bone from a Case of Thrombosis of Cavernous Sinus.

By H. J. Davis, M.B.

A woman, aged 36, who was still suckling a child 11 months old, was admitted on October 25. Four days before admission a right-sided otorrhœa, which had persisted for twenty years, suddenly ceased; "headache, shivering attacks and sickness followed."

Temperature 105° F.; no optic neuritis, but slight rotatory nystagmus on looking to the right; typical mastoid signs absent. Complete mastoid operation forthwith; the bone was of ivory hardness; antrum full of pus and granulations blocking aditus; no erosion noticed in either fossa; lateral sinus exposed but not involved; wound left open; temperature fell to 99° F. Next day patient had rigors with cervical tenderness. At the second operation, at which my colleague Mr. Armour gave invaluable help, the mastoid process was chipped away and the lateral and sigmoid sinus freely exposed; it was engorged, but there was no thrombus. The middle fossa was then opened and an extradural abscess evacuated; it was situated in the angle between the middle and posterior fossæ over the superior petrosal sinus; all overhanging bone was removed to allow of free drainage, and the wound packed with gauze and left open. Next day there was conjugate deviation of eyes to the right (towards the lesion), with violent spontaneous horizontal nystagmus towards the left, producing intense vertigo, unless the eyes were closed. Proptosis and ecchymosis with blindness of the right eye followed; there was facial paralysis, and later the left eye became affected. The patient gradually sank, and died on the fifth day.

Post mortem: Brain, lateral sinus and jugular vein unaffected, but a thrombus extended from the right superior petrosal (as can be seen in the specimen exhibited) to the cavernous sinus across the circular sinus to the ophthalmic veins of the opposite side. The diagnosis of septic thrombosis was correct, but it was in the opposite direction to what was anticipated until the ocular symptoms developed.

DISCUSSION.

The President (Mr. A. Cheatle) said the type of bone was the infantile—a common and dangerous one. He gathered that the course of the infection was through the roof of the antrum by means of the small veins passing from the antrum to the superior petrosal sinus.

Mr. SYDNEY SCOTT said Dr. Davis's demonstration of the path of infection from the middle ear to the cavernous sinus was of interest in connexion with recorded cases of cavernous sinus thrombosis associated with ear disease, but in which no line of infection was mentioned.

Mr. SECCOMBE HETT said that he had been very interested in Dr. Davis's description of the case of cavernous sinus thrombosis without thrombosis of the lateral sinus. He himself had had a case of double cavernous sinus thrombosis without thrombosis of the lateral sinus. The patient, a boy aged 8, was admitted to the Throat Hospital, Golden Square, with discharge from both ears, and pain and tenderness over the right mastoid region. A few days previously the tonsils and adenoids had been removed, and the discharge was said to have come on twenty-four hours after the operation (subsequently there was found to be a history of old otorrhœa). The right mastoid was explored and pus found in the antrum and round the sinus. The sinus itself was found to be normal. The general condition became worse, with symptoms of high fever, and a second operation was performed. The mastoid was opened up, and pus found exuding from the neighbourhood of the jugular bulb—a probe passed along the track led down to the internal carotid. The jugular was exposed and found healthy; the left mastoid and lateral sinus were then explored but nothing abnormal found. The boy had œdema of the right eyelid twenty-four hours before death. At the post-mortem the right cavernous sinus contained pus, and there was thrombosis of the circular sinus and left cavernous sinus. There was no thrombosis of the lateral sinus, and both superior and inferior petrosal sinuses were normal. Pus tracked along the internal carotid into the cavernous sinus, and he wondered whether the infection had travelled through veins passing along the carotid canal.

Extradural Cerebellar Abscess tracking through the Jugular Foramen into the Neck; Sloughing of Middle Third of Internal Jugular Vein; Operation; Recovery.

By H. J. DAVIS, M.B.

CLINICALLY, the history of the case is instructive. A girl aged 19, after being ill a fortnight with vague symptoms resembling enteric fever, was certified as such and admitted into one of the London fever hospitals, where she remained three weeks; she was then transferred to the West London, owing to the presence of a large œdematous swelling extending down the neck and gradually increasing; Widal's reaction had been negative. There was a history of occasional slight otorrhœa for eighteen months, and since the commencement of illness five weeks ago, "sickness and shivering fits."

I saw the patient late one night (Oct. 31), shortly after admission. She was sensible, but very ill; she complained of headache and intense thirst; the right pupil was widely dilated, and this has existed ever since; it looked like a case of typhoid, but this was not the disease from which she was suffering. Double optic neuritis present; temperature, 104° F. There was œdema over the scalp and mastoid, and a large swelling in the neck resembling a unilateral enlargement of the thyroid, and pus was streaming out of the ear. Operation forthwith under A.C.E. On opening the antrum, pus pulsated out of the cavity at each systole; the sinus, covered with granulations, bulged at once into the wound; it was far forward, and exposed at the second blow of the chisel (16 mm. Alexander's pattern); pus had tracked between the dura and bone over the posterior and anterior surfaces of cerebellum (the lateral sinus did not appear thrombosed), and had then tracked through the jugular foramen, as well as through a hole in the mastoid, along the digastric groove into the neck. A double incision was made in the neck and a tube inserted. The patient was infused and returned to bed with the brain cavity and neck incision open. The next two days she was much better, but complained of intense hunger and thirst; she was allowed what she fancied; her particular cravings were "for an apple and acid drops."

On November 3, as improvement was maintained, a second operation was performed, and, with the help of my colleague Mr. Addison, the internal jugular was tied at the level of the clavicle. The mastoid process was removed, as also the bone down to the jugular foramen; on pressing the neck, pus welled out between the jugular bulb and the bone. An incision was made down the neck to the clavicle, and the middle third of the internal jugular was found to be sloughed, forming part of the abscess cavity; this was syringed through in the usual way; the vein, or rather the remains of it, were left *in situ*, but all clot removed. The patient, who collapsed, was given 3 pints of saline by infusion through the arm veins, and returned to the ward. A few hours later she became again suddenly collapsed and cyanosed, and appeared to be dying (pulmonary infarct as a result of a detached thrombus from ligatured vein ?). Strychnine, oxygen, and continuous subcutaneous infusion were given; she received 3½ pints under right breast and 2½ under the left and 1 pint per rectum, making 10 pints in all since operation. I have little doubt this saved her life, for cyanosis faded and the patient recovered consciousness; she rallied in the night, and since then improvement has continued daily.

November 8: The wound, an extensive one, commencing 2 in. above the pinna and extending to the clavicle, is dressed daily by plugging with cyanide gauze saturated in a "soap emulsion" which has cleansed the cavity in a remarkable manner. Dr. Dundas Grant first brought the solution to my notice. The formula is as follows: Potash soap, ʒi; soda soap, ʒi; olive oil, ʒi; distilled water to Oi. A mixture (liq. ammon. fort. ♏v in water) is administered every two hours. Ammonia, in addition to its stimulating properties, reduces liability to thrombosis by its action on the blood.

DISCUSSION.

The President said it was not often cases were reported in which pus had reached the neck through the jugular foramen from a posterior extradural abscess. He had only had one case of his own of the kind and he had not seen another reported.

Mr. West said the case was a remarkable one and Dr. Davis was to be congratulated on the happy issue. But he wished to raise the legitimacy of the term "cerebellar" as applied to such cases. They were not cerebellar abscesses, but abscesses in the cerebellar fossa. "Posterior fossa" abscess, or "cerebellar" fossa abscess, was a better term.

Dr. Davis, in reply, agreed that the better term was "abscess in the posterior fossa," but the term "extradural abscess" alone might mean an abscess anywhere. The vein was tied low down, at the level of the clavicle, because the clot extended as far as that, and, though the vein was thrombosed where the ligature was applied, the clot could not have been a septic one. A large vein tracking down the neck in the direction of the internal jugular was ligatured, being at first mistaken for the latter owing to its size and position. It probably was a communicating anterior jugular, or one of the distended inferior thyroid veins. On the following day the wound would be closed in the neck.

[December 6: The skull wound and neck wound are now quite healed and the patient is up and about the ward. The right pupil still remains dilated.]

Lateral Sinus Thrombosis (Streptococcal), with Early Pulmonary Metastasia; Recovery.

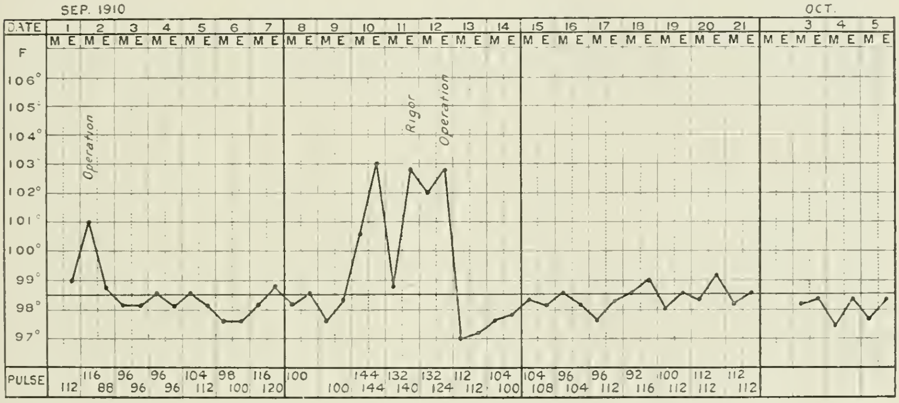
By Sydney Scott, M.S.

N. H., female, aged 11, was admitted to St. Bartholomew's Hospital on September 1, 1910, under the care of Mr. Scott, for discharge from the ears and pain in the head.

History: Bilateral otorrhœa and defective hearing since aged 3. Measles in infancy, and scarlet fever when aged 9. One week before admission the child complained of left earache.

Condition on admission: Poorly nourished. Temperature 99·6° F.; pulse-rate 120. Tongue furred. Left ear: Meatus full of muco-pus; tympanic membrane obscured by granulations. Mastoid not swollen or œdematous; very tender to pressure about 2 cm. behind pinna, which was not displaced. Deafness of middle-ear type. Right ear: Meatus full of muco-pus; tympanic membrane obscured by granulations. Mastoid region normal; deafness of middle-ear type.

Day after admission, September 2: Temperature 101° F.: pulse-rate 116.



Temperature chart of case of lateral sinus thrombosis.

Operation: Left radical mastoid performed, with drainage of extradural parasinous abscess. The antrum and a few cells in its outer wall contained granulations and offensive pus. The malleus and incus and tympanic membrane had been destroyed by the disease. A track of soft bone led backwards from the antrum into the posterior cranial fossa, where an extradural collection of pus along the mesial wall of the lateral sinus was found (streptococci were discovered in films of this pus, together with Gram-negative bacilli; well-spread-out cultures yielded only coliform bacilli). The adjacent sinus wall was covered with plastic lymph, but the rest of the sinus was normal and the contents fluctuated freely. The overhanging bone was cut away to the limits of the extradural abscess. Panse's plastic operation adopted. Cavity packed lightly with gauze, and wound closed with drainage at lower end. A mass of adenoids, which had caused trouble during anæsthesia, was removed with curette.

Subsequent course: September 10—All went well until this day, when the temperature rose to 103·2° F.; pulse-rate from 100 to 144; slight sore throat, but nothing definite discovered. September 11—Morning temperature fell, but evening temperature rose with rigor to 103° F.; pulse-rate 144. Tenderness noticed in cervical glands left side below angle of jaw; vomited once; cough began; also pain in left side of chest; respirations rose to 42 per minute. September 12—Early morning temperature fell to 100° F., but soon rose to 102° F. Chest: A patch of dullness was discovered on the left side, with breath-sounds weakened and friction râle.

Second operation (September 12, 5 p.m.): Ligature of left internal jugular vein. Exposure and drainage of left lateral sinus. The vein was tied at the level of the common facial, above the junction with which it was found to be collapsed. The wound in the neck was closed by suture. The mastoid wound was reopened and the lateral sinus fully exposed. This was now found to be more thickly coated with lymph, and no longer yielded to pressure. The upper limit of a thrombus was soon reached, and a partly disintegrating adherent clot was removed from the sigmoid sinus with curette. The wound was loosely packed with gauze, and dry dressed in the usual way. An exploratory puncture of the chest was made, but no free fluid withdrawn.

Subsequent course: The temperature and pulse-rate fell next day with corresponding decrease in respiratory frequency, and the child made an uninterrupted recovery. A month later Mr. West kindly closed the cranial wound by secondary plastic suture.

Comments: The following points of interest are raised: (1) The necessity for continued caution in prognosis during convalescence after mastoid operation when intracranial suppuration exists; (2) the importance of suspecting sinus infection early, when under similar circumstances signs of sepsis arise; (3) the value of exposing the jugular vein before exploring the cranium, thus confirming the diagnosis of thrombosis; (4) as regards the infection, the discovery of streptococci in the films, masking of streptococci in culture by coliform bacilli, and the probability of the former being responsible for the systemic infection; (5) the lung seems able to deal with early metastatic foci when the primary source of infection has been controlled; (6) the routine removal of adenoids during the first operation on the ear.

DISCUSSION.

Mr. MARRIAGE said it was of distinct interest that the lateral sinus did not become involved until ten days after the operation. He had not had a case exactly similar, but he had had a case of acute mastoid disease necessitating operation, in which symptoms of cerebellar abscess developed after the mastoid had completely healed and the discharge from the meatus had stopped. The first symptom did not appear until six weeks after the original operation for the acute mastoid condition. In that case he thought it better not to interfere again with the mastoid, but trephined over the cerebellar area, and passing his finger between the dura and the cerebellum he felt a hard lump in the upper and anterior part of the cerebellum, and on inserting a trocar into this, pus poured out. It was interesting because there was an abscess round the lateral sinus at the time of the first operation, and the sinus wall was granulating; but as the sinus pulsated freely and there was no sign of thrombosis inside, he left it alone, and the mastoid wound healed up completely. There was no direct infection through bone to the cerebellum, as, although he removed no bone from the mastoid process at the second operation, the wound healed up perfectly. The boy had had no more trouble from his ear, and for two years had been quite well.

Dr. DAVIS, commenting on the remark "the routine removal of adenoids during the first operation on the ear," said that when he read the case he thought the long complication was attributable to the removal of the adenoids. At one time in nearly every case, if the child had adenoids, they were curetted after the bone operation was completed, but on three occasions somewhat disastrous results had occurred in his experience. In one case pleurisy developed, and in another tracheotomy had to be done, as the child stopped breathing. Another child had acute otitis in the opposite ear, which had to be relieved by incision of the drum. Therefore, on the whole, he now preferred to leave the adenoids to be removed subsequently.

Dr. W. MILLIGAN asked Mr. Scott whether it was not possible that in this case there might have been a mural thrombus, and what his experience was of pulsation in the sinus in cases where a mural thrombus was present. He also wished to know whether the infection was streptococcal and whether there was evidence of trouble in the neighbourhood of the sinus; also whether it was not a good plan to puncture the sinus with a hypodermic needle and to examine the blood.

Mr. WAGGETT said it would be valuable to have information concerning the diagnosis of mural thrombosis. It was very difficult to diagnose this condition. Tests from the blood-stream did not seem to be satisfactory. He recently had a case, in a woman, in which the sinus appeared healthy. She had rigors and a temperature until he opened her apparently healthy sinus, the mastoid having previously been operated upon. From that moment she had no more temperature. There was evidently a small mural thrombus which had escaped detection.

Mr. West, dealing with the question of the removal of adenoids in such cases, said it was still his habit to remove adenoids at the time of the mastoid operation or of Schwartze's operation if they were present. He had had good results, but he might have been undeservedly lucky. In any large series of adenoid operations there would be cases of acute otitis media sooner or later. He failed to see why a septic throat should produce pleurisy, except as a matter of general sepsis. It was a serious point to submit a patient to a second anæsthetic when it was possible to avoid it. In the case of the Schwartze's operation the removal of a mass of adenoids at the time hastened the cessation of the discharge from the middle ear.

Dr. Watson Williams said that when there was reason to believe that the symptoms were due to streptococcal infection, it was desirable to give a considerable dose of antistreptococcic serum, as that tended to prevent general infection from the operation; and if in such conditions, before adenoids were removed at the same time, these prophylactic injections were given a few hours before the operation, it would lessen the tendency to systemic infection through the nasopharyngeal wound.

Mr. Scott, in reply, said that for five years he had removed the adenoids as a routine procedure after completing the operation on the ear, even in acute cases, particularly when they caused obstructing symptoms during general anæsthesia, and certainly thought the patients recovered all the better because this was done. He thought Dr. Davis's remarks were important. With regard to mural thrombosis, if one had to resort to puncture of the sinus for the diagnosis we should have to do this in every case in which we found pus on the sinus wall. He preferred, however, to make the diagnosis by observing the general signs of infection, the condition of the patient, leucocyte count, bacteriological examination of the blood, and state of the temperature and pulse. It would be difficult to depend upon puncture for the diagnosis, for he had examined a sinus histologically from a patient who died from sinus septicæmia and infective endocarditis secondary to an unsuspected mural endophlebitis. There was no occluding thrombus; in fact, the endophlebitis was quite localized, and had almost healed, after the organisms had got into the blood-stream. As a rule, in early cases, he would give antistreptococcic serum only when the general symptoms persisted after the operation on the sinus.

A Case of Retrocerebellar Abscess; Autopsy.

By Sydney Scott, M.S.

H., male, aged 32, was first seen at St. Bartholomew's Hospital on August 22, 1910, to which he was admitted under the care of Mr. Scott.

History: The limited mastoid operation (of Schwartze) had been performed in 1900 by the late Mr. John Langton. The ear had given no trouble to speak of until about a month or more before admission. During the last two weeks the patient had complained of persistent headache, and was said to have shivered once or twice, and had vomited once; there was difficulty in walking, with tendency to fall backwards.

Condition on admission: There was a mastoid scar of the previous operation on the left side. The tympanic membrane was perforated posteriorly; no granulations visible. The tuning-fork tests pointed to middle-ear disease on the left side; the right ear was normal. The patient complained of pain in the occipital region and above the left ear, where there was great tenderness to pressure, but no swelling or œdema; the mental state was clear; no amnesia. Attitude of head: Slight retraction, with chin turned towards the right shoulder. Kernig's sign was well marked; no nystagmus on deviation; no muscular hypotonia in limbs; inco-ordination was discovered by the finger-to-nose test; it was impossible to say on which side the inco-ordination was more marked; all superficial and deep reflexes were obtained; lumbar puncture yielded fluid under hyper-tension; this was cloudy, and afterwards proved to be sterile.

Operation: The left mastoid was opened up in the usual way through a post-aural incision; the antrum and a few outer accessory cells contained granulations and pus, and a soft wide track led backwards through softened bone into the posterior fossa; an extradural abscess was found situated in Whitehead's area; the lateral sinus was exposed and was seen to be dense and fibrous; it was not opened; a considerable area of bone was cut away below and behind the sinus, to give relief to the intracranial pressure; the incus was dislocated and removed, but the malleus and the tympanic membrane were undisturbed; the wound was left widely open and lightly packed.

August 23: Patient no better; headache somewhat relieved, but temperature rose from 100° F. to 102° F.; pulse-rate 80 to 76.

August 24: Patient not so well, and inclined to drowsiness. Eyes: Optic neuritis plainly seen in both eyes, being more marked in the left than the right; conjugate deviation of the eyes to the right; nystagmus produced by attentive fixation to the left with a wide horizontal excursion and sluggish movement; there was no giddiness; the inco-ordination (finger-to-nose test) was still present on both sides, but was more advanced on the left side; dysdiadokokinesia was also now evident on the left side; the patient felt weaker in the left hand than in the right, and hypotonia was discovered in the extensors of the left forearm; all reflexes unchanged.

Second operation: Exploratory puncture of cerebellum, Whitehead's area; several punctures to a depth of 3 cm. in the region usually found to be the seat of cerebellar abscess; no pus discovered; further decompression permitted by removal of part of occipital bone; pulse-rate rose from 68 to 120 after escape of cerebrospinal fluid. The house surgeon was instructed to have instruments ready to transfix the left lobe of the cerebellum should the breathing become embarrassed, but it was hoped the decompression would be sufficient until the seat of pus was revealed. Some four hours later the patient stopped breathing, and Mr. Pretty (house surgeon) incised the cerebellum as directed from Whitehead's area, as far back as the occiput, and let out a small quantity of thick pus. Unfortunately, artificial respiration proved of no avail.

Autopsy (August 25, 1.30 p.m.): Complete examination permitted. All organs congested; otherwise no metastasia or disease discovered apart from the head. Head: Disease restricted to the left temporal bone and posterior cranial fossa. Left sigmoid sinus completely occluded by fibrous tissue for nearly 1·5 cm. Cerebellum contained no pus. Intradural space: A circumscribed abscess was found on the postero-inferior surface of the left lobe of the cerebellum, close to the mesial plane, but not directly involving the vermis. The occipital bone was normal. A narrow track of adhesions in the great horizontal fissure of the cerebellum was the only evidence of a path of infection from the parasinous region to the retrocerebellar abscess. The abscess had been opened by Mr. Pretty's transcerebellar incision. Death appeared to be due to compression from spreading œdema in the roof and side of the fourth ventricle. The other parts of the cranium, including the right temporal bone and labyrinth, were normal.

Summary of principal points: (1) Latent mastoid disease. Formation of extradural parasinous abscess, followed by remote retrocerebellar intradural abscess. (2) Headache, hyper-tension of cerebrospinal fluid and Kernig's sign. (3) Cerebellar attitude of head, triple displacement. (4) Bilateral equal muscular inco-ordination, with unequal inco-ordination (increased on left side) after decompression. (5) Absence of nystagmus, of hypotonia, and of dysdiadokokinesia until cerebellum was decompressed. (6) After decompression, conjugate deviation of the eyes to the contra-lateral side, with nystagmus of cerebellar type, induced by looking to ipsolateral side. (7) Path of infection along great horizontal fissure of cerebellum. (8) Procedure suggested by this case when no abscess found in usual situation. Justification for adopting alternate or occipital route.

DISCUSSION.

Dr. MILLIGAN said the risk of sudden death in cases of suppuration of the posterior fossa was known to all. He asked if Mr. Scott agreed that there were two mechanisms of death in these cases: one in which death was due to cardiac failure, the other to respiratory failure. The cases he had seen were those in which there was sudden cessation of respiration, but in which artificial respiration, resorted to immediately, pulled the patient round.

Mr. SCOTT, in reply, referred to Drs. Grainger Stewart and Gordon Holmes's paper on "Cerebellar Tumours" in *Brain*, 1904.[1] Dysdiadokokinesia was not always present. He had interested himself pathologically in the extension of the infection along the great horizontal fissure of the cerebellum, and agreed that the infection was primarily in the tissues outside the cerebellum cortex. He could not offer Dr. Milligan any evidence in favour of any other explanation of the sudden cause of death in cerebellar cases apart from the respiratory failure, and he thought this doctrine was also sound, for instead of abandoning all hope they were encouraged to make efforts to revive the patient by artificial respiration while the abscess was being opened.

Infiltration of both Auricles (? Chronic Lymphangitis) in a Woman aged 32.

By DAN MCKENZIE, M.D.

BOTH auricles have been thickened for many years. The thickening is subcutaneous, and is of the nature of solid œdema. It is most marked in the conchæ, which present a rounded bulging in place of their normal concavity. The skin here and elsewhere over the auricles is thick and coarse, but does not show any tendency to exude moisture or serous discharge, save perhaps in the meatus. The lining of both meatuses shares in the process, and it has never been possible to see the membranes. Whether or not there is any middle-ear suppuration is not quite settled; the exhibitor thinks there is none. The case has been obstinate to treatment. A rich variety of ointments, lotions, and reputable anti-eczematous remedies have been tried, but to no purpose. A vaccine was prepared from the moisture in the depths of the meatus and injected subcutaneously, with the peculiar result that certain fleeting attacks of local cutaneous inflammation have ceased to appear. With the idea of draining the waterlogged conchæ a small bundle of horsehair sutures was threaded in at the concha, led by means of a needle from the concha to the adjoining region of the cheek, and left *in situ*. The horsehair is still there, but this measure of lymphangioplasty has had no very decided effect upon the auricles. Lately, considerable improvement has followed the daily application of hydrogen peroxide, followed by alcohol, and then by 5 per cent. argent. nit. in aq.

---

[1] *Brain*, 1904, xxvii, pp. 522-591.

The exhibitor believes that the essential pathology of a condition such as this is the blockage of efferent lymph-vessels by the products of an inflammatory agent—such as that which induces erysipelas. The evanescent attacks of cutaneous redness and swelling to which the patient was formerly subject support this view. He does not favour the application of the term "eczema."

DISCUSSION.

The PRESIDENT said such cases were always obstinate ones, and he agreed that they could not be classed as eczema, the septic infection usually being in the opening of the meatus itself. The thickening was on the outer surface of the concha, its posterior surface being usually free.

Mr. WEST asked what was the nature of the growth which was obtained from the cultures, and from which a vaccine was prepared.

Dr. KELSON said that three years ago[1] he showed a somewhat similar case with a small pinhole meatus, and most of the Fellows present thought it was ordinary eczema of the meatus. It was treated for a long time with various remedies for eczema, but without avail, as it continually relapsed. Finally he cut away the whole of the posterior portion of the wall, making a very large meatus; and although that contracted down somewhat, a year afterwards there was a large meatus, and the condition was practically cured.

Dr. McKENZIE replied that he thought the culture was simply *Staphylococcus albus*. When a bacteriologist reported that organism as being present, it might be regarded as suspicious, because of the possibility of accidental contamination of the specimen by that organism. The vaccine had had no effect on the ear condition, but had a curious effect on the fleeting attacks of inflammation to which the patient was subject. Most improvement had been brought about by the use of the recently-applied hydrogen peroxide, followed by nitrate of silver. He thought Dr. Kelson's method of operating was worthy of imitation.

Chronic Dermatitis of the Pinna due to Infection by Staphylococcus aureus, under Treatment by Vaccine.

By C. E. WEST, F.R.C.S.

FEMALE, aged 35. For several years both pinnæ had been swollen, cracked, and desquamating; from the cracks a serous fluid oozed and formed crusts. The condition was more aggravated on the left side;

---

[1] *Proceedings*, 1908, i (Otol. Sect.), p. 14.

here there was great thickening of the subcutaneous tissue, and the skin was bound down to the cartilage. The condition did not extend beyond the entrance of the meatus. The exuding fluid gave a pure culture of *Staphylococcus aureus*, and the case was put under vaccine treatment with great resulting improvement.

DISCUSSION.

The PRESIDENT asked how long the case had been under treatment by vaccines, and whether improvement was taking place.

Mr. SCOTT asked what dosage and the number of organisms Mr. West had given.

Mr. WEST replied that it was a very chronic case, and was always relapsing, especially in cold weather. Her ear was weeping, and was covered with cracks and scabs. She had been under treatment with lead lotion and calamine lotion, and ammoniated mercury ointment and ichthyol ointment. He would rather say the case was associated with, than due to, *Staphylococcus aureus*, because he did not know that the deep-seated part was due to *Staphylococcus aureus*. She had been under vaccine treatment for three weeks, and improvement had been marked. There had been no local treatment. There was now practically no surface exudation. He did not know whether the improvement was due to the vaccine. With regard to dosage, he would look the facts up and communicate them, to be included in the report.[1]

A Case of Chronic Infective Labyrinthitis.

By C. E. WEST, F.R.C.S.

FEMALE, aged 21. Right chronic discharge since early childhood. First seen on September 29, 1910. Right facial paralysis for about a week; total right deafness and loss of thermal excitability; marked asymmetry between reaction to rotation on right and left sides; tympanum full of granulations. Diagnosis: Chronic infective labyrinthitis. Operation, October 5: Radical mastoid; posterior part of promontory and ampullary parts of external and superior canals replaced by sprouting masses of granulations; facial nerve absolutely isolated from bony support between them. Double vestibulotomy. Present condition: Ear nearly dry; total deafness remains; some slight improvement in condition of face.

Mr. WEST, in answer to the President, said the type of bone was that which Mr. Cheatle had familiarized members with under the name of "infantile."

---

[1] Vaccine of *Staphylococcus aureus* (patient's own strain) has been given in weekly doses, commencing with 50 million and rising to 200 million. There has been no marked general or local reaction.

The Brain of a Patient who died from Cerebellar Abscess.

By W. H. Kelson, M.D.

L. A., female, aged 43, was operated on in hospital on February 2, 1910, for nasal polypi, and returned home on February 5. Left-sided acute otitis media, February 7. Had had middle-ear suppuration as a child, but not recently. March 1: Readmitted to hospital; temperature 100·5° F., pulse 78; wasted; drowsy (could be roused, but seemed horribly bored thereby). Complained of giddiness, and repeatedly said that she thought she was falling out of bed. Left pupil larger than right; both reacted to light and accommodation; coarse, slow nystagmus seen to the left on one occasion. Pus and granulation seen at bottom of left auditory meatus. Speaking voice heard at 1 ft. on this side. Rinné negative.

Knee-jerk more marked on left than right side. The patient vomited several times, both before and after entering the hospital; complained both of occipital and frontal pain. She preferred to lie in a flexed position on her right side. Obstinate constipation was present. On syringing the left ear with cold sterilized water a normal vestibular reaction was obtained. The limbs on the left side seemed weaker and more flaccid than on the right.

The following, though carefully examined for, were never observed—viz., optic neuritis, paralysis of third, fourth, sixth, or other cranial nerves, conjugate or skew deviation, ankle clonus, extensor plantar reflex, dysdiadokokinesia.

Mr. Hunter Tod and Mr. C. E. West kindly saw the case with me: all were agreed on the probability of an intracranial abscess, and the latter strongly advised examination of the cerebellum first. This was done on March 3 by the inner route, and an abscess containing about 1½ oz. (estimated) of pus tapped at once in the left hemisphere; this was found to contain streptococci. Patient was very much better the next day, but considerable difficulty was found subsequently in keeping up satisfactory drainage. She gained flesh, however, and seemed to be slowly progressing. Having to leave town for a few days, I left her in the hands of a colleague on March 24, but her strength failed, and, respiration becoming embarrassed, she died on March 28. The brain shows the site of the abscess surrounded by œdema, and a small lesion (? abscess) on the dorsal surface of the left hemisphere of the cerebellum.

DISCUSSION.

The PRESIDENT asked what was the type of bone. The case opened up the question of the proper drainage of cerebellar and cerebral abscesses, but that was too large a subject for that meeting.

Dr. KELSON replied that the bone was very hard. The pupil on the same side (left) was dilated, and directly after the operation it became small again. But whenever the drainage began to fail, the pupil dilated at once.

Large Tuberculous Polyp removed from the Middle Ear of an Infant aged 7 months.

By H. J. MARRIAGE, F.R.C.S.

PATIENT at the age of 3 months developed otorrhœa on the left side, a polypus appearing two months later. This was removed under choloroform by the local practitioner, but recurred. When seen by me for the first time on January 5, 1909, the child being then 7 months old, there was a profuse discharge from the ear, and a polyp which filled up the tympanum and projected into the meatus. On pressure over the mastoid antrum there was apparently slight tenderness, but no redness or œdema. A few small glands were present on the left side of the neck, but the child was otherwise healthy. The child had been fed by the bottle; no history of tubercles on either side of the family. Radical mastoid operation performed; the antrum was filled with liquid and inspissated pus and granulations; the diseased bone was gouged away, a mere shell of bone being left. The tympanum was about three times its natural size, and growing from the floor and internal wall was a large polyp the size of a cherry; this was removed and smaller granulations scraped away. The child bore the operation well and had no bad symptoms. Twelve days after operation an injection of tuberculin was given, followed by two more injections at intervals of ten days.

At the end of three months the cavity was healed except for a small area near the opening of the Eustachian tube. In the middle of March, 1909, tuberculous dactylitis of proximal phalanx of both index fingers appeared, with slight involvement also of the second metacarpal bone of the right hand. Under treatment by strapping the trouble completely subsided in about three months. In June, 1909, enlarged tuberculous glands removed from left side of neck.

Child has grown into a fine sturdy boy, but from time to time still gets very slight discharge from the upper part of the Eustachian tube.

Dr. L. S. Dudgeon examined a section of the polyp microscopically and reported: "I think the sections represent the best specimen of tubercle I have ever seen."

DISCUSSION.

Mr. WEST asked for more accurate information as to where the space was gained to enable the tympanum to enlarge to three times its natural capacity. Had the inner wall of the tympanum been destroyed?

Mr. WESTMACOTT asked whether Mr. Marriage noticed a change in the glands in the neck after the first operation on the ear. He felt a particular interest in this case, as he had made observations on the point, and found that if one dealt with the ear, even after the glands were almost breaking down, it arrested the process of destruction and the glands became quite small or disappeared, provided the skin over them was not infiltrated.

The PRESIDENT said he had a specimen of early tuberculosis of the temporal bone, in which sections were made of the lining membranes; the tuberculous process started in the middle layers of the lining membrane, and the bone was healthy below.

Mr. MARRIAGE replied that those who saw the polyp would agree that it could not get into the tympanum unless the latter were three times its normal size. The enlargement seemed to be in every direction; the roof was pushed up, the anterior wall pushed forward, and the floor pushed downwards. He agreed with Dr. Horne that most of the cases of tubercle of the ear arose in bone, and probably in this case the disease started in the antrum, as this was filled with granulation tissue and inspissated pus. At the time of operation the glands in the neck were very small. They did not become enlarged until five months afterwards. When they showed signs of softening they were removed. After the dactylitis appeared the enlargement of the glands came on.

Deformity of Left Auricle.

By G. SECCOMBE HETT, F.R.C.S.

THE patient is a boy, aged 4. The left auricle is deformed and smaller than the right. There is no sign of a meatus. There is a perivascular tubercle on both sides. The mastoid process is present. There is a well-developed left sterno-mastoid. The face is asymmetric. The boy has enlarged tonsils and speaks indistinctly. An operation is said to have been performed, and the patient's father says that the adhesion which is present between the tragus and the antitragus is the result of this.

Otological Section.

January 20, 1911.

Mr. ARTHUR H. CHEATLE, President of the Section, in the Chair.

The Bacteriology of Chronic Post-nasal Catarrh: A Preliminary Study from Fifty Cases.

By C. ERNEST WEST, F.R.C.S.

THE importance of chronic post-nasal catarrh in the ætiology of many forms of deafness and in the perpetuation of many aural suppurations does not stand in need of elaboration. The futility of attempts to remedy the effects of a chronic Eustachian catarrh by such methods as inflation, the use of bougies, or oto-massage, while the cause of the catarrh goes on uninterruptedly in the post-nasal space, again need not be insisted on, for it will, I think, be granted that the problem of the treatment of the catarrhal ear is essentially that of the cure of catarrhal processes in the post-nasal space, and that, until this problem is solved, the question of treatment addressed to the ear itself may for practical purposes be shelved.

In common with, I suppose, everyone else, I have done my share in the application of lotions and sprays and inhalations in these cases; their utility in the less obstinate I willingly grant. Where there is any definite local condition unfavourable to the recovery of the catarrh, the presence of adenoids, of nasal obstruction, of diseased tonsils, or, only second to adenoids in children, and of far greater importance in adults, of septic teeth, it is clear that these conditions should be rectified if the chances of recovery of the post-nasal mucosa are to be the best possible. But when all these conditions have been excluded or eliminated by treatment, there remains a large and harassing class of patients who have a continued post-nasal catarrh and nothing else as the cause of their deafness, and as the certain cause of its increase; and many of these, in my experience, absolutely fail to get any appreciable permanent good from any usual form of treatment. These cases are the reproach of otology. If we consider the post-nasal condition of these patients, there is in nearly all cases some observable catarrh; it may be obvious, with the secretion of considerable amounts of mucus or mucopus, but in the more typical instances which resist ordinary treatment so completely, the amount of secretion present is small, often there is no visible mucus. The mucosa is somewhat swollen and engorged, especially over what may be called the "adenoid area," which often stands out sharply demarcated from its surroundings by its deeper colour. The mucosa over the pharyngeal aspect of the Eustachian cushions is generally swollen and pale; that under the shelter of the hood frequently shows conspicuously pale and dry-looking. Often there is mucus in the lateral fornix of the pharynx above the tubal ridge when none is to be seen elsewhere.

These conditions are clearly infective, but the bacteriology of the nasopharynx has been unsatisfactory and the results obtained have generally been subject to suspicion of contamination either from the nose or mouth in collection. It appeared to me, therefore, that a fresh start must be made if I was to learn anything of serious value to myself about the infections in post-nasal catarrh.

I devised a post-nasal cannula with a stylet, the end of which could be kept shielded from contact with the mouth and could be protruded with an attached swab when the cannula was in position behind the palate. After the swab has been well rubbed over the pharyngeal vault it is withdrawn into the cannula and the latter removed from the mouth. The outside of the cannula is then wiped free of saliva or mucus, the swab is protruded, and is removed from the stylet with sterile forceps. Plate cultures on blood-agar are made either directly from the swab or as soon as possible; if delay is inevitable, it is a good plan to keep the swab in an atmosphere saturated with aqueous vapour, so as to prevent any drying up of the mucus, very little of which is removed in some cases. This is easily effected by placing the swab in a sterile test-tube along with a plug of sterile cotton-wool moistened with sterile water, and stopping in the usual way with cotton-wool. In this way a minimal quantity of mucus may be kept without drying for many hours.

I have tabulated the culture results of fifty consecutive inquiries, excluding only some few in which the growth either failed altogether or was inadequate to entitle it to be considered a fair sample. Such cases have always been investigated a second or more times if necessary. At one time a contaminated batch of wool gave me trouble by overgrowths of *Bacillus mesentericus* in a series of cases; apart from this, the plates have been strikingly free from contaminations by air-organisms.

It must be borne in mind in looking at these results that the cases have all been strictly chronic post-nasal catarrhs: no case in the course of a cold has been consciously admitted; nearly all had been under observation for a considerable time before culture was made, and in the great majority of cases an alkaline nasopharyngeal wash had been in use for weeks or months. The bacteriological findings, therefore, are not to be compared with those ascribed to acute nasal catarrhs.

Those who have any experience of the difficulties of the bacteriology of the post-nasal space will appreciate the amount of work which this short series of cases means. For the bacteriological results I have to record my grateful thanks to Dr. Gordon, whose name is a guarantee of their accuracy, and to Mr. Gillies, my late house surgeon, who, as Luther Holden research scholar, has taken a share in the work under Dr. Gordon's guidance. The difficulty of coping with a piece of work such as this in addition to the routine work of a busy pathological department is the reason why, for the purposes of an introductory study, we agreed to allow the streptococci and the Gram-negative cocci other than catarrhalis to stand without further differentiation, except in a few cases. The staphylococci have been to some extent analysed. The scheme includes the whole of the organisms grown from these fifty plates.

At a first glance over the table (*see* p. 46) perhaps the most striking point is the small number of categories into which the organisms found may be grouped. It will be seen that, apart from a diphtheroid of non-pathogenic type which occurred in small numbers once, we have only seven columns. I grant at once that the streptococci which are grouped together form a complicated group, but, as will be seen, they are of secondary importance in these infections. The group of Gram-negative cocci other than catarrhalis is of great interest and is badly in need of differentiation, but this is a large piece of pioneer work in itself, and has for the present been deferred. The next point is that indiscriminate mixtures have been almost entirely absent; among the many hundreds of colonies present on the plates it is very rare for organisms

TABLE SHOWING ORGANISMS PRESENT IN FIFTY CONSECUTIVE CASES.

Pneumococcus, pure or dominant	Pneumococcus	Streptococcus	Staphylococcus	Gram-negative	Catarrhalis	Friedländer	Diphtheroid
	+					+ +	
	+		+ albus	+		+	
	+		+ aureus				
	+		+ aureus				
+							
+		+					
	+		+ albus				
+				+		+	
+			(+)				
	+		+				
	+	+ + pyogenes					
+			(+) aureus				
+				+			
	(+)				+ +		
	+	+	+ albus	(+)			
	(+)		+ + aureus				
	+	+					
		(+)	(+)				
		+	+ aureus				
		+	+				
	+					+ +	
+				+			
				+ meningococcus		+	
	(+)	+ mixture	(+)				
				+ dominant			+
	+			+			
	(+)	+	+ aureus				
	+	+	+				
+			(+)	(+)			
+	Same patient						
+							
	+		+ albus				
+			+ aureus				
+			(+ aureus)			(+)	
+			(+ albus)				
	+		+ aureus				
+			+ aureus				
			+ + aureus	Same patient			
			+ + aureus				
		+ +	+ aureus				
		+ mixture	+ mixture				
	+				+		
		+	(+) aureus		+		
	+		+ aureus		+		
	+		+ aureus				
+		(+)	(+)				
+		(+) salivary					
+			(+) aureus	Same patient after vaccine			
	(+)		(+) aureus			+ +	
+				(+)			
18	17	12	21 aureus 13 + (5)	7	4	6	1

of more than three groups to be present; often there are only two perfectly well marked types of colony—e.g., pneumococci and *Staphylococcus aureus;* in a few cases an absolutely pure plate of a single organism is obtained. This last has happened three times among these cases, and also twice among private cases, with the pneumococcus, and on two occasions, in cultures from the same patient (a nurse) with *Staphylococcus aureus;* also in two other private cases with *Staphylococcus aureus.* The absence of indiscriminate mixtures and the constancy of the types of combination afford, I think, very strong presumption that we are dealing with actual pathogenic organisms. For example, pneumococci and *Staphylococcus aureus* occur together without other admixture seven times.

(It should perhaps be said that certain plates have been rejected on account of contamination by mesentericus; where this has occurred there has usually been at the beginning only a single colony of this organism from which spread has taken place, a circumstance making it almost certain that the organism was accidentally introduced, and was not derived from the nasopharynx.)

If we consider the categories in succession, the pneumococci are seen to stand pre-eminent in frequency of occurrence; counting only those cases in which this organism was present in large proportion, we see that it occurred in this way in no less than 70 per cent. of all cases, while it was much the principal organism, or was in actually pure culture, in 36 per cent. In very few plates, indeed, is the pneumococcus entirely absent. The clinical appearance in these cases of dominant pneumococcal infection show a certain measure of uniformity: the catarrh is of the most chronic type, generally presenting itself on the aural side as a pure Eustachian obstruction without loss of lustre or milkiness of the membrane; in the nasopharynx there is usually very little mucus to be seen; often the mucosa appears rather dry and inclined to be glazed; in the middle line of the vault, over the "adenoids area," there is frequently a quadrate patch of rather thicker mucosa, with a slightly pitted appearance, conspicuously redder than its surroundings. The mucosa over the pharyngeal aspect of the cushions is frequently swollen, but rather pale, while that in the shelter of the opening of the tube may be so pale as to seem almost white. This appearance may easily be mistaken for pus hanging in the mouth of the tube.

Considering the immense frequency of streptococci in the mouth, and the bad state of the teeth and gums of most hospital patients, in connexion with which streptococcal infection is now known to be the dominant factor, the comparatively small number of cases in which streptococci were at all numerous in these cultures is remarkable, and affords an incidental piece of evidence of the effective prevention of contamination by saliva in the method of culture. Streptococci were in considerable numbers in 24 per cent. of the plates, but were dominant only twice: they never occurred in pure culture. Of the two dominant streptococcal cases, one was undoubtedly an ordinary *Streptococcus pyogenes;* of the rest, the majority were probably of salivary type, though this point has not been settled by sugar reactions, except on one occasion. Among private cases, however, this has been inquired into, and the streptococci have been in a majority salivary. If the frequency with which septic teeth are associated with post-nasal and Eustachian catarrh is considered, it does not seem far-fetched to refer most of the streptococci found to extension of infection from the buccal cavity. Admixed with streptococci there have been about equal numbers of pneumococcus and staphylococci, the latter generally aureus. It is rather curious that in no single case were all three organisms present in significant numbers, either pneumococci or staphylococci being scanty as the case might be.

Of the staphylococci, aureus is the only one on which I feel justified in laying stress from these cultures. The white staphylococci have not been passed through the sugar tests, and I am unable to say how many of them are albus pyogenes; in addition, among this group are included some white Gram-positive cocci of the sarcina type. *Staphylococcus aureus* is one of the most conspicuous organisms in the cultures, occurring in large numbers in 26 per cent. of cases, and, as has been mentioned, twice pure. It has generally been mixed in about equal numbers with either pneumococci or streptococci. I think in most of these cases of *Staphylococcus aureus* infection there has been rather conspicuously a considerable quantity of mucus in the nasopharynx.

The Gram-negative cocci form a little explored province, and apart from the few cases in which catarrhalis was present, they have frankly been neglected in this inquiry up to the present. They are, however, of great bacteriological interest, and I hope at a later time to be able to do something to unravel them. One point is worth mention, that on several occasions we have found a coccus morphologically indistinguishable from the meningococcus in considerable numbers, this organism in one instance being in almost pure cultures admixed only with a small number of colonies of a non-pathogenic diphtheroid. Catarrhalis was found in large numbers once only, and in all only four times, 8 per cent.; it had disappeared a week later on repetition of the culture in several cases, and I am inclined to think that catarrhalis is not a constant inhabitant of the nasopharynx, but may come and go with small exacerbations of the catarrh.

The group of cases in which the pneumobacillus of Friedländer was present is small, but interesting: it occurred altogether in 12 per cent. of cases, and was a dominant organism in half of these. All were in a very chronic condition: in one case, a profuse post-nasal catarrh had persisted since an "influenza" some five months previously: it was in this case the dominant organism present. The smallness of this group, together with the comparatively high proportion in which the organism was dominant rather suggests its survival from some more or less specific type of acute catarrh. Friedländer's bacillus is clearly not one of the common organisms of post-nasal catarrh. A private case may be worth mentioning in which this organism occurred in pure culture in a very chronic post-nasal catarrh after months of douching, and without any intercurrent catarrhal illness.

I have been making trial of vaccines in some of these cases, but I am not as yet prepared to make any final statement of my views as to the efficiency of the method in chronic post-nasal catarrh. The proportion of favourable results seems to vary according to the class of infection present: though it is an accidental arrangement from this point of view, the fact remains that the headings of the table read from right to left correspond pretty closely with the degree of effect produced in my experience by vaccines. Friedländer infections seem to do well, and catarrhalis quickly disappears under vaccine treatment; though here one is bound to note the frequent spontaneous disappearance of this organism, so that the effect of the vaccination cannot certainly be isolated. *Staphylococcus aureus* seems to be a favourable case for vaccination, and the streptococci certainly diminish or disappear in some cases. On the other hand, the pneumococcus has so far defied my efforts, and has appeared to be just as numerous and just as effective in maintaining catarrhal processes after lengthy courses of vaccine as before them. I had an interesting case in point here the other day: a lady who on two occasions gave a plate from the nasopharynx on which three organisms only appeared—a pneumococcus, a streptococcus, and a diphtheroid resembling Hoffmann's bacillus. After four doses of mixed vaccine I made a fresh culture: the catarrh had to some degree diminished during the interval, the secretion of mucus being certainly much less; the fresh culture gave a pure pneumococcal plate, the diphtheroid and the streptococcus having entirely disappeared. It may be that fuller investigation will show a more successful method with pneumococcal infections, either by the use of still larger doses, or by employing a vaccine from a specially virulent strain. So far I have made use of a vaccine from the original plate or the first subculture.

It seems to me that we may have here a method of treatment which may be of use in a class of cases which too often defies other means of cure.

The question of controls in these cases is a difficult one, for it has been almost impossible to discover samples of the ideally healthy nasopharynx among a town population. In addition, I must confess to having been pressed for time over this section of the work, and I do not put forward such controls as I have as anything more than tentative. They may be said to represent throats not the subject of complaint, not showing obvious post-nasal catarrh, in patients not deaf, and free from adenoids. The relative importance of soil and seed is the governing question, and since all are exposed to the infections, it is probable that the amount of catarrhal reaction is of more importance than the number of organisms. It may be that we have to deal with specific weaknesses of resistance as the principal factor in post-nasal catarrh, and in this case it is possible that a successful treatment might result, not in the expulsion of the organism, but merely in its ceasing to be pathogenic for the particular individual, though still existing in quantity as a normal inhabitant of the nasopharynx. I have examined six controls: they are insufficient, but are all that I have available. Of these, three show a dominant growth of pneumococcus, one of them being mixed with a white staphylococcus; of the rest, one grew a mixture of pneumococcus and *Staphylococcus aureus*, one a mixture of staphylococci, streptococci, and a Gram-negative coccus in about equal numbers, and one, a case which nearly realized my ideal of a healthy nasopharynx, a white staphylococcus in pure but very scanty culture.

So far as any conclusions can be come to, it would appear that the flora of chronic nasopharyngeal catarrh is not materially different from that of the average nasopharynx, and that the difference lies principally in the amount of reaction produced by these organisms.

DISCUSSION.

The PRESIDENT (Mr. Arthur Cheatle) expressed the thanks of the Section for the interesting paper. It might be looked upon as a most valuable preliminary study; first, because one wanted to know the bacteriology of the healthy nasopharynx, and of the diseased nasopharynx, and then to connect the aural troubles with those diseased conditions. The last Mr. West had not attempted to do at present. He supposed that in all the cases in which the author had taken cultures from the nasopharynx he had excluded anything of importance in the nose. With regard to the appearances in post-nasal catarrh, Mr. West spoke of a small mass in the adenoid region, but that might be looked upon as the remains of the nasopharyngeal tonsil. The mouth of the normal Eustachian tube was white, with vessels coursing over the surface.

Mr. SYDNEY SCOTT had found organisms morphologically resembling pneumococci and meningococci co-existing in both tympanic cavities in a child who suffered from meningococcal lepto-meningitis; death followed the onset of pneumococcal pneumonia. The internal ears were free from organisms. He presumed these organisms had reached to the middle ears from the nasopharynx. There was no perforation of either tympanic membrane. Speaking of vaccination cases, he had also noticed the disappearance of organisms in the same order as Mr. West had described, but with persistence of pneumococci in the nasopharynx. He described a case in which vaccines appeared to have an influence on the form of rhinitis. A female patient, aged 17, had been under his observation from time to time for nearly eighteen months with deafness and moist catarrhal rhinitis and nasal obstruction, due to the swollen mucosa. Adenoids had been removed, nasal mucosa had been cauterized, apparently to no benefit. Cultures were taken and vaccines prepared from pneumoccocci and *Micrococcus catarrhalis* which were found. Beginning with a dose of 25 million, a weekly injection was made subcutaneously, each dose being twice as large as the preceding one. The fifth dose was "400 millions." The swelling in the mucosa had quite subsided, the nose and pharynx had become dry, and there was an adherent and very tenacious exudate in the nasopharynx, which could only be removed with forceps. As an isolated case, it was not worth basing much upon, but if similar cases were encountered, they would furnish data from which conclusions as to the influence of vaccines applied in this way could be drawn. The speaker thought that data accruing from researches such as Mr. West was undertaking would ultimately influence the treatment for the affections with which these and other forms of nasopharyngeal infection were associated.

Dr. WATSON-WILLIAMS said the paper was all the more valuable because Mr. West had been chary of drawing definite conclusions. The author's results had been very similar to those obtained by other observers, and had been carried out in a most systematic manner. Though he (Dr. Watson-Williams) had from time to time employed vaccine treatment in such cases as Mr. West had described, he felt bound to say that, as regards the Friedländer bacillus, the results had been disappointing. The best results were obtained in cases associated with the presence of streptococci and staphylococci. Another point he considered noteworthy was that the macroscopic appearance of the secretion being non-purulent or otherwise afforded no indication as to the degree of virulence of the causal organism. Sometimes when the secretion seemed normally clear and slight in amount it was found to be associated with quite virulent organisms. He recalled one patient of his who had recovered from an acute phlegmonous inflammation of the pharynx and larynx, and had persistence of clear post-nasal discharge. He found that it contained virulent streptococci, the secretion coming from some of the nasal sinuses. In many cases one obtained a more satisfactory result by opening up those cavities and so, by using lavage, bringing about effective drainage, than was obtained by vaccines. When there was a meningeal infection unassociated with obvious aural disease, he believed that the infection probably got in through the lymphatics connecting the posterior ethmoidal and sphenoid cell cavities with the intracranial cavity. He hoped Mr. West would continue his researches and give the Section the benefit of further observations and conclusions.

Mr. MARK HOVELL said he thought the interest of such observations would be increased if swabs were taken simultaneously of the nasal cavity, so that the bacteria in the two cavities could be compared.

Dr. FITZGERALD POWELL said he must add his tribute of thanks to the author of this paper, which he considered a valuable addition to the speciality. In reference to the mode of arrival of these bacteria in the post-nasal space, he doubted very much if buccal infection occurred to any great extent in the nasopharynx. He thought the path by which they entered was largely by the nose, and then only when the nose was abnormal. Some time ago Dr. StClair Thomson[1] made very interesting experiments with regard to air entering through the nose. On taking swabbings, very large quantities of bacteria were found in the nose itself, while those taken from the post-nasal space were practically free from infection. Abnormalities of the nose, causing obstruction or conditions such as the various forms of rhinitis, appeared to him to alter the arresting or possibly germicidal power of the secretions, and in this way infection might gain admission. Also in suppurative disease of the ears it is not uncommon to find pneumococci and other bacteria, and it is quite possible they might make their way through the Eustachian tubes to the nasopharynx. The paper brought before us the practical point—viz., the treatment of post-nasal catarrh and other forms of infection; that suggested by the paper—namely, the use of vaccines, was in its infancy, and it was much too early to give any opinion regarding it. But he wished to emphasize the necessity for very careful treatment of the nasopharynx in infective conditions and also when associated with ear troubles. Most of the douching and cleansing by the aid of alkaline and antiseptic douches through the nose was not thorough and was not sufficient to get rid of the infecting organisms, and in addition thorough irrigation of the post-nasal space through the mouth was necessary, and the application of suitable remedies directly to the part infected. If this was carefully done the most obstinate cases of post-nasal catarrh could be cured, and post-nasal bacteria destroyed.

---

[1] *Med. Chir. Trans.*, 1895, lxxviii, p. 239.

Mr. WEST, in reply, thanked members for the way in which his professedly slight paper had been received. He accepted the President's definition of it, that it was a preliminary of a preliminary. The cases which had been under investigation were obstinate chronic cases of post-nasal catarrh; they had had deafness of middle-ear type, with the exception of the control cases, in every way suggesting an Eustachian origin for the deafness. He thought it would be taken for granted from what he said, that infection of the nasal sinuses had been excluded. He did not agree with the President as to the colour of the mucous membrane underneath the hood of the normal Eustachian tube; it was, he considered, pink. He was interested to hear that Mr. Scott had had results similar to his own, particularly with regard to the persistence of pneumococci under vaccine, possibly he had not gone far enough in big doses of autogenous vaccines in those cases. Sometimes he even had great difficulty with the usually easily displaced organism *Staphylococcus aureus*, and he had a girl under treatment who was at present having doses of 2,000 million of vaccine, and her staphylococci had not yet been got rid of. Dr. Watson-Williams's point as to there being no necessary connexion between the virulence of the organism present and the quantity of secretion was particularly true of the Klebs-Loeffler bacillus. In the paper he had insisted on the presence of a definite flora in the nasopharynx in nasopharyngeal catarrh; not necessarily causative in the sense that the flora were not there if there was no catarrh. If one wanted to make cultures easily and rapidly from the post-nasal space, a post-nasal cannula should be used, by which means one could exclude contamination from mouth or nose very completely. He considered there was a future for vaccine treatment in such cases, but it was a purely provisional opinion, and he would not put it forward as being adequately based.

Case in which Disease in a Large Sinus Tympani caused Persistence of Discharge after the Radical Operation; Caloric Tests applied to the exposed Inferior Crus of the Posterior Semicircular Canal.

By Arthur H. Cheatle, F.R.C.S., and G. J. Jenkins, F.R.C.S.

A boy, aged 9, had undergone a radical operation for chronic suppuration of the right middle ear one year previously, but the discharge persisted. A large granulation occupied the deep meatus. On opening the wound, granulation and carious bone were found in the mass of bone below the level of the external semicircular canal; while removing this a small opening was found below the external canal and behind the descending part of the facial nerve. It was noticed by Mr. Jenkins that mopping the middle ear produced a movement of what looked like granulation tissue, presenting at the opening. A probe was passed through the hole into a cavity, which was found to be a large sinus tympani. Thorough exposure showed the cavity reaching from the external canal above to the level of the floor of the meatus below, bounded behind by the smooth inferior crus of the posterior semicircular canal, and passing behind and internally to the descending part of the facial nerve to the middle ear. The cavity contained granulation tissue and inspissated pus, and the granulation which had been seen in the fundus projected from the natural opening of the sinus in the posterior wall of the middle ear.

This unusual exposure allowed of the application of the caloric test, precisely to the posterior semicircular canals.

A specimen exhibiting an identical sinus tympani in the left temporal bone of an adult was also shown. The exhibitor pointed out that the opening of the large sinus into the middle ear was extremely small.

The method adopted by Mr. Jenkins was as follows: A copper disk was welded to the end of a piece of copper wire, 1·5 mm. diameter and 5 cm. long; asbestos thread, impregnated with rubber, was wound on the wire so that only the tip of the conductor was left exposed. The instrument was placed in position, with the exposed tip of the wire resting on the posterior semicircular canal, and the disk sprayed with ethyl chloride. The cooling was by this means rapidly produced, and immediately nystagmus appeared the instrument was removed.

The test was applied to the patient standing, with head erect and face directed forward. The following effects were observed: Eyes directed upwards—Medium to coarse vertical nystagmus. This movement was almost pure at first, but there was also a fine right to left rotatory nystagmus, which became more marked. Eyes directed downward—A coarse right to left rotatory nystagmus, with fine oblique up and to left movement. Eyes directed to the left—Right to left rotatory nystagmus, with fine oblique movement; horizontal nystagmus appeared later. Eyes directed to the right—Very fine, indefinite movement.

There was also marked vertigo produced, and when the tests were applied to the patient standing, with head erect and eyes shut, the following was obtained: With face forward—The patient fell backward and to the right. With face to the left—The patient fell to the right and forward. With face to the right—The patient fell backward.

DISCUSSION.

Mr. SCOTT considered that this case would become classical in otological literature, for, so far as he knew, it was the first one on record in which direct caloric observations had been made upon the exposed posterior semicircular canal in the human subject, independently of the superior and external canals. He would like to have seen the reactions in this very important case; there appeared to have been a mixture of vertical and rotatory nystagmus. Some doubts already existed as to whether the posterior canal is concerned with pure vertical movements. He hoped they would hear more of this case. He was familiar with the anatomical condition described and displayed in the specimen being handed around. The method of Mr. Jenkins had a touch of genius about it, and was to be much commended.

Dr. DAN McKENZIE agreed with Mr. Scott that the case was unique as regards the testing of the posterior canal. The mixed nystagmus obtained by cooling the canal seemed to be different from what one would have expected, the classical idea being that each canal has its own proper function. But caution should be observed before accepting this result as due to a stimulation of the posterior canal alone, because there was the possibility of the cooling having affected more than the exposed canal. The nystagmus was purely vertical at first, but later it became mixed, and that suggested that the cooling process had first of all affected the exposed posterior canal and then had diffused in such a way as to stimulate the other canals also.

Dr. ALBERT GRAY pointed out that the physical laws governing the movements of fluids made it impossible, strictly speaking, to get anything but mixed results.

The PRESIDENT (Mr. Cheatle), in reply, said it was hoped that the boy would be shown at the meeting, but he had had measles and some temperature. He was a very plucky lad, and helped the investigation greatly by his willingness. The great difficulty was to keep the artificial opening into the sinus tympani patent. He would not have known what condition he was dealing with if he had not previously seen the exhibited specimen. He agreed that the results were mixed. The earliest form of nystagmus must be looked upon as the result of pure posterior canal stimulation.

Case of Exostoses in the Middle Ear.

By ARTHUR H. CHEATLE, F.R.C.S.

A WOMAN, aged 20, had suppuration from the right middle ear in early life. Healing has taken place, leaving a patent perforation in the posterior segment of the membrane. A small exostosis can be seen springing from the promontory and two exostoses from the floor of the middle ear. An adhesion passes from the remains of the articular process of the incus to the head of the stapes. The stapedius tendon is unusually well seen. The remainder of the membrane is opaque.

Case of Lateral Sinus Thrombosis following Acute Otitis Media in a Boy; Operation; Recovery.

By W. J. CHICHELE NOURSE, F.R.C.S.Ed.

THE patient, a boy, aged 11, was admitted into the Central London Throat and Ear Hospital on June 14, on account of pain and tenderness over the right mastoid region, fever, and marked general malaise. There was no mastoid swelling. The right membrana tympani was reddened and slightly bulging. The illness began eight days previously with violent pain in the right ear, persisting for forty-eight hours, and followed by a slight watery discharge. Aching in a less degree continued all the week. The day after admission the temperature rose to 104·8° F. The membrane was freely incised. On June 16 a mass of adenoids was removed; after this the pain and tenderness rapidly subsided, and the boy became quite cheerful, though the temperature continued to oscillate for four days between 99° F. and 102° F. The fever then subsided, and on June 24 the patient was allowed to get up.

Dr. Wyatt Wingrave reported that the fluid from the tympanic cavity contained staphylococci, *Bacillus proteus vulgaris*, and Pfeiffer's bacillus. A culture showed a growth of *Staphylococcus albus*.

Two days later the patient complained of severe pain below and behind the right ear, with great tenderness around the tip of the mastoid process. The neck was held stiffly in one position with the head inclined to the right; movement of the head was painful. Temperature 100° F. The next day the symptoms were more marked, there are some fullness in the neck below the mastoid. A blood-count showed leucocytosis. A cortical operation was then performed. The bone was vascular, but there was no caries. No pus was found. Blood from the antrum contained staphylococci, *Bacillus proteus vulgaris*, and Pfeiffer's bacillus. Following this the fever abated, but the stiffness of the neck continued, and the patient's general condition was not good. He complained greatly when the wound was dressed, and lifted his head with both hands when he wished to change his position. Drowsiness and slowness of pulse were noted.

On July 1 there were drowsiness, slow pulse, and considerable wasting. The knee-jerks were normal; Kernig's sign was absent. Babinski; extensor on the left. No nystagmus. Lumbar puncture, pressure increased, cerebrospinal fluid clear, normal. The operation wound was re-opened, and the lateral sinus explored. It was normal in appearance, but was plugged with clot in which were a few drops of greyish fluid. This fluid contained staphylococci and Gram-positive diplococci. The sinus was cleared out and plugged. On July 3 an attack came on early in the morning, characterized by nausea, vertigo to the right, nystagmus to the opposite side, a distressing sensitiveness to noises, and prostration. The pulse became slow and irregular. The symptoms ceased about noon; afterwards the patient became quite comfortable. In the evening he vomited once. The tuning fork on the vertex was localized in the right ear. The following day the patient's condition was still unsatisfactory; he was more fretful, the pain in the ear and side of the head continued. The pulse was 66; there was increased leucocytosis. The middle fossa was explored, but nothing abnormal found. The dura pulsated, and there was no undue intra-cranial pressure. The dura was not incised. The bone was extremely vascular. From this date the pain and fever gradually subsided, and recovery followed.

The patient's condition is now altogether satisfactory. His hearing is good.

Mr. NOURSE said that the jugular vein was not tied. The boy had no rigors.

Case of Acute Inflammation of the Mastoid treated by Bier's Method.

By G. C. CATHCART, M.D.

THE patient, a little girl, aged 6, had been in bed for a week with influenza, when she developed pain in her left ear, for which her medical adviser prescribed some drops of carbolic and glycerine. The pain continued throughout the day and there was tenderness and swelling over the mastoid. When I saw the case in the evening the temperature had suddenly shot up to 104·2° F. and the ear had just begun to discharge, so I advised a continuance of the same treatment. Next morning the temperature was 103·8° F. and the discharge was still present. A perforation in the drum could be plainly seen, and the pain and swelling over the mastoid were most marked at the apex. Mr. West was called in in consultation, and it was agreed to apply moist heat to the mastoid and await events. That evening the temperature was still over 103° F. and the pain and swelling had not subsided. The next morning at half-past eight the temperature was 102·8° F., and the ear was so painful that the child held herself quite stiffly and shrieked if anybody approached her. A Bier's bandage was then applied, and by eleven o'clock the same day, though the swelling was increased and the discharge from it also increased, the pain behind the ear had so much decreased that the child allowed the ear to be freely handled. The drops were discontinued and nothing done to the ear except wiping it out with a dry swab. The next day the swelling was increased owing to the congestion, but the pain did not trouble her at all. A swab was then taken and a cultivation made, when a large number of pneumococci and staphylococci were found. The same treatment was continued for a week, the bandage being put on in the morning and kept on for twenty hours. At the end of a week from this first application another swab was taken, when it was found that the pneumococci had disappeared but staphylococci were still present. As the pain and swelling had completely disappeared and the discharge was not so abundant the bandage was then only kept on for twelve hours during the day. At the end of three weeks the ear was completely healed. Of course one has seen many cases of discharge of the ear recover without any treatment, but the remarkable thing about this case, and the reason that I bring it before the Section, is to show how quickly the pain disappeared after the application of the bandage.

DISCUSSION.

The PRESIDENT asked what Dr. Cathcart meant by "acute inflammation of the mastoid." Was it acute periostitis, or osteitis, or inflammation of the lining membrane, or was there serum or pus in the mastoid cells? Also, could he quite exclude enlargement of the gland immediately below the auricle, œdema from which often spreads on to the tip of the mastoid process?

Dr. DAN MCKENZIE said that Bier's method of treatment of suppuration in the ear and adjoining cavities had been made the subject of at least two investigations abroad, with the result that it was declared to be a dangerous method because it masked pain, and hence the patient was not aware of the onset of dangerous symptoms. If Bier's method were applied, the patient should be kept under close observation, and the surgeon be in readiness to do an operation. That criticism did not apply to cases on which the mastoid had already been opened. He had used it with good effect in such a case, and it hastened the filling up of the large cavity in the bone with granulations.

Mr. WEST remarked that as he saw the case with Dr. Cathcart he would say that, in his opinion, it was a case of acute otitis media, with acute empyema of the antrum; the antrum being full of inflammatory exudate under pressure. There was probably osteitis around the antrum, and the tenderness was periosteal. The patient was suffering from acute antritis. He thought at the time that the case would resolve under ordinary lines of treatment. But from what Dr. Cathcart told him—he did not himself see the case afterwards—the relief of the pain by the Bier's method must have been very gratifying. The importance of the masking of pain by the method seemed to have been a little over-pressed, because pain was a very uncertain factor to take for guidance, rather than such factors as the state of the temperature, the pulse, and the local condition as shown by inspection and palpation.

Dr. FITZGERALD POWELL said apparently the result of the Bier treatment had in this case been very satisfactory. With regard to the danger of the method, there was quite a large perforation to be seen, and he did not see any danger in the bandage, provided there was free drainage. And freedom from pain was a thing devoutly to be desired.

Mr. WAGGETT asked what were the intervals of bandaging and non-bandaging in the case.

Dr. CATHCART, in reply, confirmed Mr. West's opinion. He did not agree with Dr. McKenzie that there was danger in masking the pain, provided that there was free discharge. In answer to Mr. Waggett, he said that the bandage was kept on for twenty hours continuously, and left off for four hours. It was wrapped twice round the neck, and the criterion of whether it was correctly applied was a feeling of "fullness" in the face, with flushing, and obliteration of pain. Dr. Cathcart then demonstrated the correct method of applying the bandage.

Case of Unilateral Deafness, probably Complete, associated with Vertigo.

By DAN MCKENZIE, M.D.

THE patient, a man aged 43, has been growing deaf in the right ear for two or three years. For the last fifteen months he has been afflicted with tinnitus of peculiarly distressing character, and during the same period he has experienced daily attacks of vertigo, from five minutes to two hours in duration. The vertigo, though severe, has never made him fall; it is associated with subjective movement of external objects. He has been sick and has vomited on one or two occasions. The patient complains, further, of pressure on the top of his head; he sleeps badly, dreaming much, and starting and talking in his sleep. All these symptoms have moderated lately, probably as a result of the bromide he has been taking.

A general survey of the patient has not hitherto disclosed any other abnormality, save the presence of a fine tremor in the tongue and a somewhat excitable tendency in the man himself. There is no sign of involvement of any of the cranial nerves, if we except the right auditory. There is no history of syphilis. Tested in the ordinary way, with the sound ear stopped up by the finger, the bone conduction would seem to be only reduced in the affected ear, but when tested with the aid of Bárány's noise machine, he is unable to hear the tuning fork at all in contact with the mastoid of that side.

The vestibular tests (cold caloric) work out as follows: Right (affected) ear—Slight nystagmus in ten seconds, followed by decided nystagmus in fifty seconds; vertigo. Left (sound) ear—Marked nystagmus in ten seconds; vertigo.

The case is shown to illustrate the claim made for the Bárány noise machine, that it enables us to exclude the one ear when testing the other.

The diagnosis in this case is at present uncertain. The combination of prolonged and severe deafness in one ear only suggests, no doubt, a local rather than a systemic cause.

DISCUSSION.

Mr. West asked whether Dr. McKenzie could advance even a conjectural pathology in connexion with the condition.

The President remarked that the patient stated that he had worked amidst great noises for many years, and that the right ear was more exposed to noise than was the left. But that would not explain the vertigo, which was undoubtedly auditory.

Dr. McKenzie, in reply, said he was unable to advance even a conjectural pathology. There was much work yet to be done in that region of the speciality. One would suppose that prolonged and severe deafness in one ear was due to a local rather than to a systemic cause. Yet he had shown a case before the Section of unilateral deafness occurring in tabes. In this case the presence of the tremor of the tongue, and some slurring of speech, suggested a very early stage of general paralysis. There was no history of insanity, and the pupils acted normally. He had brought the case chiefly to show the effect of Bárány's noise machine. When there is complete unilateral deafness the tuning fork placed on the mastoid of the affected ear may be heard by the sound ear and referred by the patient to the affected ear. If, however, the Bárány noise machine is applied to the sound ear during the test the patient will be quite unable to hear the tuning fork in contact with the affected mastoid. There was, however, a possibility of error in using the noise machine. The din made by it at the sound ear might distract the patient's attention so much that he would be unable to perceive the gentle stimulation produced in the affected ear by the tuning fork. This possible fallacy should not be forgotten.

Two Cases of Cerebellar Abscess.

By W. Milligan, M.D.

Case I.—Cerebellar Abscess following Otitis Media Suppurativa Chronica.

A. D., male, aged 24. History: Right ear had been discharging for eighteen years and is said to have followed rheumatic fever. Three weeks before admission patient had commenced to have severe headaches and occasional attacks of vomiting, bearing no relation to food; there had also been momentary attacks of giddiness, and constipation for the last few weeks.

Condition on admission: Rather drowsy, but able to answer questions; no staggering or uncertainty in gait; pulse 56, temperature 97° F., respiration 18. Local condition — Right meatus filled with pus, an aural polypus protruding; little pain over mastoid; patient complained chiefly of headache; marked tenderness over right side of occiput; high- and low-toned tuning forks localized to right ear. Eyes —Nystagmus present on right lateral tension—just before operation on left; extensive optic neuritis, no squint or ptosis. Muscles—Kernig's sign present, dysdiadokokinesis marked, no muscular weakness. Reflexes—Knee-jerks increased, ankle-clonus (left), no Babinski's reflex. Cerebrospinal fluid—Clear, under tension, no cells or organisms, albumin slightly increased.

Operation: Radical mastoid first performed; cessation of respiration; cerebellum explored from in front of sinus; abscess found; ℥ss—℥i of pus. Uninterrupted recovery.

Examination of pus: Gram-negative diplococci and a few streptococci.

Case II.—Cerebellar Abscess; Operation; Recovery.

N. G., FEMALE, aged 20. History: Right ear discharging for five years. Patient was quite well till three weeks ago, but then began with headache and vomiting; no giddiness; bowels very constipated.

Condition on admission (April 8, 1910): Almost comatose, unable to make any statement; pulse 60, temperature 97° F., respiration 14. Local condition—Right meatus filled with pus; perforation in posterior segment of membrane; pain over mastoid, tenderness over cerebellum. Eyes—Nystagmus on right lateral tension, optic neuritis. Muscles—Kernig's sign present; impossible to estimate strength of muscles. Reflexes—Knee-jerks absent, no ankle-clonus or Babinski. Cerebrospinal fluid clear, not under tension, no cells or organisms.

Operation: Radical mastoid first performed; the cerebellum was then explored in front of the sinus and an abscess found; ℥i—℥ii of pus drained off; a rubber drainage-tube was inserted into the abscess. Progress—Uninterrupted recovery.

DISCUSSION.

Mr. West asked, in regard to the first case, whether the bacteriological result was based upon films, or were there cultures made? He would be glad if Dr. Milligan would give a few more details on this point.

Mr. Sydney Scott asked how Dr. Milligan drained the first case, and how much he did to the cerebellum.

The President asked Dr. Milligan to describe the exact method of drainage employed in the two cases.

Mr. Waggett spoke of the difficulty of detecting multiple cerebellar abscesses.

Dr. Davis did not think a rubber drainage-tube as satisfactory as gauze in draining a brain abscess. The tube lumen could become occluded by softened brain tissue and drainage be thus interfered with. This did not apply with a gauze drain.

Dr. Milligan, in reply, said the examination of the pus was made by means of films only. With regard to drainage, there was always trouble in such cases, whatever kind of drain was used. He had tried rubber tubes, decalcified bone tubes, silver tubes, and gauze. He generally used a rubber drainage-tube, and wrapped gauze tightly round it, so that the rubber drain carried away any broken-down brain tissue and débris of an inspissated nature, and the gauze around the tube carried away the fluid contents of the abscess cavity. He also found that making a second opening into a brain abscess and putting two tubes in was an excellent method of treatment That applied especially to temporo-sphenoidal lobe abscesses where he made an opening through the antro-tympanic roof, and a second opening higher up and further back. In cerebellar abscesses the size of the drainage-tube depended to some extent on the size of the opening which could be made between the groove of the lateral sinus and the internal auditory meatus. The bigger the drainage-tube opening the more satisfactory the result. The length of the tube he did not know, but it was sufficiently long to be able to be stitched to the mastoid incision. His experience of multiple cerebellar abscesses had been limited to one case. He thought the so-called second abscess was only a diverticulum of the original abscess.

Suppuration of the Labyrinth; Facial Paralysis; complete "Bridge" Operation; Recovery.

By W. Milligan, M.D.

A. U., female, aged 13, was brought to hospital complaining of discharge from the right ear and occasional headaches; no vomiting and little pain.

Condition on admission: The right ear was discharging slightly and the middle ear was apparently filled with granulations; the ear had been running for one year. Afterwards parents remembered that the ear had discharged in infancy. There was no marked pain or superficial tenderness over the mastoid, but there seemed to be a little deep-seated tenderness; no tenderness on percussion over the temporal lobe or cerebellum. There was well-marked facial paralysis affecting the right side of the face. Pulse 70, temperature 97° F., respiration 18. Eyes: No nystagmus; unable to close right eye owing to paralysis; some slight congestion of the right optic disk. Bárány's tests: All syringing tests were negative, no nystagmus being induced by any of them. Hearing: Tuning forks lateralized to left side; watch not heard on pressure. Muscles: Some marked weakness of grasp of right hand, also affecting muscles of left leg, but to less extent. No Kernig's sign. Reflexes: Knee-jerks normal, no ankle-clonus or Babinski, no plantar reflex; supinator- and biceps-jerks normal. The temperature varied between 97° F. and 97·6° F. during the next two days. Facial paralysis got slightly worse; other symptoms remained the same. Patient seemed rather stupid mentally. Temperature still subnormal. Just before operation nystagmus was noticed on directing eyes to the left side.

Operation: Usual mastoid incision. On opening the mastoid cortex it was seen to be quite normal and healthy in appearance. The antrum contained a few granulations, but no pus. On inspecting middle ear a few granulations were seen and curetted away. The radical mastoid operation was completed and a large flap cut; the middle ear was then again inspected and a sinus seen in the promontory, which ran into the cochlea. This opening was enlarged with a burr and a large quantity of cholesteatoma seen and removed. The facial nerve was seen to be exposed (it had been covered and pressed upon by the cholesteatoma, thus causing the facial paralysis). The semicircular canals were then opened up above and behind the facial nerve and a probe passed through the opening thus made and then enlarged with a fine labyrinthine burr. The bone over the middle fossa was seen to be carious, and this was removed and the temporal lobe exposed. The wound was packed lightly with gauze and left open.

After operation there was marked improvement in general condition. Temperature remained normal or about so, except for slight rise on the evening after operation. Facial paralysis gradually improving.

Case of Chronic Otitis Media Suppurativa; Necrosis of External Semicircular Canal; Fistula full of Bone Granulation Tissue; Operation; Recovery.

By W. MILLIGAN, M.D.

L. H., FEMALE, aged 22. Ear has been discharging for the last three months; she has been deaf for many years in left ear. For the last week or so there has been pain in ear and tenderness over the mastoid. Headaches and occasional attacks of giddiness for the last two or three weeks and tendency to fall to the left side, especially when looking round quickly. Facial paralysis of a week's duration.

Condition on admission: Local—Left ear discharging slightly; fair-sized perforation, and pale granulation tissue protruding through it. Marked tenderness over mastoid, especially in region of antrum and over middle fossa; no œdema. Temperature 97° F., pulse 106, respiration 26. Marked facial paralysis affecting left side of face, eye, and corner of mouth. Eyes: No marked nystagmus, but suspicion at times on right and left lateral tension; unable to close left eye; no optic neuritis. Reflexes: Knee-jerks much exaggerated; no ankle-clonus; normal plantar reflex. Bárány's tests: No fistula nystagmus; syringing with hot water—no nystagmus; syringing with cold water—slight nystagmus → right. Tuning forks (not quite conclusive) apparently lateralized to right ear; faintly heard in left ear if held on left mastoid. Hearing: Watch not heard, even on pressure; voice just audible, though words not distinguishable.

After admission the nystagmus became more marked, but was never much; it was only present on right lateral tension. The temperature rose once or twice to 101° F.; patient had a bad cough and some bronchitis, which probably accounted for the temperature.

Operation: Usual mastoid incision; very slight mastoid disease, ordinary radical mastoid operation performed. The facial nerve was seen to be exposed, and the chorda tympani was also seen on its way to the Glaserian fissure. A large fistula was seen leading into the external canal, full of granulation tissue; slight discharge of pus. The fistula was enlarged, and the external canal opened along its whole length. It was decided not to open up the cochlea.

Temperature and pulse both remained low after operation—temperature 97° F. to 98·4° F., pulse 52 to 64.

DISCUSSION.

The PRESIDENT asked what was the type of bone in Dr. Milligan's four cases.

Mr. WEST objected to Dr. Milligan's use of the term "temporal lobe." He supposed he meant that he exposed the dura mater. He asked whether in the last case there was a record of an apparent movement in any particular direction with the attacks of giddiness. With regard to the tuning fork tests, was Bárány's sound producer used, and if so, did it throw any light on the patient's condition? With regard to the exposure of the whole length of the external canal, he asked whether that was inclusive or exclusive of the ampulla, or inclusive of the roof of the vestibule. A further important point was as to the present condition of that cavity—had it dried up completely or not?

Mr. SYDNEY SCOTT asked what was the temperature of the water for the warm and the cold tests. He thought it important to state this, as hot water had had no reaction.

Dr. MILLIGAN, in reply, said the type of bone was not infantile. With regard to the tuning fork tests, Bárány's noise apparatus was not used. The external semicircular canal was opened right up to the ampulla and the upper wall of the vestibule. His house surgeon carried out the hot and cold water tests, and he believed water was used at slightly below the body temperature. The ear was now dry.

Otological Section.

February 17, 1911.

Mr. Arthur H. Cheatle, President of the Section, in the Chair.

Report on the Fatal Termination of Case of Deafness and Discomfort in the Right Ear[1] as Early Symptoms in a Case of Epithelioma originating near the Right Eustachian Tube.

By Edward Law, M.D.

Mr. Wilfred Trotter has kindly given microscopic specimens and the following notes :—

1909 : June 4—Tumour of nasopharynx removed after an osteoplastic resection of the maxilla.

1910 : January 13—Patient complained of considerable pain, mainly in the second division of the fifth nerve, but there was no other sign to suggest a recurrence. The patient attended hospital regularly until August, the pain getting gradually worse and worse, but with no sign of local recurrence in the nasopharynx. In August he was admitted into University College Hospital and injections of alcohol were made into the second and third divisions of the fifth nerve without any relief. On September 28, Mr. Trotter removed the infra-orbital nerve from its canal, but the pain still persisted. On October 11, Mr. Trotter explored the Gasserian ganglion and found it involved in a recurrence of the growth. The bone below it was soft, and an exact location of the ganglion was not easy. The patient recovered from the operation, and the relief from pain proved that the ganglion had been removed

---

[1] Brought before this Section on February 5. 1910; see *Proceedings*, 1910, iii (Otol. Sect.), p. 28.

DISCUSSION.

Dr. D. R. Paterson asked whether Mr. Trotter could state the degree of success he had had in the operation for this condition. He had now under observation a woman, aged 70, and wondered whether that was too great an age to stand the shock of such a procedure. The lesion was around the Eustachian tube, on the right side, and the symptoms were practically the same as in Dr. Law's case.

Mr. Herbert Tilley said he had the opportunity of seeing the case with Dr. Law and Mr. Trotter before the operation, and since that time he had seen three other cases. When this case was discussed last year he said he thought the disease was not so rare in that situation as many considered, as he had then seen four cases. In each of those cases a prominent symptom had been some nasal obstruction on one side, associated with deafness, but the chief feature had been the intense pain. His fourth patient sought relief because of excruciating pain, for which he had been having injections of morphia, &c., and there were so many involved glands in the neck that operation was considered inadvisable. The three suggestive diagnostic symptoms were, therefore, nasal obstruction, deafness, and intense pain. Three of the cases had anæsthesia on the right side of the mandible. All his cases had been males, and the affection in three of them was on the right side.

Mr. Wilfred Trotter said there were three points which he wished to mention particularly; he proposed to deal with the subject more fully elsewhere. He had now operated upon eight cases of infiltrating malignant tumour on the lateral wall of the nasopharynx, and it was upon those cases he founded his conclusions. Five or six cases of the eight followed a clinical picture which was extraordinarily precise. The first symptom was usually pain, which was typical trigeminal pain, localized to the anatomical distribution of the nerve, usually beginning in the third division, spreading to the second division, but practically never involving the first. During the earlier months of the disease there was no other physical sign, except perhaps, anæsthesia, partial or complete, on the chin, due to the involvement of the inferior dental nerve. The second symptom was deafness, of Eustachian tube character, which might be relieved by the passage of an Eustachian catheter. There was a difficulty in the passage of the catheter in the present case, and it was that which forcibly drew attention to the nasopharynx. The third symptom was some abnormality in the soft palate on the same side, not due to involvement of the nerve supply of the palate, but to implication of the levator palati muscle. Therefore the first defect was in connexion with lowering, not raising, the palate on that side, because the muscle was so infiltrated by the growth that it could not be relaxed. The palate was seen to be asymmetrical, and one was inclined to think there must be some weakness, but the patient could be seen to raise the palate well. After a time some œdema was seen on that side.

The sequence of events was characteristic and was due to the fact that the growth was developing in a very small region ; the Eustachian tube, the inferior division of the fifth nerve, and the levator palati muscle all being within ¾ in. of each other, so that a lesion as large as a shilling could involve all those structures. Nasal obstruction was not always present, and might not be complete. In four out of his eight cases, that side of the nose was comparatively free. The tumour was not essentially one of the mucous membrane, but probably originated outside it, therefore its projection into the pharynx might be but slight. He exhibited the specimen of a tumour which he removed the previous day : it showed the growth surrounding the Eustachian tube, but not occluding it. So it would have been possible to have passed a Eustachian catheter and to have been none the wiser as to the condition present. The diagnosis could only be made by digital examination of the nasopharynx, but much projection into that cavity was not necessarily present. In Dr. Law's case the tumour had a pedunculated projection, but in the other cases he could find only a flat hard mass in the lateral wall of the nasopharynx. He had no experience of treatment of the condition other than by removal. He had not seen a case which had remained free from recurrence for more than fifteen months ; but all the cases he had seen had come for treatment comparatively late ; there had generally been neuralgic pain for months before the condition was recognized, and there might be no other obvious signs. If the source of the symptoms were recognized and the operative treatment undertaken earlier, there might be an opportunity of getting satisfactory results. The only way to remove the tumour was to do an osteoplastic resection of the upper jaw ; this gave admirable access and there would be no deformity following. In regard to resecting the upper jaw, it was better instead of the classical operation to include the malar bone with the maxilla and not divide the malar process of the upper jaw, but divide the malar bone in the external angular process and the zygoma. That gave perfect access to the base of the skull and pterygoid region. It was necessary to remove the whole internal pterygoid muscle, certainly the third division of the fifth nerve, and possibly also the second division, with some of the muscles on the front of the spine. It might be necessary to divide the internal carotid. He had done that in two cases, and had had to ligature the artery in the neck, but the patients seemed none the worse. It was generally necessary to remove the glands of the neck, because those tumours frequently invaded the lymphatics. The tumour in this case was an endothelioma, not epithelioma ; and that was a point which perhaps made the cases more favourable for operation, because such endotheliomata were usually of slow growth.

In answer to the President, he said this patient (Dr. Law's case) died because the disease ultimately involved the brain through the foramina of the base of the skull. There was a persistence of trigeminal pain, and he removed the Gasserian ganglion to relieve it ; but the symptoms of cerebral tumour supervened, and death was due to infiltration of the base of the brain.

Caries and Necrosis of Temporal, Parietal, and Occipital Bones following Mastoiditis due to Scarlet Fever in a Girl aged 5; Operation; Recovery.

By H. J. Davis, M.B.

An operation had been performed some months previously to my seeing the child, presumably to drain the mastoid antrum. This had not been reached, but the dura mater in the middle fossa exposed instead; pus tracked between the dura mater and bone, perforated the occipital, and presented as an occipital abscess. This was opened, and the dura mater over these areas denuded of bone; a radical mastoid was done at the same time. Two plastic operations were necessary later to cover an extensive wound. The child was very ill, but is well now. There is still occasional discharge from the ear.

DISCUSSION.

The President (Mr. Arthur H. Cheatle) asked where the parietal bone was affected in this case. He would also be glad to know why the radical operation was done. It was an acute case, and there seemed to be an increasing tendency to do the radical operation immediately if an acute case did not soon do well. He disapproved of this tendency, as the desire should be to save the middle ear as much as possible.

Mr. West asked what was the area of necrosis.

Dr. Dan McKenzie asked if the responsible organism was found, and if so, what it was.

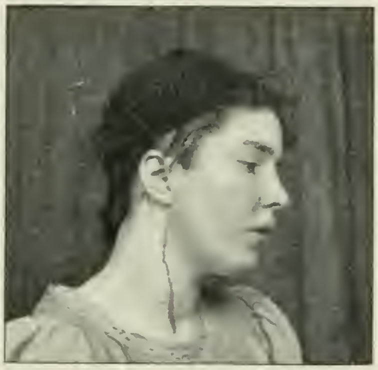
Dr. Urban Pritchard asked whether, in this case, the failure to reach the antrum was due to working too far back, or to not having worked deeply enough. Some surgeons in former days used to go too far back, so that they did not enter the antrum.

Dr. Davis, in reply, said he saw the case a considerable time after it left the Fever Hospital, and he gathered that the child had had acute inflammation of the mastoid, and there was a discharge from a sinus behind the ear, much further back than the antrum. The middle ear was full of granulations and pus, and merely opening the mastoid behind would not have been of much use. That was why the radical operation was done. He supposed the middle fossa had been opened and mistaken for the antrum, consequently the pus had tracked down under the bone posteriorly to the occiput. When the abscess was incised there was a small hole leading into the occipital bone, and in this was a large gutter, which had to be chipped away. The child was ill for a long time, and much of the skin had to be removed. He did not know the nature of the organisms found.

Extradural Cerebellar Abscess tracking through the Jugular Foramen into the Neck.[1]

By H. J. Davis, M.B.

The patient is a girl, aged 19. It will be noticed that the right pupil still remains dilated from irritation of the sympathetic. The mastoid process has been entirely removed, but there is no facial paralysis. The internal jugular vein, the middle third of which had sloughed, has been ligatured at the level of the clavicle.



Extradural cerebellar abscess tracking through jugular foramen.

Acute Vertigo, with Repeated Falls always to the Left, during the last Two Months.

By H. J. Davis, M.B.

The patient, a parlourmaid, aged 21, was admitted into hospital on December 2, 1910, with violent nystagmus, giddiness, and tinnitus; otorrhœa of left ear on and off for years; the mastoid and left side of the head were so tender that she could not tolerate the slightest touch; no optic neuritis, no sickness. Next day the antrum was opened; the bone was of the infantile type; no fistula or erosion into either fossa detected, and nothing further was done. The post-aural wound was left open with a view to a possible extensive operation being necessary.

---

[1] The full notes of the case were reported to the Section on November 18, 1910, and appear in the *Proceedings*, p. 28.

The vertigo was not in any way relieved by drainage, and the tinnitus was so distracting that she used to scream and knock her head against the wall, and was so troublesome in many ways that relief for herself and others was only obtained by hypodermic injections of morphia.

When out of bed she would fall to the ground quite suddenly, but always to the left; she had three fits, epileptiform in character, but not (?) epileptic, and she refused food for a whole week; headache was constant, and vertigo periodical and violent. My colleague, Mr. Armour, saw her, and advised against any further operation, and Dr. Grainger Stewart, who has examined her several times, stated "that he could find no evidence of organic disease of the nervous system, of auditory tumour, or cerebellar disease." She improved, and left the wards a fortnight ago. Briefly, her present condition is as follows:—

(1) Complete anæsthesia of the left half of the body, eye, nasal mucosa, tongue, lips, ear, and larynx.

(2) She hears nothing on the left side, and feels nothing even when a probe is passed into the meatus.

(3) Syringing with hot and ice-cold water produces no reaction whatever in the labyrinth; she does not even feel that this is being done.

(4) The tongue cannot be protruded straight, but deviates to either side.

The case is a complicated one, and it is a question whether the real nature of the disease is labyrinthine, intracranial, or due to hysteria; there is no doubt that the patient is neurotic, but has she an organic lesion as well?

DISCUSSION.

Dr. Davis added that the vertigo was most violent; the patient's bed seemed to her suddenly to spin round counter-clockwise, and she had the sensation of falling down into the ward below. She had become completely anæsthetic on the left side, and since the last twenty-four hours on the right. She had heard nothing during the last five days. He had syringed the right ear the day before with cold water to see if there was any reaction. After two minutes she fell over on the right side. One could syringe the left ear without the patient knowing it was being done.

Mr. West asked what was the character of the nystagmus.

Dr. Dan McKenzie said that, looking at the case from the point of view of the vestibular reactions, it was interesting, as he had shown one or two cases before the Section in which, in hysteria, the vestibular reactions were active.

Some of the features of the present case were very suspicious indeed of hysteria; the hemianæsthesia, and the subsequent occurrence of anæsthesia on the right side, involving the whole of the cutaneous surface. So that, unless subsequent developments justified a different view, he thought it might be taken to be a case of hysteria in which vestibular reactions were absent.

Dr. PRITCHARD said one would, a priori, conclude it to be a case of hysteria. He asked whether the retina was examined, and, if so, whether the field of vision was contracted, as that was said to be an accompaniment of hysteria.

The PRESIDENT raised the question as to whether it might be organic ear trouble, with a neurosis which would account for the remainder of the symptoms. He did not regard spontaneous nystagmus as hysterical. Had any search and inquiry been made to ascertain whether there was congenital syphilis?

Mr. SYDNEY SCOTT said he considered that the case was not hysteria alone, but that there must be some organic lesion to account for the absence of vestibular reactions on one side.

Dr. DAVIS replied that the case at first appeared to be one of acute suppurative labyrinthitis; but it could not have been that as the ear would not have dried up as it had done. Her vertigo was very violent, and two attempts made by the ophthalmic surgeon to examine her eyes were not very successful, as she became so giddy on opening her eyes that her fields of vision could not be taken. He did not think it could be pure hysteria, but that there must be some intracranial trouble as well. Wassermann's reaction proved negative. He did not think it was possible for a patient to be in such a condition unless there was something organic the matter with the nervous system. It had been suggested that she was insane, but that was not his (Dr. Davis's) view: her memory was good, and she knew that what she was doing was unreasonable. Mercury and iodide had no effect, but full doses of hydrobromic acid relieved the vertigo. Vertigo due to labyrinthine involvement was violent at first but tended slowly to pass off, whereas, vertigo due to an intracranial lesion persisted. This might be the case with hysteria, too.

Fistula of the Ampulla of the Superior Semicircular Canal on the Left Side. Bilateral Radical Mastoid Operation; Instrumental Exploration of the Left Vestibule; Total Vestibular Ablation-reactions (Left Side); Cochlea unaffected.

By SYDNEY SCOTT, M.S.

C. O., A CARPENTER, aged 35; admitted to hospital on July 21, 1910. History: Bilateral otorrhœa following scarlet fever in childhood. Intermittent for previous two years. November, 1909, laid up in bed for five weeks with vertigo and vomiting, provoked by the slightest movement of the head. Returned to work June, 1910. Earache on the left side was followed by facial paresis, early in July.

Otoscopic appearances: The meatus on each side contained pus and epithelial débris; large defects in tympanic membranes; landmarks obscured.

Hearing tests: Left ear—Conversational voice between 1 and 2 metres. Rinné negative. Schwabach doubtfully negative [hypersensitiveness of the mastoid, which prevented usual pressure of fork]. Low limit not recorded before operation. High limit of tone range, 12,000 (Edelmann). Right ear—Conversational voice not heard quite as well in the right as in the left ear. (Tested with meatus closed and with Bárány's "Lärm-Apparat" in opposite meatus.) Low limit of tone range, 26 d.v. (Bezold's fork). High limit, 12,000 (Edelmann).

No spontaneous nystagmus or forced movements. No Rombergism.

Caloric tests applied to left ear only (before operation): Cold (60° F.) —No nystagmus in erect position; no horizontal nystagmus when face upwards. Very slight irregular nystagmus, chiefly rotatory to left when lying face upwards, and slight dizziness when seated upright, after irrigating for over five minutes. No meatal air-compression reaction.

Radical mastoid operation (left side), July 23, 1910: Granulations and cholesteatoma. Dense mastoid cortex. Fistula of ampulla of superior canal about 1½ mm. in diameter. This admitted a seeker, which entered a cavity corresponding to the vestibule, above the facial nerve and in front of ampulla of the external canal. Malleus and incus not discovered. No fistula of the oval window. Panse's flap; no skin-graft.

The following day there was spontaneous rotatory nystagmus to the right side, and the patient was noticed to lose balance on one occasion when sitting up in bed, and fell to the left. The giddiness rapidly subsided, and in two weeks the nystagmus could only be recognized behind opaque glasses. The post-operative nystagmus could be abolished temporarily by rotation in a chair with face downwards, at the rate of six turns in eighteen seconds.

August 12, 1910: Caloric tests—Right—Hot and cold caused normal reactions. Left side—Hot, 115° F., and cold, 60° F., gave no reactions, and no sensations of dizziness.

Rotation tests: Erect, ten turns in twenty seconds. Clockwise caused slight horizontal nystagmus to the left for ten seconds. Counter-clockwise rotation caused stronger horizontal nystagmus to the right lasting twenty seconds.

HEARING.

Right		Left
10 cm.	Whisper	25 cm.
8 cm.	Acoumeter	22 cm.
	Tone range	
26 d.v.	Low tones (Bezold)	23 d.v.
13,000	High tones (Edelmann)	10,000
To the right	Weber's test (e′)	—
Negative	Rinné (e′)	Negative
Positive	Schwabach (meatus open)	Positive
Neutral	Schwabach (meatus closed)	Neutral
Neutral	Schwabach (modified by use of auscultatory tube after Bárány's method	Neutral

Voice: Questions asked in quiet conversational voice at a quarter of a metre from either ear easily heard when the "Lärm-Apparat" is buzzing loudly in opposite meatus.

December, 1910: Radical mastoid operation on right side. Condition similar to that found on left side, but no fistula. Ballance's flap and skin-graft.

Tested February 10, 1911: Left cavity dry everywhere; right side not quite dry in tympanic region. Caloric and rotation tests unchanged (see above).

HEARING TESTS.

Right	Tone range	Left
24 d.v. heard 22 d.v. not heard	Low	90 d.v. heard 80 d.v. not heard
11,500 (Edelmann)	High	10,500 (Edelmann)
—	Weber (90 and 150 d.v.) to left	—
	Rinné	
Negative	(e Bezold clamped)	Negative 20 secs.
Negative 10 secs.	(c′ Bezold unclamped)	Negative
Loss of 90 secs.	Air conduction (c′ Bezold unclamped)	Loss of 150 secs.
Loss of 70 secs.	(c‴ Bezold unclamped)	Loss of 40 secs.
9 cm.	Acoumeter	11 cm.
30 cm.	Voice with "Lärm-Apparat" buzzing loudly in opposite meatus (single words)	36 cm.
About ½ metre	Sentences	1 metre

Patient's own statement as regards his sense of *hearing is* that it is *better in the left ear than in the right*, and although there is but little alteration he does not hear quite so well during general conversation as he used to do.

Chief points of interest: (1) Fistula of superior semicircular ampulla which is uncommon; (2) the direct (operative) and indirect evidence of vestibular exploration; (3) localization of disease without spread to cochlea; (4) effect of healing on lower tone range; (5) greater acuity for middle tones on the left side, which shows greater loss for low tone appreciation.

DISCUSSION.

Mr. WEST said the Section was much indebted to Mr. Scott for the full details he had given. These would constitute a useful reference to members should they meet with a similar case. He asked if any observation was made as to forced movement of the eyes under anæsthesia when the probe was introduced into what must have been the upper part of the vestibular cavity. Also if the opening in the ampulla was filled by granulations, or if it was a simple, open, gaping hole. Also if there was the appearance of abnormal contents in the vestibule itself. It reminded him of some cases in which there seemed to be a continuance of definite hearing after an operation on the labyrinth, and in particular he remembered one case in which there seemed to be no question about the presence of hearing after exfoliation of part of the cochlea. Mr. Cheatle once brought a case of the kind before the Otological Society. The question arose as to how far in some of the cases infections of the labyrinth might invade the vestibular cavity, and still be limited by inflammatory lesions or new tissue, so that the cochlea was shut off. If so, what was the physiology of the cochlea? How did the cochlea function under those conditions?

Dr. DAN MCKENZIE joined in the expression of admiration of the way in which the details of the case were given. An interesting feature was that stated in the report of August 12, 1910, showing there were no reactions on the left side with the caloric test, and yet on rotation there was a reaction ten seconds after rotation in such a direction as to simulate left labyrinthine trouble. This apparent discrepancy between the caloric test and the rotation test showed that the caloric test was much more emphatic an indication of vestibular activity than the rotation test. The localization of the disease to the canals, and the sparing of the semicircular region was important from the point of view of diagnosis and treatment. If one got a case in which there was a history of vertigo, and in which the vestibular reactions were absent, and yet the patient could hear with the affected ear, then his opinion was, that one should hesitate about operating on the labyrinth, because he would doubt the presence of severe disease.

Mr. HUGH JONES asked whether grafting would have the effect of reducing the power of hearing for middle tones on the right side. He asked because the difference between the right and left sides in that respect was so marked. He would also be glad to know whether the loss to low tones in the left ear might not be due to implication of the vestibule or of the cochlea in the disease.

Mr. A. L. WHITEHEAD asked if he understood correctly that no operation was done on the labyrinth—that a probe was merely passed in and withdrawn.

Mr. SCOTT replied that there was no deviation of the eyeballs during the cold irrigation while the patient was under the anæsthetic, but he could not be certain that the eyeballs were examined at the time the probe was passed into the vestibule. There were no granulations at the mouth of the fistula, nor did blood or pus escape from it. With regard to Mr. Hugh Jones's suggestion, the patient was always more deaf on the right side, before either operation was done. The subsequent loss of low tones he considered to be due to the scarring in the tympanum of the left ear, because on August 12 the patient could hear tones on the low forks, although the vestibule had been explored previously to this. It was only during the healing process that low tone appreciation was lost. No part of the bony labyrinth was removed at the operation.

New Growth of Unknown Nature in the Tympanum.

By W. H. KELSON, M.D.

A WOMAN, aged 48, suffering from middle ear deafness, on the left side. On examination was found a small rounded growth, firm to the touch, projecting downwards and inwards into the external auditory meatus from the deepest part of the posterior superior wall. It was impossible to say whether it actually involved the tympanic cavity or not. It was the size of a small pea, and cut off the view of the manubrium almost entirely. It had not quite the hardness of an exostosis. There had been no discharge from the ear for many years. The patient was shown to elicit opinions as to the nature of the growth and the probability of improving the hearing by an operation. The right ear was practically normal.

DISCUSSION.

Mr. WESTMACOTT said examination with a speculum seemed to show that it was a tumour of the tympanic membrane posterior to the handle of the malleus—a fibroma arising from the tympanic membrane or from the periosteum of the malleus. It was very sensitive to touch with the probe. He would be inclined to make a semicircular incision below the growth, and by separating the two edges of the wound, to test the hearing, and if it was improved to excise the growth.

The PRESIDENT asked if the case had been under observation for any length of time, and if so, if the swelling was found to be increasing.

Dr. KELSON replied that he had seen the case for six months. The growth was not increasing, but the patient was much worried by the deafness in the ear, and was anxious to have something done. He thought something of the kind suggested by Dr. Westmacott might be tried, care being taken not to damage other parts of the ear.

Otological Section.

March 17, 1911.

Mr. Arthur H. Cheatle, President of the Section, in the Chair.

Injuries of the External and Middle Ear.

By W. Milligan, M.D.

Synopsis.

Injuries of the auricle: Incised wounds; contusions.

Malignant degeneration of scar-tissue.

Othæmatoma: Causation; points in the pathology of othæmatoma.

Deformities of auricle from self-mutilation; cleft lobule.

Injury to the walls of the meatus from the introduction and during the extraction of foreign bodies.

Fracture of the walls of the meatus.

Injury to the membrana tympani from direct and from indirect violence.

Injury to the ossicular chain: fracture of the manubrium mallei; dislocation of the stapes.

Trauma in its relation to malignant disease of the middle ear.

Accidents to the structures of the middle ear during the performance of permeatal and post-aural operations.

Injury to the pharyngeal end of the middle ear cleft during the performance of post-nasal operations.

The external ear, from its exposed position, is liable to injury from blows, cuts, stabs, scalds, contusions, abrasions, bites, and tears. As a rule, incised wounds heal readily and, provided that the raw surfaces are accurately coapted, with little or no deformity. Cases of complete avulsion of the auricle from injury, and of complete amputation from duelling, have been recorded (Rauch, Politzer) in which immediate reposition and suture of the severed organ was followed by rapid union and healing. In cases of extensive injury to the auricle and cartilaginous meatus care should be exercised to prevent contraction or complete occlusion of the meatus from cicatricial stenosis. Bites of the auricle, although not commonly met with in this country, are said to be fairly frequent abroad—e.g., in the Tyrol. They are prone to be followed by sloughing and the formation of unhealthy ulcers, which in course of time heal, with a resulting and considerable deformity. In fact, wherever the injury is deep enough to involve the underlying perichondrium, subsequent deformity is prone to follow. An interesting and important point in connexion with incised wounds of the auricle is the occasional malignant degeneration of the resulting scar tissue. Of this I have seen two instances, the original injury in both cases being due to an incised wound almost dividing the auricle into two portions. Diffuse hyperæmia of the auricle and erysipelas are fairly common as the result of injury.

Traumatic othæmatomata develop as the result of blows, falls, contusions, &c., and are fairly commonly met with in football players and wrestlers. They are rarely bilateral, and occur much more frequently upon the left side. A type of hæmatoma designated "spontaneous" is met with at times in asylums, and, although doubtless generally due to cerebral degenerative changes, is probably frequently of traumatic origin, more especially so from the fact that it is usually met with upon the left side, and is seldom bilateral. Pathologically a traumatic hæmatoma is a bruise—in other words, as the result of violence an effusion of blood from torn vessels takes place between the perichondrium and the underlying cartilage, or into the cartilage itself. This is due in part to the special histological characteristics of the region, and in part also to the fact that the auricle is in apposition with an unyielding surface which offers a fixed resistance to such an injury as a hard blow, fall, &c. The peculiarity of the auricular perichondrium is that it is formed of a dense connective tissue, continuous on the one hand with the subepithelial reticular tissue, and on the other with the stroma of the cartilaginous matrix. In places it is continuous with strands of fibrous tissue which actually pierce the cartilage, and along which small arteries and nerves run. Hence the frequency with which effusion is found upon both sides of the cartilage. The extravasated blood is, however, more frequently found upon one side, most usually its anterior surface, where it tears up the loose connective tissue which separates the skin from the underlying cartilage, and which in reality is a part of the perichondrium. Should the injury be slight, and the effusion of blood slow, an œdematous condition of this reticular connective tissue results which, when absorption takes place, may be followed by no deformity. When, on the other hand, an extensive and rapid effusion is present the cartilage may be torn and lacerated. Provided no inflammatory reaction sets in, and provided also that the hæmatoma is not irritated by excessive manipulation, the effused serum gradually becomes absorbed and the clot shrinks, while the fibrin remains either as a tolerated foreign body or becomes organized. Should the effusion become infected from one or other cause, suppuration results, accompanied often by extensive cellulitis and necrosis of tissue. In these cases a certain amount of deformity is invariable, and of this the patient should be warned.

The custom of piercing or boring the lobule for the introduction of ear-rings or other ornaments leads at times to the formation of fibrous tumours, often of considerable size, or keloid scars. This custom is one of great antiquity, and was employed at one time as a method of branding servants. In Exodus xxi, 5, 6, in speaking of the master's relations with favourite servants, the following passage occurs: "He (the master) shall also bring him to the door or unto the doorpost, and his master shall bore his ear through with an awl, and he shall serve him for ever." It is a custom adopted by almost all the races of mankind. Amongst the indigenous African, American, Australian, and Indian tribes extraordinary and weird ornaments are worn, the day for the performance of the operation of boring the ear being kept as a festival. As the result of wearing heavy ear ornaments, the lobule is at times completely torn through, producing the unsightly deformity known as "cleft lobule," the treatment of which consists in paring the opposed epithelial surfaces and uniting them accurately with horse-hair, or it becomes so elongated as to produce a most unsightly deformity.

The walls of the auditory meatus, both cartilaginous and bony—more especially the latter—are at times injured as the result of the introduction of foreign bodies, but more frequently from the crude and unskilful methods adopted to extract them. Speaking generally, a foreign body rarely, unless actually forced in, passes beyond the junction of the cartilaginous with the bony portion of the meatus. Its removal is seldom a matter of urgency. Experience shows that it is infinitely better and safer to wait and perform extraction under suitable conditions rather than to attempt it with an inadequate light and an unsuitable armamentarium. Many cases of foreign bodies remaining in the meatus for years without injury to the ear have been recorded. Thus Winterbottom relates the case of a cherry-stone in the ear for sixty years, and I have myself recorded two cases where in one the foreign body, a boot-button, remained in the ear for thirty-seven years, and in the other a slate pencil for thirty years, without doing any damage whatever. In both cases it was known to the patient that there was a foreign body in the ear. The most untoward results at times follow unskilful efforts of extraction. In the *British Medical Journal* of 1877 a case of exploration for a supposed foreign body in a child's ear is recorded, in which death took place one hour and a half afterwards from profuse hæmorrhage. Schmiegelow records a case of death from tetanus after efforts to extract a foreign body. In a case coming under my own notice the membrana tympani and the malleus and incus were removed in the endeavour to extract some molten lead which was supposed to have lodged in the ear; in addition the Fallopian aqueduct was so injured that permanent facial paralysis ensued. When the use of the syringe in the hands of the family practitioner fails to dislodge the foreign body the case should, in my opinion, be referred to an expert. The walls of the meatus are occasionally injured on purpose, and foreign bodies inserted into the ear with fraudulent intention, to excite pity, to obtain charity, to secure admission into hospital, &c.

Gunshot injuries of the meatus are uncommon. In a case coming under my care a bullet passed obliquely through the face, the middle ear, the external auditory meatus, and the posterior portion of the mastoid process. Considerable cicatricial deformity resulted. In a case recorded by Gamgee[1] a police constable received a stab in the ear, the point of the knife passing through the middle meningeal artery, with a resulting fatal hæmorrhage. Fracture of the osseous walls of the meatus may be produced by direct or indirect violence. Fractures from direct violence are rare; from indirect violence they are more common and are generally the result of a fractured skull. The anterior wall of the meatus may be fractured as the result of a severe blow upon the

---

[1] *Lancet*, 1875, i. p. 535.

the lower jaw, its condyle being forcibly driven up against it. Suppuration, as a rule, ensues, often followed by necrosis, sequestration, and stenosis. Fissures of the posterior and superior meatal walls are, as a rule, part and parcel of basal fractures, and will be dealt with by my colleague, Mr. Ballance. In passing, I may, however, say that a limited fissure of the posterior wall, with subsequent infection of the mastoid cells, and development of sinus thrombosis, results at times from injury to the back of the head.

A very interesting speculation and point for discussion arises as to how far injury—direct or indirect—or the traumatism produced as the result of protracted suppuration, predisposes to the development of malignant disease of the tympanic mucous membrane or lining of the auditory meatus. At a former meeting of this Society I brought forward a case of rupture of the membrane from direct violence in which suppuration ensued, followed nine months later by the development of a middle-ear epithelioma, which rapidly spread and killed the patient. I have also at the present moment a male patient under my care who has suffered for twenty years from suppurative otitis media. Three months ago he received a severe blow upon the ear. This was followed by an increase of suppuration, by pain, and recently by several attacks of spontaneous hæmorrhage. He is now suffering from malignant disease of the middle ear. What relation, if any, has the recent injury had to the development of malignancy? The medico-legal bearings of this question are obviously of great importance and one as to which it would be desirable to hear the opinion of those present to-day.

Injury to the membrana tympani (congestion, interlamellar hæmorrhage, thickening, &c.) may be produced from the introduction of foreign bodies into the meatus, surf bathing, irritating applications. Rupture of membrana tympani may be produced by direct or indirect violence. Injury from direct violence, such as from the introduction into the meatus of a pencil, pin, hairpin, toothpick, &c., to relieve some temporary irritation, causes, as a rule, rupture of the posterior superior segment of the membrane, the nature of the rent depending largely upon the instrument producing it. Suppurative otitis media is prone to follow such direct injury to the membrane, from the fact that the foreign body is septic, and also on account of infection from the meatus. If the rupture be seen within a few hours of its receipt its edges will be found to be irregular and covered by a thin layer of extravasated blood, while blood will also be found in the meatus. If, however, the case be not seen until after the lapse of a few days it is practically impossible to distinguish such a traumatic perforation from the perforation produced by disease. In a case which I once saw the posterior segment of the membrane was literally ripped from top to bottom. The accident happened to a child who was playing under an apple tree. A twig of a swinging branch entered the meatus and completely divided the membrane without injuring the walls of the meatus.

In cases of direct violence the foreign body may not only rupture the membrane but may also injure the ossicular chain and even the internal ear. In a case coming under my notice the handle of the malleus was fractured. Incomplete union resulted, so that the lower end of the manubrium was easily moved with a Siegle's apparatus while its upper end remained fixed. Dislocation of the stapes has also been recorded. In a case seen many years ago a hairpin had been thrust through the membrane and into the internal ear through the fenestra ovalis—septic infection of the labyrinth, followed by septic meningitis, and death ensued.

The membrane may also be injured accidentally, or on purpose, from the introduction of hot fluids—caustics, acids, molten metal, &c. In such cases very extensive sloughing is prone to result, followed by suppuration, caries, necrosis, and at times meningitis. Spontaneous hæmorrhage into the tympanic cavity occurs occasionally in caisson workers, divers, and aeronauts, accompanied at times by rupture of the membrane and permanent injury to the delicate structures of the middle ear. When the chorda tympani nerve is injured, either by direct or indirect violence, disturbances of taste upon the same side of the tongue (its anterior two-thirds) are often to be noted, although rarely complained of.

Indirect injury, such as results from the pernicious habit of boxing the ear, violent sneezing, forcible blowing of the nose, forcible inflation, diving, explosions, the use of the telephone, &c., most usually affects the inferior segment of the membrane. In many such cases weakening from previous disease is frequently present. Recently I have seen two somewhat remarkable cases of rupture from indirect violence. In one a gentleman, when out shooting, was hit upon the side of the head by a grouse which he had wounded, and which came on with such velocity as to actually knock him over; in the other a woman suddenly threw her arms round the neck of a man beside whom she was sitting, inserted her tongue into the meatus of his ear, and pulled it out with such force as to cause a large rent in the inferior segment of his membrana tympani. Rupture of the membrane has also been recorded as the result of a stroke of lightning. In cases of indirect injury, healing, provided that the meatus is first disinfected and afterwards packed with sterile gauze, is, as a rule, rapid and complete.

Injury is at times inflicted upon the tympanic contents during the performance of operations upon the ear—both permeatal and post-aural—by the slipping of instruments, too great energy in the use of the gouge or burr, inadequate appreciation of the anatomy of the parts operated upon, &c. Occasionally the jugular bulb, when it happens to present through a dehiscence in the floor of the tympanum, is wounded during the performance of a paracentesis. As a rule the hæmorrhage, which is profuse, is readily controlled by tight packing of the meatus. The facial nerve is at times injured or completely severed during the performance of the post-aural operation, more especially at the moment when the "bridge" is being removed or when the facial spur is being levelled down. The fact also that the Fallopian aqueduct is at times incomplete, either from physiological or pathological causes, renders the nerve liable to injury during curettement of the middle ear. In infants and young children the nerve is also injured at times at its exit from the stylo-mastoid foramen when the post-auricular incision is unduly prolonged and deepened beyond the mastoid apex. The external semicircular canal, from its exposed position upon the inner antral wall, is similarly liable to injury. The symptoms produced are luckily, however, as a rule, only transient. In curetting the inner wall of the middle ear—always a matter of great delicacy—the stapes is at times dislocated and the vestibule opened up. To avoid such an accident, which may readily lead to purulent labyrinthitis, curetting should only be undertaken when the parts are fully exposed to view, and should be performed from behind forwards rather than from before backwards. The varying relations of the knee of the lateral sinus to the middle fossa and to the mastoid cortex are responsible, in part at any rate, for occasional injury to one or both structures.

Two somewhat unusual accidents to the lateral sinus have occurred in the course of my practice. On one occasion when making the ordinary post-aural incision in a case which had previously been operated upon elsewhere the sinus was opened at the first incision down to the bone. The profuse hæmorrhage was at once stopped by plugging, and the parts carefully examined. It was then found that at the previous operation a small portion of the sinus wall had been exposed and had become adherent to the overlying skin during the process of healing. Upon another occasion, when making a conchal flap, the point of the knife punctured an exposed sinus wall, with resulting profuse hæmorrhage.

The sigmoid sinus may also be injured as the result of direct injury to the mastoid process. In a few cases the internal carotid artery has been injured when working forwards and inwards for the relief of cario-necrosis of the pars petrosa. The pharyngeal end of the middle ear cleft is also injured at times during the removal of nasopharyngeal growths, by the breakage of a catheter or Eustachian bougie when in situ. Injury during the removal of adenoids is more prone to take place when the Eustachian cushion is very prominent, when the curette is not kept in the middle line, and when the lateral wall of the nasopharynx is under treatment. I have on two occasions known the Eustachian cushion to be literally amputated, but curiously enough upon neither occasion with any bad result.

Foreign bodies in the nasopharynx at times cause injury to the mouth of the tube. Of this form of trauma I have, however, seen only one instance—viz., a case in which a marble, from some unaccountable reason, became wedged in the nasopharynx and caused ulceration followed by sloughing and ultimate cicatrization of the mouth of one tube. The tube is also seriously damaged at times from gunshot wounds and stabs, with resulting complete occlusion and the formation of secondary middle-ear changes.

Injuries of the Middle and Internal Ear.

By C. A. Ballance, M.V.O., M.S.

Synopsis.

How caused.—(1) Blows on the head; fractured base of skull. (2) Explosions. (3) Foreign bodies deliberately introduced into the meatus; unskilful attempts at extraction; fatalities. (4) Other accidents in treatment; slipping of fine nozzle of syringe; irritating substances used too strong in error. (5) Gunshot injuries. (6) Burns with closure of meatus. (7) Accidental penetration of foreign bodies into meatus. (8) Injury by way of Eustachian tube by catheter or irritating substances.

Diagnosis.—Significance of watery discharge from ear, of hæmorrhage. of facial palsy, and of deafness.

Consequences.—Hæmorrhage; purulent otitis; meningitis; loss of vestibular function, and of hearing; facial palsy.

Principles of Treatment.—Removal of foreign bodies; disinfection of middle ear; operations for injuries implicating the temporal bone.

Injuries to the tympanum and internal ear may be, when viewed as mere injuries, slight, and yet may cause great loss of function, or be extremely dangerous from infective complications; or, owing to concomitant injury of one or other of the large blood-vessels in the vicinity, may immediately threaten life. They occur from direct or indirect violence in many ways, and in many circumstances, and always demand careful, thorough, and appropriate treatment. The injury may vary from a slight abrasion of the membrana tympani to extensive fracture of the petrous and damage to adjacent structures.

To those attached to a large general hospital fractures of the base of the skull will be familiar cases: in many of these the middle and internal ear, and sometimes also the facial nerve, suffer damage. When there is extensive intracranial hæmorrhage or injury to the brain, the ear symptoms are overshadowed by the cerebral, though the aural symptoms may assist in the localization of the graver injury. Fractures of the base of the skull are mostly caused by falls, or by blows on the vertex, or occiput, sometimes by falls from a height on to the feet or buttocks: sometimes also the lower jaw is dislocated with such violence that the displaced condyle causes a fracture of the temporal bone.

A fracture of the base in the middle fossa may run alongside the petrous, parallel with its long axis, or across its base, implicating the tegmen antri and tympani, but not extending into the substance of the petrous; or it may pass through the petrous itself splitting it longitudinally or, more rarely, transversely near its middle, or the tip may be broken off. According to some authorities there is a constant relation between the direction in which force is applied and the direction of the resulting fracture, so that the one can be inferred from the other. For example, a blow on the back of the head will cause a fracture at right angles to the long axis of the petrous, while a fracture resulting from a blow on the vertex or side of the head will be more or less parallel with the long axis of the petrous. It is impossible to determine the extent of the fracture during life, and it is not easy to do so even when the brain has been removed. Körber, indeed, goes so far as to say that the extent of a fracture in the petrous cannot be ascertained until the bone has been macerated.

In fracture of the base of the skull the labyrinth capsule itself may be broken through and the membranous labyrinth torn, but the bone forming the labyrinth capsule is too dense and strong for this to be the common result: the damage to the labyrinth is more usually caused by hæmorrhage into the tissue of the membranous labyrinth and into the labyrinth cavities. Should the injury not prove rapidly fatal from damage to the brain there is always a risk that in the further course of such fractures of the base, suppuration of the labyrinth and meningitis may occur, and these grave consequences sometimes commence a considerable time after the injury. When recovery takes place the nerve endings often suffer much damage from scar tissue or even callus. Also the auditory nerve, and even the more resistant facial, may be compressed by extra-dural hæmorrhage. Intra-meningeal and intra-Fallopian hæmorrhage may also occur and temporarily or permanently arrest the functions of the nerves. In the labyrinth, as elsewhere, effused blood may, if not infected, be absorbed, and so restoration of function may occur, though of course some scar tissue must inevitably replace the absorbed blood-clot.

The following case is one of those described by Barnick, who made careful *histological* examinations. It illustrates the conditions found when the labyrinth is examined *soon after* an injury:—

A man, aged 29, sustained a transverse fracture of the right petrous on the night of January 2, 1896, through a fall on the occiput. The symptoms were loss of consciousness, fever, vomiting, slow and irregular pulse. There was no blood in the meatus, but hæmato-tympanum on the right side. He died on January 7. The dura mater, the sinus, the internal ear, and the tympanum proprium were not directly implicated in the injury. The bleeding into the tympanum had been caused by a fracture through the tegmen antri. In the auditory and in the facial nerve were many small hæmorrhages, and there were also hæmorrhages in the scala tympani of the basal coil of the cochlea, in the branches of the vestibular nerve, and in the neighbourhood of the maculæ acusticæ.

The following case, related by Politzer, is an example of *fatal meningitis occurring some weeks* after the injury :—

A man, aged 40, fainted and fell on the hard-frozen ground. When seen by Politzer six weeks later he was completely deaf, and staggered in walking. The tympanic membrane was intact. Eight days later the man died from acute meningitis of three days' duration. A fissure was found in the petrous on both sides. Each labyrinth was split in two through the vestibule. On the left side the vestibule, the semicircular canals and the cochlea were filled with green pus, which also surrounded the auditory and facial nerves in the internal meatus. The soft parts of the labyrinth were broken down and covered with pus. Only traces of the organ of Corti remained. On the right side, where the fissure did not extend into the internal meatus, a dull red mass, flesh-coloured in places, was found in the vestibule and superior semicircular canal, in the other canals there was a yellowish-red mass; in the cochlea was fluid of a fleshy colour. The membrane of Corti was much thickened. So that there had been extensive hæmorrhage into both labyrinths, and on one side suppuration.

In 1869 Weber published a case which proved fatal, with *labyrinthine suppuration twenty-one months* after the injury :—

The patient, a man, had been struck on the head by a falling beam ; for a short time he was unconscious ; after two months he had headache and vertigo, deafness on the left side and pain in the left mastoid region, the tympanic membrane was normal. Thereafter the patient's condition varied considerably ; sometimes he was able to work. Finally he died suddenly one and three-quarter years after the accident. Suppuration in the labyrinth was found, but it is not stated whether there was or was not meningitis.

Passow, in his admirable volume, to which I am much indebted, in commenting on this case, suggested that an extra-dural abscess had been present for a long time in the *vicinity of the porus acusticus internus*.

Graf had the hearing tested in a number of patients who had recovered from fracture of the skull, in which there was no reason to suppose that the pyramid had participated; the hearing was normal in only twelve out of thirty-nine cases. It has been suggested that the ear suffers damage analogous to the eye defects known as commotio retinæ. Passow also suggests that defective hearing may be caused not by damage to the internal ear but by damage to the central acoustic nerve paths.

It seems at first sight strange that *so dense a bone* as the petrous should be so often fractured, but its strength is sapped by the numerous excavations and channels which hollow it out, such as the jugular fossa, the carotid canal, the internal auditory meatus, the facial and other canals.

SYMPTOMS.

The general cerebral symptoms attending fracture of the base of the skull do not come within the scope of this discussion; they often quite overwhelm the special symptoms of fracture of the petrous and of damage to the ear. The symptoms pointing to fracture of the petrous and injury to the auditory and vestibular apparatus are: (1) Bleeding from the ear; (2) escape of cerebrospinal fluid from the ear; (3) deafness, with or without tinnitus; (4) vertigo; (5) facial palsy; (6) loss of taste on the injured side. They are not all present in every case, and they vary considerably in degree and in duration.

Hæmorrhage from the Ear.—When the tympanic membrane, which in fractures of the petrous is usually ruptured, happens to remain intact, hæmato-tympanum will be present, but there may be no bleeding from the ear at all. Bleeding from the ear may be slight, or may be so free that life is immediately threatened thereby. It may come from the external meatus or the tympanic membrane alone, or from the mucous membrane of the tympanum or from one of the larger vessels which are in relation with the petrous. The source of bleeding from the external canal can be recognized by examination, the amount may be considerable; bleeding from the membrana tympani alone is always small in amount, from the tympanic mucous membrane sharp bleeding may occur, but it soon stops. If the bleeding is profuse and venous a sinus is probably damaged, and the proper treatment is to open the bone and plug the sinus. In the case at St. George's Hospital, related in Holmes's "System of Surgery," the man died quickly from hæmorrhage from the lateral sinus. In fractures of the petrous the smaller sinuses may be damaged, but, quite independently of that, continuous and considerable hæmorrhage may occur from the bone or the meninges. The internal carotid artery in the carotid canal, though not infrequently eroded by disease, usually escapes injury in accidents. The sinuses are closely applied to the bone, but the artery is not; it is surrounded by a venous plexus and a lymph space, and can be pushed aside. When the internal carotid artery is injured the tear is between the anterior and posterior clinoid processes, and an arterio-venous aneurysm may result. Nélaton related such a case, in which the internal carotid artery was injured by a thrust wound.

Escape of Cerebrospinal Fluid.—The older surgical writers have remarked upon the escape of considerable quantities of watery fluid from the ear as a phenomenon occasionally observed in cases of injury to the head. O'Halloran recorded a case which occurred in 1760 in which at least 12 oz. escaped in twenty-four hours. Van der Wield, in a work published in 1787, related the following case :—

> A woman was struck over the left parietal bone with a ninepin with such severity that she lost consciousness and had complete muscular relaxation. She also vomited and had moderate bleeding from the left ear. A bruise was seen but no fracture of the bone could be made out. After venesection and the application of the remedies usual on such occasions she gradually recovered; but for four or five days there was a continuous watery discharge from that ear in such quantity that, in my estimation, it would have filled four half-pint cups. Several towels became daily soaked through with the said fluid. The woman, however, soon recovered, with complete subsidence of symptoms.

The nature and source of this fluid were until fairly recently variously interpreted. By some it was supposed to be serum from the blood transuding through a sinus wall or coming from a blood-clot, by others, the liq. Cotunnii; but it is now known to be the cerebrospinal fluid. The labyrinth does not contain a secreting surface large enough to produce considerable quantities of fluid in a short time, and blood-clots do not pour out serum day by day. It is conceivable that a colourless fluid may be furnished in some quantity by an *otitis serosa*, but it is easy to distinguish such an inflammatory fluid from the liquor cerebrospinalis. The quantity of cerebrospinal fluid so escaping is sometimes very great, as much as 600 c.c. in a day. Escape of cerebrospinal fluid is a less common symptom than bleeding from the ear, and usually occurs after it. It is a sure indication that the subarachnoid space has been opened, and in the injuries we are considering the tear in the arachnoid is usually over the tegmen tympani or in the sheaths of the nerves in the internal auditory meatus.

Deafness is on the side of the lesion. It is of the nerve type, is usually severe, and often absolute. It is sometimes permanent, and sometimes it is recovered from for the reasons already given. *Tinnitus* is often present, and is sometimes severe and persistent. *Tinnitus and vertigo* have long been noticed as occasional and important symptoms in cases of injury to the head. Fabricius Hildanus (1560-1634), for example, relates the case of a nobleman who recovered from an injury to the head, but the hearing of the right ear, which he lost in the accident, "could by no art and by no diligence be restored." He was also tormented by an almost constant tinnitus in the same ear, and was frequently oppressed by vertigo. Seven years later the patient had a severe attack of dysentery, after which the tinnitus ceased, but the deafness remained permanent.

Vertigo.—This is only of value as a diagnostic symptom of injury to the labyrinth after the general symptoms of cerebral concussion have passed off, or if coupled with other evidence of destruction of the labyrinth. In most cases of injury to the head the signs of destruction of the labyrinth cannot, for obvious reasons, be sought for with the same care and precision as in cases of ordinary ear disease, but it is well to bear them in mind. They have been so recently brought to our notice by Bárány that they need not be here further referred to. The symptoms of damage to the labyrinth are much more pronounced in injuries such as *stab wounds, which do not cause cerebral concussion.* Voss published such a case :—

A male, aged 19, was stabbed in the right ear on April 30, 1901. He immediately complained of tinnitus and vertigo, and later in the day vomited. On admission next day : temperature 97·8° F., pulse 60 ; pronounced nystagmus to the left, could not stand with the eyes shut. Woolly sensation at tip of tongue. In front of the right tragus was a wound 1 cm. deep, with clean-cut edges. Wound in the anterior wall of the meatus, and in the tympanic membrane, from which latter a few drops of blood escaped. Whispering voice sound not heard on right side. Complete right facial palsy. Nausea, vertigo, vomiting, and pain were the other symptoms. Temperature remained normal, the hearing improved, and the vertigo and unsteadiness subsided, but the facial palsy persisted.

The labyrinth symptoms were very pronounced in the case of a nurse who came under my own observation, where the damage was caused by the violent impact of the fine nozzle of a syringe. The patient did not completely recover for six months.

Facial palsy resulting from injury is of the peripheral type; it may occur immediately as the result of direct injury to the facial nerve, or, secondarily, as the result of suppurative otitis; it is more often permanent than is the deafness. It is sometimes rendered permanent by slight displacement of a fracture passing through the Fallopian canal, and also by the pressure of inflammatory exudate in this small canal.

Loss of taste occurs in the anterior two-thirds of the tongue on the side of the lesion when the chorda tympani is damaged. Sometimes the whole side of the tongue is affected, and then damage to the nerve of Wrisberg may be inferred.

Explosions and Loud Noises.—Again, it is an old observation that the organ of hearing may be damaged from these causes. Fabricius Hildanus recorded two cases:—

> (1) In the year 1593 Johann Textor, a neighbour and relative of mine, aged about 40, when encamped at Hilden in the course of some military operations, lay down on the ground and went to sleep. It happened that some of his companions, by way of a joke, fired off a pistol close to his ear in order to wake him up. The sleepy Textor, semi-conscious, was so concussed by the report that on account of the great cerebral commotion so produced he rolled over from side to side, and with great pain vomited up crude and undigested food. When called to him I shaved his scalp and rubbed in oil of roses, administered an enema, opened his brachial vein, and administered all remedies which seemed to me necessary. At length, by the singular mercy of God, the pain in the head and the other symptoms abated, and he gradually got better, but he had lost the hearing of that ear, and did not regain it until after the lapse of a long time.
>
> (2) An old priest rang the bells in his church tower himself, a thing he had not done for many years; he thereupon felt severe pain in his head and complained of tinnitus, and became deaf. He was purged and cupped; the pain ceased, but the hearing did not return. No doubt, explains Fabricius, on account of his age and of the disruption of the tympanic membranes.

Rupture of the tympanic membrane may, and no doubt does, sometimes occur in such circumstances, but less commonly than is popularly supposed. It is quite reasonable to infer that hæmorrhages in the labyrinth, similar to those that have already been described as occurring in fractures of the petrous, may occur from commotion of the skull and labyrinth, unaccompanied by fracture, as in blows of less severity, or the shocks caused by explosions and other loud noises. Schwartze says: "In the minor degrees of commotion of the nerves of the internal ear occurring once only the disturbance of function is temporary only. It is clear from the rapid disappearance of the symptoms that there has been no gross lesion of the labyrinth. We must infer that molecular disturbances of the labyrinthine structures have occurred, or that a passive hyperæmia of the labyrinth has been brought about by transitory paralysis of the vasomotor nerves, which are, it is well known, extremely susceptible to traumatic disturbance." I am not speaking of the damage caused to hearing by repeated loud noises met with in boiler-makers, gunners, and others, but only of the damage sometimes caused by a single explosion or loud noise.

A few remarks on *rupture of the tympanic membrane* may not be out of place here. The membrana tympani may be ruptured by slender or sharp-pointed bodies which have entered the meatus, or may be destroyed by hot or caustic substances, or by steam; in such cases the wall of the meatus is also damaged more or less severely. According to Passow, the tympanic membrane is not infrequently perforated by twigs, or branches of straw, in woodsmen and field-workers, but this kind of injury is not common in England. The tympanic membrane may be ruptured by indirect violence, as in fracture of the base of the skull, and by accidents which cause increase, especially sudden increase, of atmospheric pressure in the meatus or tympanum, such as a box on the ear, a detonation, a too violent inflation through the Eustachian catheter, spasms of coughing, working in compressed air, diving, &c.

Gruber estimated that the normal tympanic membrane was able to withstand a pressure of 140 cm. to 160 cm. of mercury. He relates two cases in which, notwithstanding this great strength, the membrane was ruptured by a kiss on the ear. Such kisses must have been given with Teutonic fervour! The same author relates a case of ruptured tympanic membrane from an explosion, perhaps like the case cited from Fabricius Hildanus. At the battle of Königgratz a hand grenade exploded, killing three men and leaving an officer unconscious on the ground. Gruber found, on examination, a large perforation in the anterior and inferior segment of the tympanic membrane.

The following case was recorded by Tarrance, of Newcastle, in 1881:—

A boy, aged 16, was struck over the right ear by a snowball on January 10, 1881; he immediately felt acute pain, which made him shriek and tremble. When seen forty-eight hours later a slit-like rupture was found in the membrana tympani, behind and parallel to the handle of the malleus; there was general vascular congestion of the drum. He complained much of tinnitus aurium—a loud buzzing in the ear, with confusion in the head—which was set down to concussion of the labyrinth, and probably correctly so, as the tuning-fork was heard less distinctly in the injured ear, and a watch which should have been heard at 20 ft. was only heard at about 2 in. For days after the accident there was a feeling on the same side of the tongue as if something cold had been rubbed over it, and the taste on that side was impaired, but the sensibility of the tongue to touch remained intact. Leeches were applied to the tragus and a warm douche to the ear. There was no history of previous ear trouble. The rupture healed perfectly in a few days, but the hearing only slightly improved.

Diminution of atmospheric pressure also, though less commonly, causes rupture of the tympanic membrane as in aeronauts, or a too energetic aspiration with Siegle's speculum. A patient, whose tympanic membrane was ruptured by a box on the ear, related with great precision that he clearly felt a sensation of pressure, followed immediately by a stabbing pain in the ear at the moment that the hand which struck him was withdrawn, so that aspiration caused the rupture in this case. The hand of the assailant was wet when the blow was struck. Simple rupture of the tympanic membrane is often followed by diminution of hearing, sometimes considerable, but, unless infected, does not usually give rise to other serious consequences. Concomitant concussion of the labyrinth is often responsible for the deafness. It is popularly regarded as a grave injury, and is not infrequently the subject of actions for damages. Passow relates a curious incidence: A schoolmaster boxed a boy on the left ear, and the right tympanic membrane was ruptured. It was denied that any connexion between the injury and the blow on the ear existed, but it transpired that the boy flinched as the blow was delivered, and was knocked against the table, so that the right side of the face fell flat against the table top; this, Passow held, was the cause of the rupture.

Burns and scalds of the pinna are common enough, but the tympanum and internal ear are not often injured in this way, but instances of injury by potato stew, molten lead and iron, hot oil, steam, and other substances have all been recorded. Passow relates a case in which a drop of hot water from a tap fell directly, through the unusually wide meatus, on to the tympanic membrane of a man as he was stooping beneath a boiler and destroyed the greater part of the membrane.

The criminal introduction of irritating substances into the ear is rare, but accidents occasionally happen in which irritants are introduced by mistake in treatment, and I have known scalding water injected into the ear deliberately as a freak treatment. Sometimes patients ignorantly introduce strong irritants to relieve the pain of toothache. It is generally difficult to make out the extent of the injury. Another freak treatment I have seen is the painting of the drum with turpentine for the cure of deafness. This may result in acute otitis and mastoiditis.

Burns within the meatus heal best if protected from infection and let alone. The pain, which is generally severe, may be relieved by instillations of cocaine. Middle-ear suppuration commonly results, in which case operation will be required. Stricture of the meatus is a not uncommon consequence of burns, and requires a plastic operation.

Foreign bodies in the meatus seem to have been a sort of bugbear for centuries. A curious method for removing them was recommended by Celsus. The patient was bound upon a table, which could be tilted on a central axis, with the affected ear next the table; the table was then tilted so that the head was downwards, and the other end of the table struck with a hammer, when the foreign body dropped out. Fabricius Hildanus devoted considerable attention to the subject and devised quite an armamentarium for the extraction of foreign bodies from the meatus. Unlike many others, he seems to have used his appliances with skill and judgment, and certainly appreciated the importance of not driving the foreign body further in. He recorded a curious case in which he removed a glass bead that had been lodged in the meatus for eight years, and had caused a reflex neurosis of the type of hysterical hemiplegia on the same side. All the symptoms disappeared on the extraction of the foreign body. It is curious to read of his elaborate instrument in three pieces for extracting a pea.

If the tympanic membrane is undamaged, inert foreign bodies may be tolerated in the meatus for a considerable time, but when the middle ear has been opened, secondary infection and suppuration almost always result. Haug relates an unusual instance in which suppuration was caused by a foreign body after the mastoid operation had been performed.

A man, aged 38, had had chronic aural suppuration from childhood. The complete mastoid operation was successfully performed. After cicatrization was complete he was accustomed to keep a small plug of wool in the meatus and got careless about removing it. One day he lost a piece of wool and made violent but unsuccessful attempts to extract it. Some few days later violent pain in and behind the ear. When seen, redness and swelling in meatus and all about operation area, with renewal of discharge. In the night severe rigor. Temperature, 104·5° F. Pain on swallowing. Two days later a peritonsillar abscess was opened, through which the plug of wool was extracted.

Down to quite recent times the most lamentable histories of attempts to extract foreign bodies may be found, and even now the principles of surgery, and indeed ordinary common-sense, are occasionally, though happily rarely, set at defiance in dealing with them. The two following cases, cited from Passow, are illustrations:—

A child, aged 4, inserted a stone into the left auditory meatus. The village barber made vigorous attempts to extract it with an instrument; the result was severe pain and bleeding from the ear, and on the following day pus was discharged. Three days later the child was brought to an aural clinic. The meatus was swollen so that nothing could be seen and thick pus was issuing from it; nevertheless, some sixteen days were employed in antiphlogistic treatment and external applications "until the discharge became and remained fœtid," and then an operation was done. First an attempt was made to dilate the meatus, the foreign body was felt, and attempts made to extract it by syringing and with a hook; then the pinna was thrown forward and the posterior wall of the membranous meatus dislocated and further unsuccessful attempts made to remove the stone by syringing and with a hook; finally the posterior and upper walls of the osseous meatus were cut down, when the stone was easily removed. The mastoid, though obviously septic, was not freely opened up. Some days later the mastoid operation was completed on account of pain, shivering and fever, but the child died on the eleventh day from sinus thrombosis and pyæmia. It was suggested that there must have been ear disease before the foreign body was introduced, but this was by no means necessarily the case.

Death resulting from a foreign body in the ear is usually due to meningitis. A foreign body in an ear already the subject of suppurative otitis is an urgent indication for the mastoid operation.

In May, 1896, a child between 3 and 4 years old inserted a pip in the ear, and after due chastisement was taken by the mother to a certain Dr. L., who commenced treatment by spanking the child and boxing its ears. He then attempted to extract the foreign body by means of a bent hairpin; his endeavours were unsuccessful and several small abrasions were produced. He then advised that the child should be taken home and that oil should be dropped in the ear, and the ear syringed with warm water. On the following day he made a further similar attempt, also unsuccessful, and then suggested that an operation might be necessary. On the following day (May 11) the child was taken to an ear clinic, as the ear was already suppurating. It was seen by a certain Dr. B., who did not consider the matter urgent and told the mother to bring the child again in four days' time, or earlier if it got worse. On May 15, the day arranged for the second visit to the clinic, the child was feverish and had headache and vomited, so the parents decided not to take the journey, but called in a Dr. S., who made an attempt to extract the foreign body under chloroform but failed; the same day "brain symptoms" arose and the child died on May 20. At the autopsy pus was found in the left external meatus, pus was spread over an area 8 cm. by 2 cm. on the anterior part of the left temporo-sphenoidal lobe, and on the under surface of the cerebellum. The pus could not be washed off and so must have been in the subarachnoid space. Turbid fluid came through the internal meatus. The foreign body, swollen to twice its normal size and measuring 12 mm. by 9 mm. by 5 mm., was found in the external meatus. The tympanic membrane was destroyed and there was pus in the middle ear.

At the judicial inquiry which followed Dr. L., who first saw the case, was condemned to three days' imprisonment and 15 marks fine. This he may well have deserved, but it seems to me that the Dr. B. who, being in charge of an aural clinic, sent away a child with a suppurating ear containing a foreign body, was at least equally deserving of censure. His conduct reminds me of those children of Ephraim, spoken of by the Psalmist, who, "being harnessed and carrying bows, turned themselves back in the day of battle."

Gunshot Injuries.—The projectiles from rifles of modern construction have commonly such a high velocity that if the ear is struck the temporal bone is shattered and life destroyed; but shots from pistols, fowling pieces, and other weapons, or from rifles at a great distance, not infrequently lodge in the temporal bone, and the tympanum and internal ear are directly or indirectly injured. If the external meatus is wide and fairly straight a shot may traverse it and enter the tympanum without any external injury. Even when the petrous or base of the skull is not fractured consciousness is commonly lost, and on recovery headache, vertigo, and tinnitus are complained of. The ossicles are usually shattered, and the temporal bone more or less extensively splintered. The projectile itself is often broken. Facial paralysis is by no means uncommon. Almost without exception the injury is followed by otitis media, which may, however, heal, even if the projectile is not extracted. It is not always easy to decide, without the help of radiography, whether a projectile has lodged in the ear, for the meatus is often so swollen and injured that no view of the deeper parts can be obtained. Many of the cases observed in civil practice are attempted suicides, and some remarkable recoveries are recorded. Rollin operated on a man who had shot himself with a revolver in both ears. The operation was done four weeks after the injury. On the left side the internal carotid artery was injured during the operation, but the patient recovered.

Preysing relates the following case :—

A boy, aged 15, was accidentally shot : the bullet entered the left cheek and was lost. Two years later the ear suppurated. The bullet was extracted by a partial operation. The patient absented himself from treatment, and a year later died from meningitis. The radical operation was done eleven days before death, and a small splinter of the bullet was found lodged in the upper wall of the meatus. The presence of this foreign body had kept up the suppurative otitis, and this in its turn had caused the meningitis. This case shows the danger of leaving a foreign body in the ear, and the folly of not doing the radical operation in traumatic suppurative otitis.

The following case occurred in my own practice :—

A man received a charge of buckshot on the side of the head, in an attack by robbers in South America. The pinna and scalp were riddled by the shot, and there was free bleeding from the ear. The patient remained unconscious for several hours. When seen by me six months after the injury there was profuse otorrhœa with complete facial palsy, and the pinna and adjacent portion of the scalp were much scarred. The complete mastoid operation was done. Several shots were found in the mastoid, and two were found embedded in the bone forming the inner wall of the tympanum ; one of these had divided the horizontal portion of the facial canal. The patient made a good recovery, but refused operation for the cure of the facial palsy.

The following case by Picqué is instructive :—

A cuirassier, in a suicidal attempt, fired three shots from a small revolver into his head. On admission the next day he was torpid, but could be roused and could reply to questions. Three bullet wounds were seen, one in the occiput and two in front of the right ear. There was no issue of brain substance or of cerebrospinal fluid ; a little blood exuded on pressure from the pre-auricular openings. Hearing was impaired on the injured side, but the face was not paralysed. There were no signs of cerebral compression. Temperature 99·6° F. Picqué decided to at once enlarge and disinfect the wounds, and if possible extract the projectiles. A scalp flap was thrown down in the occipital region, and after clearing out a hæmatoma the bullet was found flattened against the bone. An incision was next made vertically in front of the pinna through the two wounds, a probe passed through the upper track led towards the zygomatic fossa, and the flattened bullet was easily found and extracted. The lower track led towards the mastoid, and the bullet was felt wedged in between the mastoid and the base of the petrous. It was extracted after chiselling away some bone. Considerable difficulty was experienced owing to the route chosen.

Pierre Delbet relates the following case :—

A man, aged 63, shot himself in the ear with a revolver at Monaco on March 22, 1906. Facial palsy immediately resulted. A surgeon opened the mastoid the same day, after a skiagram had been taken, but could not find the ball. On April 14 the man left Monaco, and on April 21 entered one of the Paris hospitals, which he left on May 19, without anything having been done. On June 6 he came under the care of Delbet. The facial palsy was complete, and behind the ear was the cavity left by the mastoid operation: at the bottom of the cavity was a fistula discharging pus. The bullet was localized by the apparatus of Contremoulins, and was on the anterior surface of the petrous at a depth of 3½ cm. from the external surface of the skull. On June 26 the skull was opened through the squama, the orifice enlarged downwards and forwards, and the anterior surface of the petrous exposed by raising the dura mater. It was then seen that there was a splinter comprising a considerable portion of the petrous; this was grasped with a Kocher's forceps and quite easily extracted. The fragment measured 1 cm. by 1½ cm., and included the tegmen tympani, and part of the superior semicircular canal, and of the facial canal. The ball was not in the fragment, it was not in the aural cavities, and it was certainly not in the brain, for the dura mater was intact. It was deemed to be embedded in the base of the skull. "At a particular moment" (the account goes on to say), "M. Contremoulins said to me: 'You are on the ball.' The field was brought well into view by lifting up the dura mater. I looked in, and replied: 'This time your apparatus is at fault, for the bullet is not there.' M. Contremoulins took his measurements again, reapplied the apparatus, and the needle reached exactly the same spot, and he assured me that the ball was there. He was quite right. The bullet was embedded in the base of the skull, and only a small portion was visible from the inner aspect. It was, moreover, covered with a copper casing, the yellow colour of which much resembled that of the bone. I had looked for a black mass and the yellow colour deceived me." The bullet was extracted, and the patient made a good recovery. Delbet afterwards made a facio-hypoglossal anastomosis, but this failed.

Principles of Treatment.

The objects of treatment in injuries of the ear, as in other injuries, are: (1) To arrest hæmorrhage; (2) to prevent or arrest infection; (3) to repair a damaged organ, and promote restoration of function. The treatment of the minor injuries need not detain us—they should be protected from infection and not irritated. The external auditory canal and the adjoining portion of the pinna should be cleansed by wiping, and the application of 2 per cent. solution of iodine in rectified spirit, and a pad of sterilized or antiseptic wool applied and changed frequently. The ear should not be syringed, as by this means infective particles may be driven to parts previously sterile.

Treatment of Fractured Base of the Skull.—This accident seems to me to be less fatal than in the time when I was a student, probably because of the greater cleanliness of the methods employed in our surgical wards. Whether there is hæmorrhage from the ear or not, unconsciousness or other cerebral symptoms may be present and demand immediate operation; in such cases decompression, subtemporal or occipital, is, in my experience, an admirable operation; and the treatment of the injury to the ear assumes a secondary place until the issue of the cerebral injury is determined. Again, lumbar puncture, by withdrawing large quantities of blood-stained cerebrospinal fluid, may be useful in intradural hæmorrhage. The immediate danger of death from arrest of respiration may thus be obviated. Repeated lumbar puncture I have found of great service, and this method is particularly advantageous in cases of cerebral irritation following intracranial injury. In other cases bleeding from the ear may be so severe as to be itself the indication for operation. In any serious bleeding the clear indication is to open the bone and seek the source of the bleeding.

Those *suffering from chronic suppurative otitis* are, because of the impaired hearing, possibly somewhat more liable to running-down accidents than are those in health, certainly such patients are sometimes the subjects of fractured base. A fracture of the skull implicating a tympanum already the seat of suppuration is extremely dangerous, and if, when the patient is seen, there is already evidence of suppuration, operation obviously offers the only chance of safety. But whenever a fracture of the base of the skull extends into the middle ear, or the cavities in connexion therewith, it is exposed to the air, and, like any other compound fracture, is liable to direct infection from without: moreover, the ear may first suppurate, and the infection subsequently pass through the fracture into the interior of the skull.

A compound fracture of the temporal bone is, as yet, not treated like a compound fracture of the femur by operation, except for some special reason such as hæmorrhage or suppuration. Though the mastoid operation would expose the fracture and allow a Listerian dressing to be applied which would prevent intracranial infection, yet this measure is seldom carried out, and is not recommended as a routine treatment. At the German Otological Society's Congress in 1909 two cases of infection of a fracture of the petrous were discussed. In the one infection occurred early, and operation was successfully performed a few days after the accident; in the other suppuration supervened later, but no acute symptoms were observed until years after the accident. Operation was ultimately performed, but was not successful.

In gunshot injuries of the ear we have not only a compound fracture, but almost always lodgment of a foreign body, which is the more dangerous the more narrow and tortuous is the track, and no time should be lost in opening up the wound and removing the foreign body. Even if the shot cannot be removed forthwith, the danger is much diminished by a free external opening and by disinfection of the wound.

In a recently published treatise on surgery the plan of treatment of gunshot wounds of the auricular region is thus clearly stated: "The principle of early intervention advocated since 1888 by Berger is now generally accepted by surgeons. The treatment appropriate to compound fractures in general should be applied to these compound fractures of the ear. The object of early intervention is to prevent infection of the fracture, the consequences of which are interminable suppuration, and often enough meningitis from infection extending through the numerous fissures which surround the track of the projectile to the meninges. The surgeon then ought to intervene, even when no grave symptom or complication forces his hand. It should be his aim to convert the narrow and tortuous track of the projectile into a breach widely open externally. This breach, freed from splinters of bone and foreign bodies, should represent a funnel with its base outwards and its apex corresponding to the projectile, and perfectly adapted to allow escape of septic fluids, or of any fragments of bone, or of the projectile, which may have escaped observation during the operation, or may become secondarily detached." The projectile, and any detached fragments of it, should invariably be extracted, if not at the primary operation at a subsequent one, for although some surprising instances of the toleration of foreign bodies have been recorded, the rule is almost invariable that foreign bodies in this region excite and maintain suppuration. An extracted projectile should always be carefully examined, and unless it is certain that the whole of it has been extracted, the retro-auricular wound should be left open. The projectile is sometimes so much altered in shape that it is not easy to say whether it has all been removed, and part of it has been known to remain behind in the cranial cavity. The help of a skiagram should, whenever possible, be obtained, and in the more difficult cases the use of a localizing radiographic apparatus is essential. The projectile may be clearly visible through a speculum and the damage apparently limited to its vicinity, but even then a fracture may extend into the cranial cavity, for fissures around the track of the bullet in almost all cases occur. If the projectile is near the jugular bulb, or the sinus, or the internal carotid artery, the danger of extracting it is certainly increased, but is less than that of leaving it, and its extraction is indeed the more urgent, for if left in situ suppuration and caries may cause fatal hæmorrhage or infective thrombosis.

In operations for injuries of the ear, such as gunshot wounds, or suppuration from lodgment of foreign bodies, and in some fractures of the middle and posterior fossæ, the way of access should be by the mastoid. The operation should be the complete mastoid operation, and any extension thereof that the special circumstances of the case may require. No one unfamiliar with the mastoid operation is likely to be successful in cases of the kind.

Treatment of Facial Palsy.—In some rare cases the injury to the nerve may be exposed, and the nerve sutured *in loco;* in others an anastomosis, hypoglossal or accessory, may be done when all other effects of the injury have passed off.

Deafness, Tinnitus, and Vertigo.—Recent cases clearly show that persistent tinnitus and vertigo of peripheral origin may be relieved by surgical measures, but labyrinthine deafness due to injury is at present irremediable.

DISCUSSION.

Dr. DAN McKENZIE wished to testify to the instructive and detailed character of both the contributions. He was particularly interested in Dr. Milligan's series of pictures which were thrown on the screen, and which showed great diligence in their collection. There had been much discussion among folk-lorists as to the origin of mutilating operations among savage tribes, amongst those operations being the distortion of the lobule of the ear. He himself thought that all operations of this kind had sprung from some simple cause, in the present case the desire to ornament the person; but as civilization advanced and fashion changed they assumed a religious and mystic character, the presence of which obscured the original simplicity of motive. He asked whether injuries produced by foreign bodies in the ear were peculiarly liable to lead to suppurative meningitis. He drew attention to a recent paper by Stenger, published in the *Archiv für Ohrenheilkunde*,[1] in which the whole subject of injuries to the ear, particularly those causing defective hearing, was gone into. Stenger dwelt on the occurrence and ætiology of labyrinthine concussion, a subject upon which he (the speaker) had hoped to hear something in the present discussion. Stenger produced upon animals labyrinthine concussion, and the lesions caused thereby were described in the article referred to.

---

[1] See *Journ. of Laryngol.*, 1911, xxvi, p. 110.

Dr. FITZGERALD POWELL said that a very interesting case of gunshot wound had come under his notice, which probably had been seen by some other members of the Section. The man was shot by a Mauser bullet, which had entered the zygoma, had gone through the meatus, and had come out at the mastoid. The drum was intact, but hearing had been destroyed. As had already been said, the medico-legal point was a very important one for the otologist. In several instances parents had brought boys to him from school with a discharge from the ears, which was said to have been caused by the teachers having boxed the boys' ears. He, however, was sceptical about "boxing the ears" causing traumatic rupture of a healthy membrane. If there were previous blocking of the Eustachian tubes, such a result might happen. He would be glad to hear Dr. Milligan's opinion on that point.

Dr. H. J. DAVIS said he remembered an interesting case from the medico-legal aspect. The man was a tram-driver, and the overhead cable broke and fell on to him. He caught hold of it with his hand, and as a consequence he received a shock of a severe character. The man who tried to lift it off him was burned also. The patient was unable to walk and became deaf in both ears completely, and in the action claiming compensation, in which he (Dr. Davis) was called to give evidence, the question arose as to whether the trouble was in the internal ear or in the middle ear. There was no reaction in the ear, and he thought the terrific electric discharge destroyed the auditory nerve. No lesion was visible in the middle ear. Vestibular reaction was absent, and it was a troublesome case to give evidence in.

Mr. MACLEOD YEARSLEY said a discussion like this called to mind a large number of cases which one had seen, and there were one or two observations which he would like to make. With regard to othæmatoma, its pathology and its occurrence in the insane, a paper was published some years ago by Fleisch on the question of syphilis in connexion with this condition. It mentioned that a large number of othæmatomata in the insane had yielded to treatment with iodide of potassium; and Fleisch suggested that as syphilis was so often a cause of insanity, it might also be the cause of the othæmatoma. He remembered seeing, some years ago, a burn of the membrane which occurred in a servant girl who had a wide, straight meatus. She was the victim of a practical joke by a fellow-servant, a footman, who had been lighting candles and thrust the glowing end of the taper into her ear. He saw the case the same day, and found a burn of the membrane, without any apparent injury to the meatus. Another interesting case, seen at the hospital, was that of a labourer who, having been told by a fellow-workman that a good treatment for toothache was to pour oil of vitriol into the ear, put a little strong sulphuric acid into it. Seen some days later he had a ring of granulations growing from the meatal wall. He did not think Dr. Milligan or Mr. Ballance had mentioned the possibility of injury to the tympanum by bodies carried in the mouth. Adams reported, in the *Proceedings of the American Otological Society* for July, 1895, the case of a child who was playing with the mast of a toy ship in his mouth. He fell, and the mast ran into the base of his skull, internal to the ramus of the jaw, passing through the floor of the tympanum, rupturing the membrane and coming into the external meatus. It pierced the tympano-meatal floor and the membrane at the same time. He was interested to hear that Dr. Milligan once opened the lateral sinus at the first incision. Two years ago he (Mr. Yearsley) did the same thing, and in the same kind of case. He was re-opening an old mastoid in a child, and in incising the cicatrix the knife went into the lateral sinus which was adherent to the scar. He had only once seen a foreign body in the nasopharynx. This was the case of a gentleman who was sent up in consultation suffering from periodical attacks of pain in one ear. The pain was so great and so sudden in onset that when he was shooting he would, according to his doctor, drop his gun and howl with pain. On examining him with the finger in the post-nasal space, something was felt in Rosenmüller's fossa and found to be a gooseberry thorn. Nobody knew how it got there. The question of prognosis in old injuries of the ear was very important, and he would like to hear members of experience discuss it, as one was often asked whether in such cases hearing in the future would be restored.

Dr. MILLIGAN, in reply, expressed his thanks to members for their appreciative remarks about the photographs he had shown. His reply must be brief, owing to the lateness of the hour. His experience of foreign bodies in the tympanum was very limited. In the cases he had seen, the body had been pushed into the tympanum in the efforts to extract it from the meatus. That would therefore make him believe that foreign bodies in the tympanum were more liable to produce meningitis because considerable force had been used and the foreign body was probably septic. As had been said, the medico-legal aspect of injury to the ear was very important. All present must have had cases in Court in which they had had to give evidence as to whether a particular lesion was the result of disease, or whether it was traumatic. He thought that unless one saw the actual injury within a few days of its occurrence it was almost impossible to swear definitely that it was not due to disease. At the same time, what had helped him very much in the estimate of these cases had been to make a very detailed examination of the other ear. On various occasions he had derived, in that way, information which would establish the fact that the condition of the ear in question was due to previous disease and not to injury. It was difficult to lay down definite statements as to how one should distinguish one class of case from the other unless the patient were seen within a few days of the receipt of the injury. With regard to the question as to whether a normal and healthy drumhead could be broken by a box on the ear, experiments had been made which showed that the healthy drumhead could withstand a pressure of five to six atmospheres. But he had seen cases in which a healthy drum had been broken by a blow on the ear. The majority of cases, however, had had previous trouble. One case was brought to him on the day the injury was received in a public school in England. The boy and the mother stated that there never had been any previous ear trouble. There was a very definite rent in the drum and he did not think there had been previous disease. He thought a healthy drum could be ruptured by a severe blow on the side of the head. The question of suppuration was closely associated with that of treatment. When there was no treatment beyond the insufflation of boracic acid, as in Dr. Hill's cases, he did not think that suppuration occurred. The cases did not usually come to the otologist first, but to the general practitioner, who probably advised syringing, and that led to infection. With regard to Mr. Yearsley's point as to the pathology of othæmatoma and its relation to syphilis, he believed that in many of the cases there was a history of previous syphilis, which had probably caused a degenerative change in the brain and arterial system, and that there was, pathologically, a chondromalacia of the cartilage. That led to the question whether there was such a thing as "spontaneous othæmatoma" even in asylums. He had talked with many asylum people about it. They had all seen, it but doubted whether it was spontaneous. Probably in most cases it was the result of trauma. He would have liked to have heard more said in regard to prognosis after injuries, especially injuries of the internal ear, particularly after fracture of the base of the skull, so far as the auditory nerve was concerned. His own experience was that the outlook was very bad. When the history was definite and there were symptoms of fracture of the skull with deafness, he had seen very few cases recover any great amount of hearing. He would also have been glad to have heard more about concussion. What was it? It was difficult to define. There were cases of concussion in which the internal ear was badly damaged. Was there not, in most of those cases of concussion, an actual hæmorrhage? In some of the cases of concussion in which the hearing was bad for a time, there was a subsequent recovery of a good deal of hearing power. He thought that was because there had been a hæmorrhage, which had been partially recovered from. He agreed with Dr. Hill as to the comparative infrequency of facial paralysis in fracture of the base. It was remarkable how many cases one saw in which the auditory nerve was severely damaged but the facial nerve escaped injury. Another point which he was surprised had not been dealt with was one which had a very important medico-legal aspect—namely, whether an injury to the ear in the case of an old suppurative condition had any relation to the development of malignancy. He did not see why it should not. For instance, malignant disease of the breast was said to often follow a blow, and injuries of long bones were frequently followed by malignant degeneration.

Fifteen Specimens of the Temporal Bone, showing Types of Fractures.

By Arthur H. Cheatle, F.R.C.S.

Nos. 1 to 4 inclusive: Double line of fracture separating the roof of the Eustachian tube, middle ear, and meatus in one piece. Numbers 1 and 4 are of the diploetic infantile type of bone. In No. 4 the membrana tympani and ossicles were intact.

Nos. 5 and 6: Very like the preceding specimens, but the posterior line of fracture involves the posterior meatal wall and exposes the antrum.

No. 7: The posterior line of fracture extends still further backwards to the posterior fossa, and involves the upper part of the sigmoid groove. The separated part is comminuted.

Nos. 8, 9 and 10: Line of fracture more transverse, passing through the roof of the middle ear tract and mastoid cells to the posterior fossa externally to the facial nerve. In No. 9 the outer fragment is comminuted.

Nos. 11 and 12: Line of fracture passes through the bone just externally to the bony labyrinth and through the facial nerve as it leaves the shelter of the external semicircular canal. In No. 12 the outer fragment is comminuted.

No. 13: Transverse fracture passing through the semicircular canals and facial nerve, and a longitudinal fracture through the middle ear and meatus.

No. 14: Transverse fracture through the middle ear, carotid canal, vestibule and facial nerve.

No. 15: Transverse fracture through the Eustachian tube, front part of middle ear, cochlea, facial nerve, and internal auditory meatus. The intact modiolus is exposed on the outer aspect of the inner fragment.

Specimens of Fractured Temporal Bone.

By G. J. Jenkins, F.R.C.S.

(1) Right temporal bone of female, aged 1½ years. The fracture involves the roof of the middle-ear tract. Blood in the middle-ear tract, but not in the external auditory meatus.

(2) Right temporal bone of female, aged 41. Fracture involving the roof of the tympanic cavity and Eustachian tube, and inferiorly extending into the Glaserian fissure. The tympanic membrane is not ruptured. There is blood in the middle-ear tract, but not in the external auditory meatus. The section shows blood-staining of the air-cells of the mastoid element. Clots were removed from the air-cells and antrum.

(3) Right temporal bone of male, aged 69 (previously described and exhibited). Fracture involving the middle-ear tract; blood in the middle-ear tract but not in the external auditory meatus.

(4) Fracture of the left temporal bone of female aged 1½ years. The fracture involves the middle ear anteriorly at petro-squamosal antrum and Glaserian fissure. Externally it involves the postero-superior part of the external meatus, and extends to the parietal notch of the squamous element. There is blood in the middle ear and the external auditory meatus. The tympanic membrane is not ruptured, but there is a small perforation of the skin of the meatus postero-superiorly close to the tympanic membrane.

(5) Fracture of temporal bone in which the fracture involves the external auditory meatus. A large effusion of blood under the skin of the external auditory meatus fills up the lumen and prevents a view of the tympanic membrane.

Mr. G. J. JENKINS remarked that in the specimens (1), (2) and (4), in which the fracture did not involve the internal ear, he had made sections through the labyrinth, and found in specimen (1) there was blood in the neighbourhood of the foramen rotundum, and in specimens (1) and (4) there was blood at the fundus of the internal auditory meatus and in the auditory nerve. He suggested that these specimens supported Sturges's experiments.

Otological Section.

May 19, 1911.

Mr. Arthur H. Cheatle, President of the Section, in the Chair.

Deafness due to Occlusion of Eustachian Tubes by Scar Tissue.

By D. R. Paterson, M.D.

Female, aged 21, had suffered for about six years from congenital specific disease which affected the throat, leading to destruction of the soft palate and part of the hard, and to ulceration of the posterior and lateral walls of the pharynx. Various forms of anti-specific treatment had been tried from time to time, but the parts repeatedly broke down and led to loss of tissue, until eight months ago, under intramuscular injections of metallic mercury, healing was finally established. Since that time patient began to suffer from deafness, which has persisted with little or no alteration. There are no subjective sensations. Hearing: Right ear—conversational voice, 10 in.; whisper, 1 in.; left ear—conversational voice, 2 ft.; whisper, 3 in. Bone conduction is increased. The drums are flaccid in appearance. In the pharynx there is a large gap between the nose and throat, the posterior wall being formed by a dense white cicatrix. There is no appearance of Eustachian tubes.

Suggestions as to treatment were invited.

DISCUSSION.

The President (Mr. Arthur Cheatle) remarked that the question was, what could be done to improve the patient's hearing? Something ought certainly to be done. To attack it from the Eustachian orifice region was impossible, in his opinion. Something must be done through the meatus. As a preliminary, a hole should be made in the membrane to see if improvement took place, and if so, further measures could be considered. As it was impossible to keep the opening in the membrana tympani patent, the question was, what operation should be done? Whether the membrane, the malleus, and the incus should be removed, or whether the incomplete mastoid operation would help.

Dr. MILLIGAN said the condition was so beautifully healed that he would not interfere. In the case of a syphilitic lesion like this, where there had been so much trouble to get it to heal, he did not think it advisable to undertake any further operative interference.

Mr. A. L. WHITEHEAD agreed with Dr. Milligan's opinion. He thought the deafness was not progressive, and the history was that it had been quiescent for some time. It was now satisfactorily healed. In many such cases there were other changes which vitiated the result of such an operation as the President suggested—i.e., even although there might be improvement in the middle ear, it was unlikely that the hearing would get better. Some of these patients had other changes, and this girl had marked interstitial keratitis, indicating that she was the subject of extensive congenital syphilis. His experience of that kind of case was limited to one, and in that disaster resulted. He succeeded in opening the nasopharynx; he did not do the operation which the President suggested, but at a subsequent date the patient had acute nasopharyngitis, followed by acute infection of the middle ear, acute meningitis, and death. Of course that might have come on apart from the operation, but it did not encourage one to interfere in such cases. He agreed it would be wiser to leave it alone.

Mr. WEST remarked, speaking in ignorance of the practical results of the procedure mentioned, that if a preliminary opening in the membrane brought about immediate improvement in the hearing, it was worth while trying to make a permanent air vent in the tympanum. He would be inclined to do it through the mastoid, opening the antrum, and putting a tapering skin graft into the antral opening, so as to get immediate healing of the damaged surface and a permanent fistula. Possibly that would give good results.

Mr. SYDNEY SCOTT asked what was the low limit of tone perception in Dr. Paterson's case. If that limit were much raised he would not touch the patient, but if the lowest tones were audible the procedure suggested by Mr. West might be a feasible one.

Dr. WATSON-WILLIAMS agreed that the case should be left alone. His experience was that in these syphilitic cases satisfactory results were very difficult to get when interfered with surgically.

Dr. PATERSON, in reply, said he ought to have mentioned that in the right ear a perforation existed for a short time, and during the time there was no improvement in hearing on that side. He had an open mind as to what was best to be done. The question was one that should be put clearly before the patient, telling her that possibly her hearing might be worse after the operation. In answer to Mr. Scott, he could not now say from memory what the condition of hearing for low tones was, as he had not recently done the tuning-fork tests. When deafness first came on he tried to find out the position of the Eustachian tubes and had made incisions at several points, but without result. There was much destruction of tissue, a loss of the whole soft palate and much of the hard, and the whole lateral walls and the posterior wall were one large raw surface. It had now healed, and the resulting hole was much smaller than it was six or eight months ago. He considered it impossible to find the Eustachian tube from the nasopharyngeal side, and he thanked the members of the Section for their suggestions.

A Case of Erysipelas of the Head: Invasion of the Right Auditory Meatus, where an Abscess formed with extension backwards to the Sterno-mastoid Muscle, and Invasion of the whole Sheath of that Muscle.

By Arthur H. Cheatle, F.R.C.S.

A DUSTMAN, aged 49, had an attack of erysipelas, which began in the forehead along the line of his leather dustman's hat.

The right meatus became involved and an abscess formed on the posterior superior wall at the junction of the cartilaginous with the bony meatus. The abscess in the meatus discharged freely, and after fourteen days a swelling came behind the ear and spread down the upper part of the neck, when he came to the hospital. The case presented all the appearances of a mastoid abscess with Bezold's perforation. At the operation a large amount of pus was evacuated, and a ragged hole was found in the meatus. The fibres of the sterno-mastoid at their attachment were a mass of sloughs. The abscess was opened to half-way down the neck. The mastoid cells were opened and found to be normal. Subsequently it became necessary to drain the whole sheath of the sterno-mastoid. Healing has occurred, and the membrane is normal. The site of the abscess in the meatal wall can be detected by the scarring. The patient stated that the hearing was good throughout, except when the meatus was blocked with discharge. The pus was sterile to culture.

Three Specimens of Chronic Middle-ear Suppuration in each of which the Opposite Side was Normal, the six Bones being all of the Diploëtic Infantile Type.

By Arthur H. Cheatle, F.R.C.S.

Among forty-two individuals in which both temporal bones were removed, three were found to have suffered from chronic middle-ear suppuration in one ear, the other being normal. On section all six bones were found to be of the diploëtic infantile type.

Pus was present in the antrum of the suppurating bones.

(1) Male, aged 49. Right side: Complete loss of membrane, malleus and incus.

(2) Female, aged 54. Left side: Perforation involving the posterior superior segment; loss of the incus.

(3) Female, aged 77. Perforation in Shrapnell's membrane. Details were destroyed accidentally by the section.

DISCUSSION.

Mr. Cheatle showed the specimens as forming another link in the chain of evidence which showed how important that type of bone was in producing chronic middle-ear suppuration. He did not think that the problems presented by middle-ear suppuration could be understood unless the type was well recognized. He thought that the subject of suppuration of the middle ear, either acute or chronic, needed re-writing in the light of the recognition of the type. He regarded the term "infantile" as rather a mistake, because he thought that in infancy it was settled what the bone was going to be, whether cellular or diploëtic. He thought the type should be known as the "Diploëtic," always remembering that a dense outer antral wall was present and that a dense layer of bone separated the cavity of the antrum from the diploëtic mastoid mass.

Mr. West remarked that all the members, whether they were prepared to go as far as the President with regard to the diploëtic type or not, were bound to admit that those six bones formed a link—and as far as they went a rather good one—in the President's argument. That in all the three cases there should have been suppuration on one side, while the other side had no history of suppuration, and that in all six bones there should be that particular type of anatomy, was an apparently striking conjunction of facts; but he did not really think Mr. Cheatle's case would have been weakened by disagreement, nor was it much strengthened by agreement between the two sides. The experience of most people had been that it by no means followed that because the bone on one side was of the compact type, therefore the bone on the other side was of the same type. Supposing, therefore, that the other mastoids had been cellular, it would have been admissible to say that it was but a small group of cases, that it was a mere coincidence in these cases, and that it meant nothing. From the point of view of those who doubted the certainty of the significance of the infantile type as a primary condition, the converse held, that the argument from these six bones was not as strong as at first it appeared to be.

Mr. CHARLES HEATH said he considered that symmetry was the rule, although exceptions were occasionally seen. He did not consider that exceptions were as frequent as Mr. West suggested. The point was, that before operating on an ear it was not known whether the skull was of the infantile type or not; and once an operation was commenced it had to be gone through with. Therefore the difference was more a pathological than a practical one.

Dr. DAN MCKENZIE considered that it was desirable, if possible, to ascertain which kind of bone one was dealing with; and recently work done in X-ray departments had made the matter much clearer.

Dr. MILLIGAN said that although he admitted that the President's theory held good up to a certain point, and that the infantile type of temporal bone was very important in practice, he thought they were running away from the pathological aspects of many temporal bones. Many bones which were classed as of the infantile type were osteo-sclerotic, and that condition was of pathological origin.

Mr. SYDNEY SCOTT said that anyone who went through Mr. Cheatle's collection and compared what was demonstrable there with what one found during operative procedures could not fail to agree with Mr. Cheatle's teaching on that subject.

Mr. CHEATLE, in reply, said he knew that the type was not always symmetrical; sometimes the cellular type of bone was found on one side and the diploëtic type on the other. He agreed with Dr. Dan McKenzie that the X-rays would be a help in the future in ascertaining the type of bone in the living subject before operating. In answer to Dr. Milligan, he was content to leave the matter to future investigations; he was convinced it would be found that the type mentioned did exist, that it was largely responsible for chronic suppuration, and that the density of the outer antral wall was not due to osteo-sclerosis. There might be some little osteo-sclerosis in the cells which lined the outer antral wall, but it was absurd to think that the density of bone associated with the type which could be seen all through life, at all ages and in both sexes, without any evidence of disease, was due to suppuration. He could not believe it.

Dr. Milligan desired to explain that he believed in the infantile type, but he also believed in the osteo-sclerotic type.

Mr. Cheatle, in further reply, said he wished to make the position quite clear. Most of the cases of chronic suppuration, apart from tubercle, began in scarlet fever or measles, or some other infective disease. If the antrum became infected in a cellular bone a mastoid abscess resulted, but if in the diploëtic type several things might happen: (1) It might get well; (2) it might cause complications in the middle or posterior fossa, or in the labyrinth; or (3) it might result in a chronic discharge from the middle-ear tract. His point was that cases of chronic suppuration mostly originated in scarlet fever or measles, and that the type was responsible for the condition.

Periotic Bones of Fossil Cetacea.

By W. Jobson Horne, M.D.

The inner ear of the Cetacea is contained in the periotic bone, which is not attached to the other bones of the skull by an osseous connection in many species. When, therefore, maceration occurs, the bone may drop out. The specimens shown are fossilized examples of the periotic bone.

The chief interest is to be found in specimen No. 1. It will be observed that the round window is not completed in its circumference, but tails off posteriorly into a narrow cleft, which, so far as can be seen, reaches downwards to the aqueduct of the cochlea in its whole length. It is interesting to note that this condition is not found in specimens Nos. 2 and 3, and in specimen No. 4 the bone has been broken at a point in the cochlea above the junction of the aqueduct, so that it is not possible to say whether the cleft existed in this specimen. The condition has not been described in any of the living Cetacea, and, indeed, has only once been referred to at all in any animal, this being in the case of the dugong. In the latter the cleft is much larger.

For the above description Dr. Jobson Horne is indebted to Dr. Albert A. Gray.

DISCUSSION.

Dr. Gray remarked that these fossil bones were very interesting. With regard to the period in geological history to which they belonged, those particular ones were probably not earlier than the Miocene or Pleiocene strata, although the cetacea were found in the Eocene. The chief interest lay in the fact that if one found the round window and traced it round its lip there was, in one of the specimens, a cleft running backward along the floor of the aqueduct of the cochlea—i.e., the round window was not a complete ring. He found this first in the Sirenia, but had not observed it in any of the living Cetacea. Fortunately, however, Dr. Westmacott that day showed specimens from a whale caught near Spitzbergen, in which the cleft was very marked—i.e., the aqueduct of the cochlea is not a complete bony tube, but is merely closed below by a membrane. The cleft runs right up to the round window. This was interesting physiologically because it showed that the labyrinth was not a closed cavity, as we had been taught to consider it. There had been a discussion about the effect of increased tension in the labyrinth, and much had been said about affections of ossicles causing differences in the tension of the labyrinthine fluids. In the human subject the aqueduct of the cochlea was no thicker than a hair, but in most animals it was a wide tube, and there could be no differences of tension between the fluid in the labyrinth and that in the cranium. The only condition in which such differences could occur would be when the aqueduct of the cochlea became blocked up. Much had been written and spoken about the physiology of the labyrinth and the pathological conditions in the organ due to changes in tension, but those statements should be received with great caution.

Dr. Gray suggested to Dr. Horne that the value of the specimens would be greatly increased if he were to have them cut across in the proper planes by the lapidary ; because in that way one might find the link between the cochlea of the ornithorhynchus and that of other mammals. It was remarkable that there should be no transition stages between the slightly bent tube of the monotreme cochlea and the one and a half turns of the cetacea. Possibly these fossils might show it. The sections might also show the semicircular canals, hardly larger than those of the mouse, in situation.

Dr. JOBSON HORNE, in reply, said that he fully concurred with the views that had been expressed by Dr. Gray. As the specimens were of unusual interest, he (Dr. Horne) had decided to have them photographed, before being cut by a lapidary. Subsequently the sections themselves he would have photographed. He would show the sections and photographs at a future meeting of the Section.

Labyrinthine Vertigo (Menière's Symptoms—Non-infective) treated by Operation.

By G. J. Jenkins, F.R.C.S.

M. T., aged 23, single. Occupation, cutter; has never worked in a noise. The patient complained of vertigo and tinnitus and deafness in the left ear.

Previous history: Pneumonia at the age of 6. Abscess in the neck (scar at the angle of right lower jaw) soon after the pneumonia. Measles at the age of 13. Influenza two and a half years ago. The patient has often complained of cold extremities to members of her family.

Family history: An older sister has chronic otorrhœa both sides, following scarlet fever. Mother confined to the house with osteoarthritis. No history of nerve disease in the family.

History of the present condition: Tinnitus—a "steam escaping" noise was the first noticed symptom and came on about four years ago in the left ear. For the first few months the noises did not annoy the patient very much, but subsequently she consulted an aural surgeon, who treated her for anæmia. There was no improvement under treatment. Immediately after the influenzal attack the noises became much worse, and have troubled the patient up to the present time. There appears to have been some improvement in the condition at times. Vertigo—the first attack came on about nine months to a year ago, when standing cutting cloth in the workroom. During the attack, which lasted about half an hour in the acute stage, the patient said that "everything seemed to be tearing round" (? direction), and even when supported she had to "hold on" to steady herself. Patient does not remember in which direction she was inclined to fall. Soon after the onset of the attack there was vomiting and nausea which lasted about an hour. There have been attacks off and on since the above—once or twice in the month, usually associated with vomiting and increase in tinnitus. Between attacks the patient has been able to walk about without assistance, but unfit for work. Deafness—impairment of hearing noticed four years ago, but thought to be due to the tinnitus. Much worse after influenza. Patient not sure whether or not deafness increased after attacks of vertigo. There is no history of earache nor aural discharge at any time. Since the influenza she has often had severe left occipital and left upper parietal headaches.

Condition on examination (February 17, 1911): Tympanic membrane pale both sides; no cicatrices. Hearing—C^2 fork: Weber to the right, Rinné positive, both right and left. Bone conduction (Schwabach): right normal, left much diminished. Conversational voice—right, 18 ft. + ; left, 3 ft. to 4 ft. Whisper—right, 18 ft. + ; left, about 1 ft. Inflation did not improve hearing on left side. Eustachian tube patent. Rombergism—fell to the left and backwards; could not walk along straight line, falling usually to the left side; vertigo readily produced by quick movements of the head; rotation tests not applied. No spontaneous nystagmus. Caloric tests—cold water did not produce nystagmus from test to either ear. Vertigo was well marked and prompt in appearance in case of right ear, but in left was delayed and less marked.

Physician's report: The patient appears healthy except for the vasomotor disturbance noted above. There is no evidence of any lesion of the central nervous system. Blood-pressure 117 (R. R.). No blood disease. No nucleo-albumin in the urine. No evidence of exophthalmic goitre. Eyes normal.

The patient was ordered acid hydrobromic mixture and treated as an out-patient. She did not improve, and was taken into the German Hospital and treated by absolute rest in bed and given a diet of milk only; the hydrobromic acid was continued. During the ten days under treatment in hospital there was no return of her trouble whilst in bed, and only a slight attack of vertigo when allowed to move about the ward. She left hospital at the end of about two weeks, and continued treatment at home on a slightly fuller diet.

March 3.—Patient had a very severe attack of vertigo two days ago. Fell to the left and struck her nose, causing it to bleed. No vomiting, but tinnitus much worse. Rinné (C^2), right and left positive; Weber, (C^2) to right. Bone conduction (Schwabach) much diminished to left side. Conversational voice—right, 18 ft. + ; left, 1 ft. Whisper—right, 18 ft. + ; left, 2 in. to 3 in. Patient nervous and frightened. The patient looked ill and was again admitted to hospital for observation. She had two attacks of vertigo, during which she had to be supported if she attempted to move in the ward. There seemed to me evidence of increased labyrinthine pressure, and, convinced of this, I decided to relieve the tension by operation.[1]

The operation: By a post-aural incision the antrum was opened.

---

[1] I find that Mr. Arthur Cheatle had, in 1897, thought it possible that operative treatment might be advisable in such conditions (see *Archives of Otology*, New York, 1897, xxvi, p. 185).

The bone was healthy. By means of a very fine burr the membranous external semicircular canal was exposed for about 1·5 mm. and opened. My object was, if possible, to open only the peri-lymphatic space. There was no obvious escape of peri-lymph or blood. At the time of the opening of the canal the anæsthesia was light and a definite effect similar to shock was noticed. The pulse was slow and small. The patient became very pale and perspired. No deviation of the eyes was noticed (but might have been missed in the attention devoted to the general condition). A fine wick drain was placed in the lower part of the wound from the canal to the surface, and the upper part of the wound closed. Dressed with double cyanide gauze.

Three hours after the operation there was severe vertigo, spontaneous nystagmus, and almost continuous vomiting. Face upwards the nystagmus was very coarse, rotatory left to right, and oblique up and to right. This nystagmus present in all positions of the eye. The night following operation patient slept badly though given potassium bromide *per rectum*, and nausea very troublesome. On day following operation vertigo severe on slightest movement of the head. The wound was dressed and the gauze forming the immediate dressing was soaked with serous fluid. Two days after the operation the patient was feeling comfortable and sleeping well, though occasionally suffering from nausea. Vertigo readily produced by movement of the head. The voluntary nystagmus almost purely rotatory, but there was some horizontal movement. Conversational noise heard left side 6 ft. +. Volunteered statement again that noises had entirely ceased. Four days after operation the gauze wick was removed and much serous fluid escaped, as if it had been dammed back. Six days after operation, still spontaneous nystagmus similar to that described above. No tinnitus. Conversational voice heard in affected ear 15 ft., single words in some cases 12 ft. Cold water (ice cold) in the left ear did not produce any obvious change in nystagmus. When patient sat up there was tendency to fall to right.

Patient was out of bed fourteen days after operation, and left the hospital a few days later. She had some return of the tinnitus in a mild form on the sixth day after operation following the syringing.

May 6 (four weeks after operation): Hearing—conversational voice, right, 21 ft. + ; left, 15 ft. Whisper—right, full ; left, 3 ft. A well-fitting rubber plug, smeared with vaseline, was put in the right meatus when testing the left ear, as was done before the operation. With Bárány's Lärm apparatus in the right meatus, conversational voice was heard only about 6 in. by left ear. Highest tones of Galton whistle well heard; low tone limit below C^{-}. Rombergism—stands steadily with eyes shut; can walk straight line better than before operation. Vertigo readily produced by movement of the head in plane of the left posterior semicircular canal. Nystagmus—very fine indefinable movement on looking to the right. Caloric tests—tap cold water (for two minutes) does not produce alteration in nystagmoid movement. Rotation tests—vertical position: No nystagmus produced by rotation in either direction; left to right rotation produced vertigo, patient tending to fall to right; right to left rotation produced less marked vertigo, with tendency to fall backwards. There is a mild tinnitus which does not, however, annoy the patient.

DISCUSSION.

The PRESIDENT said the case was important because, as far as he knew, it was the first time that the procedure had been adopted for the relief of vertigo, tinnitus, and deafness. It was also important from a pathological point of view, as well as the physiological, because of the results obtained.

Mr. SYDNEY SCOTT said it was necessary in such cases to emphasize the fact that the vertigo was not of that type the pathology of which members knew. In this case one did not know what the vertigo was due to, because there was no middle-ear suppuration. He would like to see an indication of that embodied in the title, to distinguish it from labyrinthine vertigo secondary to middle-ear disease.

Dr. DAN MCKENZIE regarded the case as very suggestive; it opened the door to many trains of thought. There were one or two points, however, which bade one be cautious. One was the possibility of the vertigo being due to some other disease in the labyrinth. He did not see a note of an attempt being made to discriminate between the labyrinthine type of vertigo and the cerebellar type by altering the position of the patient's head in testing for Rombergism. Putting those possibilities on one side, and assuming it to be labyrinthine vertigo, it was a case which would encourage us to perform the same type of operation in other cases of the same kind. A most interesting question was, why had the hearing improved so much? There were several possibilities, one being (*pace* Dr. Albert Gray) that the relief of the tension by the escape of fluid might account for the improvement of the hearing. But he thought it was more likely that by his operation Mr. Jenkins provided a new fenestra, and by that means it was possible that the sound waves reached the cochlea in a better manner than they did formerly. The objection to this explanation was that the case had shown no sign of middle-ear disease. After the operation there was no evidence in testing the labyrinth that the semicircular system was still active. It seemed as if the operation had destroyed the function of the semicircular canal, and so removed the vertigo. Possibly the operation destroyed the action of the semicircular canals, but did not destroy the cochlear action; from which one might argue that it was possible to have a lesion in the semicircular canals definitely localized to that part of the labyrinth, and not affecting the cochlea at all. It was, of course, possible to have suppuration definitely localized to the canalicular system and sparing the cochlear part of the labyrinth.

Mr. JENKINS, in reply, said he agreed to Mr. Scott's suggestion, as it was likely there might be confusion from the title given (the title has been altered accordingly). Bárány's tests were applied and there was no evidence of cerebellar vertigo. He thought the labyrinth was certainly active on the affected side, as the caloric tests produced definite vertigo when applied to that side.

A Case of Leprosy with Involvement of the External Ears, Palate, Pharynx, &c.

By G. N. BIGGS, B.S.

FULL notes of this case, which is shown by permission of Sir Malcolm Morris, K.C.V.O., will be found in the *Proceedings of the Royal Society of Medicine* (Dermatological Section), 1909.[1] Since this date the patient has been treated with injections of nastin and a vaccine of *Staphylococcus aureus*.

It will be seen that the auricles are much enlarged, and present the typical shiny and thickened appearance which is characteristic of the disease. There are tubercles present, and in some cases these have broken down and ulcerated. The tubercles are most numerous on the palate, but also extend down and involve the vocal cords and ventricular bands. Ulcers due to the breaking down of tubercles are also present in the nasal cavities.

---

[1] *Proceedings*, 1909, ii (Dermat. Sect.), p. 41.

A Case of Syphilitic Disease of the Nose treated with "606."

By G. N. Biggs, B.S.

Patient aged 32; three children all healthy; no miscarriages. Has suffered from sore throat and discharge from the nose for five years. This patient has been thoroughly treated by mercury and potassium iodide by the mouth in large doses, and mercurial inunctions, over a period of eighteen months, and in spite of this the disease has been progressing rapidly. In January last the disease became much more active; there was extensive ulceration of the soft and hard palate, and the nasopharynx, the septum, and turbinate bones had been completely destroyed, and the nasal cavity was extensively ulcerated.

Ulceration commenced at the alæ nasi and extended over the rest of the nose, assuming almost the appearance of a malignant growth. On April 1, 1911, an intravenous injection of salvarsan (0·4 grm.) was given by Mr. R. D. O'Leary, the Resident Medical Officer at the Seamen's Hospital. At the present time—i.e., six weeks after the injection—it will be seen that the ulceration has quite healed and the growth quite disappeared.

Photographs of the patient before the injection and ten days after are exhibited to show the rapid effect of the drug.

The case is of special interest as it was one which, in spite of very active treatment, was rapidly progressing, and would have rapidly terminated fatally in the absence of this drug. The Wassermann reaction before the injection was positive.

Notes of a Case of Temporary Deafness and Blindness due to Intestinal Toxæmia.

By A. L. Whitehead, B.S.

G. M., male, aged 53. Good general health, and no defect in hearing or in vision, beyond a little short-sight, up to March 5, 1910. About that date he began to suffer from headache with general malaise, but without elevation of temperature; this continued up to March 12, on which day the headache was more intense, and he noticed that his hearing was defective. This defect rapidly increased, so that by 5.30 in the evening he could not hear even the loudest voice with either ear; about the same time his vision commenced to fail, and in about an hour he had lost even perception of light in both eyes. His medical attendant, Dr. Hogarth, of Morecambe, saw him at this time and found almost complete deafness, only very loud noises being heard; no tuning fork tests were made. No perception of light was present, the pupils were dilated and immobile, disks pale, retinal arteries reduced to threads, veins normal. Knee and plantar reflexes normal, no paralysis of any muscles; mind clear; temperature normal.

A previous history of constipation caused the administration of a large dose of calomel, this was followed by a copious evacuation of very offensive fæces, and about 10.30 the following morning the hearing commenced to return, and was, in a few hours, restored to normal. His vision gradually returned during the same day, but for some days was not equal to what it had been prior to the attack. On March 19 he was seen by me, and the hearing found to be normal; the membrana tympani, nose, &c., all being normal. Vision on this date was normal, with glasses correcting a small degree of myopic astigmatism, although the disks still appeared a little pale and the arteries small.

This seems to have been a case of intestinal toxæmia, causing spasm of the arteries, and so anæmia and temporary abolition of function of the optic and auditory nerve-endings. In the recorded cases of anæmia of the labyrinth, tinnitus and vertigo seem to have been always present; in the present case these symptoms were entirely absent. There has been no return of any aural or ocular defect during the twelve months since the attack.

DISCUSSION.

The PRESIDENT said it would be difficult for Mr. Whitehead to convince members scientifically that the case was one of intestinal toxæmia. If that were the cause of the condition, as constipation was so common, why were not temporary deafness and blindness more frequent accompaniments? He asked whether there was any question of neurosis in the case, or had the patient been taking any drugs, such as quinine, salicylates, or cinchona?

Dr. BRONNER suggested that the faradic current should be tried in such a case. A few days ago he had a patient who said she was nearly blind and that she could not hear anything. Her doctor thought it was syphilis of the brain. The faradic current was applied and the hearing and vision improved at once.

Dr. WATSON-WILLIAMS said the case was so obscure that at best one could only suggest as the cause something in the nature of a hypothesis. At a previous meeting he had brought up a case of recurring temporary deafness, the deafness coming on in a few minutes, remaining absolute for a few hours, and then passing off. That happened on several occasions, and was increased by exertion, and the patient died with symptoms pointing to cerebral hæmorrhage. No autopsy was allowed. He thought there was strong evidence in that case that there was some organic pathological vascular condition, and possibly some such explanation might account for the temporary deafness in the case now exhibited.

Dr. DAN McKENZIE asked what was meant by the phrase "anæmia of the labyrinth." Probably there was such a disease, but he did not know on what facts the diagnosis was based.

Mr. WHITEHEAD, in reply, said the patient was healthy and had been taking no drugs. There had been perfect health before this condition occurred. There could be no suspicion of hysteria. "Anæmia of the labyrinth" was a phrase of Politzer. He brought the case forward because it was a puzzle to him. The only suggestion he could make was that it was due to intestinal toxæmia. As Dr. Watson-Williams said, the defect was clearly vascular. One would think, from the condition of the retinal vessels, that the vascular supply to the nerve-endings of the labyrinth was much reduced. He believed there were cases analogous recorded in ophthalmic literature, due to fungus-poisoning, in which there was temporary anæmia of the retina. But a case of the kind in otology he believed to be rare. The only reference he could find was a casual one in Politzer's book.

Sequestration of Bony Labyrinth in a Boy aged 4.

By F. H. WESTMACOTT, F.R.C.S.

THE boy had tuberculous disease of each temporal bone, and was operated upon on both sides. Each ear showed extensive tracking in various directions. The sequestrum came from the right side some months after an incomplete mastoid operation. There was facial paralysis early in the disease on this side. Complete recovery resulted, but with permanent paralysis on right side.

The PRESIDENT characterized it as one of the most beautiful specimens of the kind in the country.

Eight Specimens of the Otic Element of the Arctic Whale.

By F. H. Westmacott, F.R.C.S.

These specimens were typical and selected examples, obtained from Spitzbergen, and showed the anatomical features in a clear and distinct manner.

Cast of Cavity in the Left Mastoid Process of a Woman aged 75, filled with Cholesteatoma—smooth polished surfaces.

By F. H. Westmacott, F.R.C.S.

History of scarlatinal otorrhœa forty years previously. Death followed from asthenia five days after operation; no suppuration occurred.

DISCUSSION.

Mr. Hugh E. Jones asked for what symptoms the operation was performed in the last-named case.

Dr. H. J. Davis said he had at present a case of a large cholesteatoma in a boy, and there was an enormous erosion of bone; on removing the pultaceous mass the lateral sinus gave way. There was very severe hæmorrhage, and later secondary hæmorrhage, but he was now going on well.

Mr. Westmacott, in reply, said the operation was performed for intense pain over the mastoid and extreme tenderness. There was a history of otorrhœa forty years before, and some cholesteatomatous material in the meatus. The cavity was a large one, the upper limit being far above the normal level. There was no opportunity for an autopsy, but there must have been a good deal of elevation of the roof of the temporal bone in the cranial cavity.

Eight Cases of Mastoid Operation.

By Charles J. Heath, F.R.C.S.

Case I.

Miss N. The right ear had suppurated practically all her life, with the exception which the patient's letter indicates: "I have always intended telling you, as Dr. Wyon said it would be of interest, that two years previously to coming under your observation the right ear, which had freely discharged all my life, stopped doing so for six months, during which I had facial paralysis. I went under medical treatment. I was in Scotland at the time, but the doctors did not seem able to understand it; however, when my face regained its normal condition, the ear started discharging again, which continued until you operated." The radical operation was performed as the ear was utterly disorganized. The left ear subsequently became involved without any apparent cause, and after suppurating for a few weeks, the patient came again under observation. Operation was performed without delay, because, symmetry being the rule in tympanic conditions, the course of suppuration in this ear was likely to be a destructive one, as it had been in the other. The hearing of the other ear was already defective. No cholesteatoma was found, though the antrum was carious, and as the bony elements appeared to be intact, the conservative operation was performed. Recovery was rapid, and the perforation healed. Some time afterwards tinnitus supervened and became severe and continuous. The hearing also became impaired. After much hesitation and delay, and consultation with her doctor, it was decided to proceed to the radical operation.

This case shows that in some cases fairly early conservative operation may put an end to the disease, yet not restore perfectly satisfactory conditions. It still appears to justify reasonably early interference. In this case, as in most of the cases exhibited, it will be seen that the antrum has been induced to fill up, thus obviating the necessity for occasional subsequent removal of desquamated material.

DISCUSSION.

Mr. Tod asked what Mr. Heath meant by saying that the antrum had been induced to fill up?

Dr. MILLIGAN asked what was meant by "the left ear subsequently became involved without any apparent cause, and after suppurating for a few weeks, the patient came again under observation. Operation was performed without delay, because, symmetry being the rule in tympanic conditions," &c. He asked whether no attempt was made to treat the ear by local measures. It could not have been anything very acute.

Mr. STUART-LOW said that in a considerable proportion of the cases that he had seen in which the so-called "conservative operation" had been performed, all that was accomplished could have been attained if the simpler and safer subcortical operation only had been done. If the teeth, the nose, and the pharynx were attended to and cleared of disease and disturbance, and the mastoid antrum opened and drained, the great majority of recent antral suppurations soon got well, and no further operation such as Mr. Heath advocated and performed in this case was necessary.

Mr. WHITEHEAD asked what was the nature and extent of the disease found in the mastoid antrum at the time of the operation, since the reported notes gave no information on this point. Would Mr. Heath also state precisely what was the degree of hearing before and after the operation.

Mr. HUGH E. JONES remarked that the patient heard his watch at 15 in. after the radical mastoid operation had been performed, showing that the hearing was not necessarily destroyed by that operation (as had been repeatedly stated by Mr. Heath to be the case).

Mr. HEATH, in reply, said none of the operations on his cases shown that day were Schwartze's. In answer to Mr. Stuart-Low, he said a conservative operation was done, the perforation healed, and the hearing was good. A few weeks later severe tinnitus came on, and because of that radical operation was performed. Naturally he thought that something was wrong with the tympanum. The radical operation was carried out against his desire, but evidently the result was perfect. The other ear had been lost, and if one ear on becoming diseased went to the bad, its fellow, if attacked, was liable to follow suit. That was his reason for operating more quickly than usual. In answer to Mr. Tod, the antrum was induced to fill up by treating it in such a way as to favour the formation of granulations, and making as small removal of bone at the operation as was consistent with removal of all disease, so that the small cavity readily filled in. He had never known the mastoid antrum opened during suppuration without finding disease. He would rectify in the notes the omission of the statement that disease was found.

CASE II.

Mr. F. Twenty-eight years' suppuration in the left ear. Gradually increasing deafness until it interfered with work, and he attended the Throat Hospital. More than the lower half of the membrana tympani had already been destroyed. The ossicles were apparently intact. The other ear having become deaf through non-suppurative catarrh, operation on the suppurating one was carried out in the hope of preserving some useful hearing. No cholesteatoma being found, the proceeding was concluded as a conservative operation. A photograph is shown of the condition of the drum at the time of operation. Within three weeks the patient returned to work with greatly improved hearing. The destroyed portion of the membrane was all replaced during the five months following operation. Now the ear is sound and hearing good.

Exhibited to demonstrate the great extent of repair which will occasionally occur, and to show that long duration does not necessarily render complete cure impossible.

DISCUSSION.

Dr. MILLIGAN said he was at a loss to know what the special operation was. In answer to Mr. Tod, Mr. Heath said he cut away a small amount of bone and that the antrum became obliterated. But this antrum had not healed up; it was an epithelialized cavity. He did not reconcile Mr. Heath's reply with what was to be seen in the patients. The antrum was not obliterated.

Dr. FITZGERALD POWELL wished to draw attention to the following sentence in Mr. Heath's report of the case: "The other ear having become deaf through non-suppurative catarrh, operation on the suppurating one was carried out in the hope of preserving some useful hearing," and said that he could not agree as to this particular operation preserving the hearing any more than the complete or radical operation did. The primary object of the mastoid operation was to make the patient's condition safe and free from the danger of further infection. The condition of the hearing did not depend on this operation or on that, but depended on the condition of the labyrinth, the inner wall of the tympanum, the stapes, and the oval and round windows, if these structures were not interfered with during operation. The hearing would be as good after the radical operation as after the one advocated by Mr. Heath—viz., a modified Schwartze's.

Dr. BRONNER asked whether Mr. Heath had tried ordinary treatment first. The weak point in all his cases was that he had not tried other means before adopting his own method. Everyone had seen cases of suppurating ears, in which the patient could not hear at all, yet by ordinary methods of treatment the hearing had become practically normal, although Mr. Heath's operation had not been done. If other methods had been tried first in Mr. Heath's cases, his remarks would have been more convincing. Surely even Mr. Heath's operation was attended by a certain amount of risk as regards life and hearing; all cases certainly did not recover their hearing. It was most unjustifiable, and contrary to the elementary principles of surgery, to submit a patient to a major operation without trying the simple ordinary methods of treatment first. Dr. Bronner strongly objected to Mr. Heath's statements that in all cases of otorrhœa, even of short duration, the antrum was diseased, and the cause of the persistence of the discharge.

Mr. HEATH, in replying, said there was disease of the antrum; he had never seen a mastoid operation for aural suppuration without disease in the antrum. In these cases he found a cavity not clean and shining, such as in health, but one which was reddened and moistened with purulent matter. In answer to Dr. Milligan, it was not a radical operation; those which were radical did not fill up; those which were not radical operations had, with one exception, filled up, and that exception he had referred to. In answer to Dr. Powell, in his experience hearing was not so good after the radical method, nor was the probability of saving hearing. He did the operation with the idea of preserving some useful hearing. The man had lost the other ear, and the remaining ear was so bad that he was threatened with the loss of his living, so that one was not justified in treating him longer with drops and lotions. The ear had been discharging twenty-eight years, and he had lost more than half his drum. In answer to the President, there was not a repetition of the original membrane; it was scar membrane; there was no aperture through it. This patient had been treated for some time at the hospital before operation, and was threatened with loss of his situation if his deafness continued.

CASE III.

Miss B. Sent for further advice by her doctor on account of headaches, with an inquiry as to whether the mastoid operation, by putting an end to the suppuration, would diminish the painful attacks. Both ears had been discharging continuously for thirty-eight years. The left, the worse ear, which showed a perforation involving one-third of the drum membrane, was submitted to operation. The tympanum appeared full of swollen mucous membrane, though there was no sign of cholesteatoma. The ear rapidly recovered the excellent condition in which it is now to be seen. The large perforation has healed. The hearing has returned. Two years later, in consequence of the patient's desire to get rid of the annoyance of continuous treatment of the other ear, that one was submitted to an operation of the same kind. This was after forty years of continuous discharge. There were two perforations in the membrane of this ear, one in front of, and the other behind, the malleus. The result is equally successful. There is perfect restoration of structure and function.

This case is shown to prove that duration does not necessarily affect the prospect of repair after operation, and that the possibility of cure depends on tympanic conditions.

DISCUSSION.

Mr. WHITEHEAD repeated his question, and would wish to do so in every one of the cases. In none had Mr. Heath stated the details, with two exceptions, and whether he found enough disease to justify the operation.

Mr. HUGH E. JONES asked what was the size of the antrum, and whether it was an "infantile" or cellular mastoid, because that made a great deal of difference in the ultimate size of the cavity left after operation.

Mr. TOD asked what was meant by the statement, "the possibility of cure depends on tympanic conditions." He asked, because in the next case it was stated that the antrum was the usual cause of the suppuration.

Dr. KELSON asked if Mr. Heath meant that after twenty-eight years of suppuration there was perfect restoration of structure and function, as stated in the notes of the case.

Mr. HEATH, in reply, said he did not recollect that the antrum was the only part of the middle ear which was diseased, for the tympanum always suffered. He eliminated what disease was apparent in the antrum only. [The PRESIDENT: I presume, then, you cannot answer Mr. Whitehead's question.] Mr. Heath, continuing, said the antral lining was diseased and removed, and that was sufficient to lead to the recovery of the case. In answer to Mr. Jones, the only large cavity, except those following the radical operations, was in Case VII, in which it still remained large, and this size was due to the disease which had existed before operation, and pointed to the necessity of interfering before such extensive destruction had occurred. In answer to Mr. Tod, the tympanic condition regulated the possibility of cure after the antrum had been dealt with. If the tympanum was not past repair, the patients would get well; if the tympanum was disorganized, the case would not get well, whatever was done to the antrum or tympanum.

CASE IV.

Mr. W. Suppuration in both ears for eighteen months. Patient being an engineer at sea, he was anxious to get rid of all disease before returning to work. There was a slightly foul discharge with eczema of the meatus on each side. Under a single anæsthetic conservative mastoid operation was performed on both ears. No sign of cholesteatoma was found. The perforations healed rapidly, and the antral cavities filled up. Now the patient has sound ears and good hearing. My colleague, Mr. Walker Wood, performed a Killian resection of the nasal septum.

Exhibited to prove that the antrum is the usual cause of persistent suppuration. Certainly until the tympanum is disorganized and destroyed as an organ of hearing.

DISCUSSION.

Mr. WHITEHEAD said it seemed scarcely of any use to ask questions, as members could not get the accurate facts with regard to the disease and the condition of the hearing, and in the absence of these facts any discussion on the cases was futile.

Mr. STUART-LOW said that he had had the advantage of seeing this patient, as Mr. Heath had sent him on for inspection. The discharge had ceased, and the hearing was good, but in his opinion, quite as good a result could have been attained if, the nose and throat being attended to, the mastoid antrum only had been opened and drained. The discharge had only been going on for eighteen months, and with such a short history, as a rule, the subcortical operation was sufficient to cure the discharge and restore the hearing.

The PRESIDENT asked what evidence of disease was found in the antrum.

Mr. HEATH, in reply, said, in reference to Mr. Stuart-Low's remark, that they were agreed that some suppurating ear cases got well after nasal operations. In answer to Dr. Permewan, he pleaded guilty to not having referred to the hospital reports in the cases; he thought the notes he gave would suffice for the foundation of a discussion. Accurate reports of the cases were kept, and he would be glad to know if it would satisfy members if he handed the hospital records in afterwards. In answer to Mr. Scott, the smallest opening was made in the antrum in every case consistent with inspection. The smaller the opening the more rapidly did it fill up. It was for this purpose that it was kept as small as was consistent with the removal of all the disease. With regard to the disease which was found in the antrum, all of them showed evidence which was not in harmony with a healthy ear. This patient was also treated for several months before operation.

Case V.

E. S. Scarlet fever seven years ago. Perforation and discharge ever since. Three operations for adenoids at the Throat Hospital. The disease became suddenly acute and the patient came to the hospital on November 9, 1906, while the writer was attending a meeting of the British Laryngological and Otological Association, and presenting similar cases. The President on that occasion (Dr. Woods, of Dublin) had said he desired to see this method of operation, and came to the hospital at 9 o'clock that evening, to witness its performance on this patient. During the operation he was enabled to see the perforation, though it could not be located through the intact meatus. He was also confidently informed that this perforation would surely be healed within a month. It was healed in nine days, and the patient left the hospital on the fifteenth day.

This case shows that if the removal of adenoids does not ensure recovery, it is unwise to delay indefinitely the performance of a conservative mastoid operation.

DISCUSSION.

Mr. Tod remarked on the statement that the disease "became suddenly acute." He asked whether there were mastoid symptoms. Was there suppuration for a day or two, or how long was there suppuration?

Mr. Heath, in reply, said the suppuration has lasted seven years, and the acute mastoid trouble supervened. There had been three operations for adenoids in the hope of bringing the ear disease to an end. Pus was present in the antrum.

Case VI.

Mr. O. Post-influenzal earache for six days before the membrane burst in Shrapnell's portion. The patient was first seen two days afterwards. There had been one attack some years before. The hearing of the other ear was defective. The patient could not blow air through the perforation. He was blistered behind the ear, ordered salicylate of soda internally, and dilute glycerine of carbolic acid drops in the meatus. Syringing was forbidden, and he was advised to come to the hospital the following week. On his coming again he was still unable to blow air through by Valsalva's method. The deafness had increased. There was slight giddiness, and occasional slight earache. The earache could not be tympanic, because the perforation was draining the tympanum, therefore it was considered that the antral secretions were retained. It was therefore decided to operate immediately, as the other ear was permanently damaged. Pus was found pent up in the antrum. An accumulation of cells, preliminary to cholesteatoma, was discovered in the attic and washed out when the cannula was used.

This case is shown to illustrate the possibility of very early commencement of cholesteatoma, and the conditions which predispose to it. Also to exemplify the delay in bursting of the membrane, when Shrapnell's portion is the only part exposed to fluid pressure.

DISCUSSION.

Dr. MILLIGAN said he desired to make a definite protest against the line of treatment adopted in this case. It was an ordinary post-influenzal otitis media, of six days' duration, yet immediately the post-aural operation was performed. Presumably there was tubo-tympanic catarrh with stenosis of the Eustachian tube, and the slight earache was due to intra-tympanic tension. Mr. Heath said "an accumulation of cells, preliminary to cholesteatoma, was discovered in the attic and washed out when the cannula was used." Was it in the experience of members that a case which had gone on for not more than fourteen days had a cholesteatoma, or even an indication of it? If the case had been treated on rational lines there would have been no necessity for any form of post-aural operation. He protested more especially because the hearing in the other ear was defective. The patient had been submitted to an unnecessary operation, and there was the off chance of abolishing hearing altogether by the operation.

Mr. STUART-LOW said that he was gratified to find that Mr. Heath had forbidden syringing in this case, as he had for years strongly deprecated all syringing in the treatment of ear discharge, as likely to drive the sepsis further afield. He attributed Mr. Heath's good result in this case to the inhibition of all syringing.

Mr. HEATH, in reply, said Dr. Milligan's protest was a natural one, but on the notes he did not think it was justified. At the time he saw the patient, a North of England surgeon also saw him, and he told that surgeon he would operate that day at the hospital for four reasons: (1) There was pain, which was not tympanic, as there was a perforation draining that cavity, therefore the pain could only be due to retention of discharge in the antrum; (2) hearing was getting worse, which implied tympanic swelling and progressive disease; (3) he was giddy, implying that somehow the labyrinth was becoming involved, probably through the pressure exerted by the tympanic swelling interfering with the position of the stapes through dragging of the long process of the incus; (4) the man had lost his other ear. The surgeon from the North saw the operation at the hospital, and said he first thought the decision to operate had been arrived at on insufficient grounds, but he was glad to see the diagnosis justified upon the operating table. There was pus in the antrum, and there was acute mastoiditis which was treated early. With regard to cholesteatoma, he believed the pathology of that was not definitely settled. He looked upon the accumulation of cells in the attic as preliminary to cholesteatoma, as it kept up irritation in that cavity. The swelling of the membrane there interfered with drainage of the cavity behind it. He washed out the accumulation, and he had washed out a similar collection by a similar operation in another case three months after suppuration began, but the ear had never dried up. When squamous cells and purulent matter were allowed to remain in the attic for three months, that ear was doomed.

Case VII.

R. T. Eight years of age, and eight years of suppuration. Polypus in the meatus, hiding the perforation. At the operation a large antro-mastoid cavity was found filled with granulation tissue, hence its present large size. On removal of polypus and examination of drumhead a large perforation was found, and some polypi removed through it. The ossicles appeared intact, and the cannula, when in use, exhibiting no sign of cholesteatoma, a conservative operation was performed. The perforation healed within three weeks. The operation was performed in 1906.

Shown to exhibit the large mastoid cavity, and the desirability of interfering before disease had caused it to assume such dimensions.

Case VIII.

J. W. T. Ten years' suppuration. The patient's ear had been tender and deaf all his life, and became painful and deafer in wet weather. He underwent a conservative operation in March of last year; repair was rapid; the perforation healed; the hearing returned; the long-standing tenderness also disappeared. A few weeks later he complained of neuralgic pains on that side of the head, which were difficult to explain. They would come on at varying times and were very severe. The ear looked perfectly healthy, and there was a natural objection to interfere with such an apparently satisfactory condition, but no other cause could be found for the pain. After much consideration and discussion it was decided to perform the radical operation. Even after this was done the pain continued for some time, not chiefly in the locality of the ear. In the course of a month or two the neuralgic attacks gradually passed off.

This is one of the small number of persons in whom the writer has found it necessary to convert a conservative operation into a radical one. Even now there is some doubt as to whether the case was one requiring it. There is a slight tendency to stenosis of the meatus.

Case of Chronic Suppurative Mastoiditis, with Abscesses in the Left Temporo-sphenoidal and Frontal Lobes.

By P. Watson-Williams, M.D.

S. S., female, aged 20, was admitted to the Bristol Royal Infirmary on January 16, complaining of discharge of pus from both ears, which she said had existed since she was a small child, but which had diminished during the past few weeks. For some months she had suffered from pain in the forehead, behind the left ear and occiput, and this had increased during the last week or two. Pain at times in the forehead severe. With some intermissions, she had continued her work as a cardboard box maker, and beyond the fact that she was poorly and suffered from pain, her family had not noticed any particular change, nor had her mental condition appeared to have altered. She had had attacks of giddiness, and vomited once before her admission.

On admission, the left membrana was found absent, the middle ear being full of granulations; the right membrana was perforated. Rinné test negative, right and left. She complained of screaming noises in the left ear. There was markedly double optic neuritis, with swollen œdematous retinæ, and hæmorrhage. Visual fields good for white and colour. There was no spontaneous nystagmus, and no nystagmus with rapid head movements. Caloric reaction, left ear, head erect: Water, 77° F.; nystagmus, 41 seconds; vomited one minute later. No fistula symptoms; no cranial nerve paralysis; reflexes normal; no dysdiadokokinesis. The cultures from the pus yielded a Gram-negative bacillus, slightly mobile, producing slight green pigment, identified as *Bacillus pyocyaneus*, in nearly pure culture; a few staphylococci; streptococci absent. (I. Walker Hall.)

January 18: Radical mastoid operation was performed, when it was found that the dura mater was exposed in the roof of the mastoid antrum, which was infantile in type, the dura mater being thickened. After the operative area had been disinfected, the dura was incised. The brain was not explored, and no pus was found. Lumbar puncture yielded clear sterile cerebrospinal fluid under considerably increased pressure. Patient went on well.

January 27: Vaccines prepared from the pure culture, *Bacillus pyocyaneus*, were commenced, the initial dose being 25 million. Subsequently she had the following doses: January 31, 50 million; February 5, 125 million; February 14, 250 million.

January 30: A considerable amount of pus escaped from the mastoid wound; she had complained of headache two days before, and was relieved.

February 3 and 4: Complained of severe headache. Wound was cleaned; no purulent discharge.

February 4: Headache continued; mastoid wound was opened up; sinus forceps introduced through the former opening in the roof of the antrum, entered the temporo-sphenoidal lobe; about ½ oz. pus escaped. Large drainage tube was inserted. Patient continued to do well up to February 14, when her mental condition, which had been bright, became dull. Temperature went up, from being normal or subnormal, to 102° F. There was no pus escaping. Pus had ceased to discharge from the temporo-sphenoidal lobe, although the drainage tube had been kept in.

February 17: A considerable amount of offensive purulent discharge escaped by the tube. She seemed brighter and considerably relieved.

February 23: Became mentally duller and indifferent to surroundings. Temperature again rising, from 99° F. to 102·8° F.

February 24: She died at 3.30 a.m.; shortly before which she was restless, sitting up in bed and screaming; Cheyne-Stokes respiration developed markedly two hours before she died.

Post mortem: Very slight meningitis. Abscess in the temporo-sphenoidal lobe was found well drained, but tracking forwards from the anterior end of the temporo-sphenoidal sinus was a narrow canal leading into an abscess in the left frontal lobe, which reached forward nearly to the apex of the left frontal lobe.

The interesting feature of this case appears to lie in the fact that a girl who was able to continue her work had a chronic abscess in her left temporo-sphenoidal and left frontal lobes, and there was no localizing symptom. Although the left temporo-sphenoidal lobe had been well drained after the operation, it would have been difficult, even if one could have diagnosed an abscess in the frontal lobe, to have effected drainage of an abscess so situated. It is noteworthy that the pus in this case yielded *Bacillus pyocyaneus* in nearly pure culture.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FOURTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1910-11

PATHOLOGICAL SECTION

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1911

Pathological Section.

OFFICERS FOR THE SESSION 1910-11.

President—

F. W. Mott, M.D., F.R.S.

Vice-Presidents—

Sir Almroth Wright, M.D., F.R.S
R. T. Hewlett, M.D.

Hon. Secretaries—

C. Bolton, M.D.
J. C. G. Ledingham, M.B.

Other Members of Council—

F. W. Andrewes, M.D.
W. S. Lazarus Barlow, M.D.
A. E. Boycott, M.D.
W. Bulloch, M.D.
E. Farquhar Buzzard, M.D.
L. S. Dudgeon.
J. W. H. Eyre, M.D.
A. S. F. Grünbaum, M.D.
Otto F. F. Grünbaum, M.D.
S. G. Shattock, F.R.C.S.

Representative on Library Committee—

W. Bulloch, M.D.

Representative on Editorial Committee—

S. G. Shattock, F.R.C.S.

PATHOLOGICAL SECTION.

CONTENTS.

October 18, 1910.

PAGE

The Comparative Neuropathology of Trypanosome and Spirochæte Infections, with a Résumé of our Knowledge of Human Trypanosomiasis. By F. W. Mott, M.D., F.R.S. 1

November 15, 1910.

The Bentley-Taylor Method of Mounting Mosquitoes, with Demonstration of Specimens. By Arthur Powell (Bombay) 41

The Biological Characteristics of *Bacillus typhosus*, with especial reference to the Fermentation of Dulcitol and Arabinose. By Burton Bradley ... 43

The Influence of Diet upon the Formation and Healing of Acute Ulcer of the Stomach. By Charles Bolton 53

Atypical Chloroma. By A. C. D. Firth and J. C. G. Ledingham 60

The Experimental Production of the Carrier State by Feeding. By G. F. Petrie and R. A. O'Brien 70

Experiments with Air-dried Mouse Cancer: (I) The Lethal Effect of Drying upon Mouse Cancer: and (II) The Inability of Air-dried Mouse Cancer, or of a Centrifugalized Extract, to produce Prophylactic or Curative Immunization. By S. G. Shattock and L. S. Dudgeon 73

January 17, 1911.

The Incidence of Gall-stones and of Primary Carcinoma of the Gall-bladder and Biliary Passages in the Insane. By J. P. Candler 87

Variability of the Gas-forming Power of Intestinal Bacteria. By W. J. Penfold 97

On the Microscopic Structure of Uric Acid Calculi. By S. G. Shattock ... 110

February 7, 1911.

PAGE
Seven Cases of Amaurotic Idiocy (Tay-Sachs Disease). By HILDRED B. CARLYLL and F. W. MOTT, F.R.S. 147
The Pathological Anatomy of a Case of Amaurotic Family Idiocy. By GORDON M. HOLMES 199

February 21, 1911.

Asphyxia under Chloroform. By A. GOODMAN LEVY... 205
Uterine Tumours in Rabbits. By A. E. BOYCOTT 225
Action of Pro- and Anti-thyroid Preparations. By WALTER EDMUNDS 233
Lamarckism and Callosities. By S. G. SHATTOCK 279

March 21, 1911.

Resection of the Cæcum and Ascending Colon, showing Chronic Hypertrophy of the Mucous Membrane, with Stenosis simulating Cancer. By C. GORDON WATSON 237
On the Relation of the *Meningococcus intracellularis* to Pseudoglioma. By GEORGE COATS and J. GRAHAM FORBES 242
Studies in Complement Fixation with Strains of Typhoid, Paratyphoid, and Allied Organisms. By H. R. DEAN 251

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

JOHN BALE, SONS & DANIELSSON, LTD., 83-91, Great Titchfield Street London W.

Pathological Section.

October 18, 1910.

Dr. F. W. Mott, F.R.S., President of the Section, in the Chair.

The Comparative Neuropathology of Trypanosome and Spirochæte Infections, with a Résumé of our Knowledge of Human Trypanosomiasis.

By F. W. Mott, M.D., F.R.S.

The study of the diseases produced by spirochæte and trypanosome infection is primarily biological. The contagium vivum is a living organism whose activities, like that of all living organisms, are for self-preservation, and especially the preservation of the species. The chemical toxins which these organisms produce are to enable them to live and multiply. It is generally believed that the spirochætes are organisms whose characters link them to the protozoa rather than to bacteria. The *Spirochæta pallida* contracts, moves, and modifies its structure in a manner different from a bacterium. The appearance of resting forms is totally different and they arise in a different manner to the spores of bacteria. Again, the clinical aspects of affection from a spirochæte invasion differ from that of bacterial diseases and conform especially to certain trypanosome infections. There is a periodicity of the symptoms altogether unknown in bacterial diseases, but what has struck me from my own personal experience and knowledge is the great similarity of the histological lesions of the nervous tissues of chronic trypanosome infection—for example, sleeping sickness and the *mal de coit* of horses—to syphilitic and parasyphilitic lesions. Again, there is similarity in the fact that lymphocytes and plasma cells are found in the cerebrospinal fluid in trypanosome diseases of animals and man—for example, sleeping sickness. Moreover, Levaditi has shown that in point of view of sensibility in respect to hæmolysing poisons, blood corpuscles, spirochætes and protozoa generally constitute a homogeneous group, and the spirochætes correspond in this respect more to the protozoa than the bacteria. It is probable that the periplasium of these protozoa contains a complex of lipoid substances similar to red-blood corpuscles and animal cells generally. This is an important fact, for it possibly affords an explanation of the action of organic arsenic compounds in the treatment of these diseases; for it may be that such drugs as atoxyl, soamin, "606," &c., have an elective affinity for the lecithin complex entering into the formation of the periplasium. By the periplasium I mean the osmotic membrane which covers the viscid protoplasm constituting the body of the organism. Moreover, we can understand why, when energetically pushed, they may injure the nervous system and produce neuritis by combining with the lecithin of the nervous tissue.

Trypanosomiasis corresponds with syphilis in being a disease characterized by inoculation, a period of incubation, affection of nearest lymphatic glands, followed by generalization in the lymph, then the blood-streams, and afterwards by successive eruptions, due to escape of trypanosomes from the blood-stream into the lymph spaces of the tissues, where they set up a similar tissue reaction. Moreover, in severe trypanosomiasis, as in severe syphilis, there is always, or nearly always, a polyadenitis.[1] In the central nervous system in sleeping sickness there is a chronic lymphangitis affecting the membranes and perivascular spaces, due, no doubt, to the escape of the trypanosomes from the blood-stream into the lymphatics in the same way as they escape into the vessels of the skin; they set up in the perivascular lymphatics a chronic inflammatory, endothelial and connective-tissue cell hyperplasia. Both in syphilis and sleeping sickness the location of the evidence of most severe irritation is in the region of the base of the brain; this fact may be due to a direct extension along the lymphatics of the large vessels and nerves entering the base of the skull, or to the greater amount of cerebrospinal fluid in this region and the relatively larger size of the fluid sheaths of the perforating vessels. There is one striking difference between the effects on the nervous system of infection of *Trypanosoma gambiense* and *Spirochæta pallida*, and that is that, whereas every case of this trypanosome infection leads eventually to invasion of the nervous system, in syphilis not more than 5 per cent. to 10 per cent. even of untreated cases result in invasion of the nervous system. I believe this fact has a biological explanation. Examination of the trypanosomes with the ultra-microscope shows them to be very active, motile organisms, giving one the impression that they could readily penetrate the delicate capillary walls of the nervous system, whereas the spirochætes show a sluggish, screw-like movement. Moreover, whereas in every case of sleeping sickness trypanosomes have been found in the cerebrospinal fluid, spirochætes have never (with one doubtful exception) been demonstrated in the cerebrospinal fluid. It is probable that they only exist in the true lymphatic sheath of Robin contained in the adventitia; here they set up chronic irritation causing a localized or diffuse gummatous condition.

---

[1] While the *Trypanosoma gambiense* is still conveyed to man by a specific fly, the *Glossina palpalis*, it is recognized that the *Trypanosoma equiperdum* owes its power of transmitting dourine, or the *mal de coit*, from horse to horse to the fact that it has acquired the habit of perpetuating its species by multiplying in the mucous fluid found at mucous orifices. Possibly the *Spirochæta pallida* has likewise acquired new habits, and, though now transmitted direct from man to man, was at one time dependent upon a biting insect, just as now is the spirochæte of tick fever.

With this brief introduction I propose to pass on to a résumé of our knowledge of trypanosome infection as it affects the human subject in the production of sleeping sickness, and I shall conclude by a comparison of the neuro-pathology of this disease with syphilis and parasyphilis of the nervous system.

Although sleeping sickness had been described by Winterbottom as early as 1803, it was not until the beginning of the twentieth century, when the economic future of the British Protectorate of Uganda was threatened by a devastating epidemic of the disease, that the Colonial Office, inspired by Manson, approached the Royal Society with the view of appointing a commission.

Manson and I had, in 1899, investigated several cases which had come to England from the Congo, and although I have had under personal observation only a few cases of the disease, I have been from this time much interested in studying the histological changes in the nervous system. As a result of the action of Manson, a committee of the Royal Society was appointed and Castellani was sent out to Entebbe to investigate the disease. Castellani discovered the trypanosomes in the cerebrospinal fluid; but, as he had already reported a micrococcus (probably the same as the Portuguese Commission had discovered) as the cause of the disease, he did not attach to the trypanosomes the importance which they deserved; nevertheless he found them in five cases, and it is quite possible, had he continued to work at Entebbe, he would have come to a definite conclusion that the trypanosomes, and not the micrococci, were the cause of the disease.

Bruce, the discoverer of the tsetse-fly disease, was sent out, accompanied by Nabarro and Greig. They placed upon a sure foundation the causal factors of sleeping sickness: (1) By confirming and largely extending the discovery of Castellani of the existence of trypanosomes in the cerebrospinal fluid and the blood of persons suffering from sleeping sickness; (2) by proving the existence of trypanosomes in a biting fly, the *Glossina palpalis;* (3) by correlating the geographical distribution of the disease with the geographical distribution of this biting fly; (4) by communicating the disease to animals, including monkeys, by inoculation with the cerebrospinal fluid and blood of patients suffering with the disease, or by allowing flies to bite patients having the trypanosomes in their blood, then allowing the same infected flies to feed on animals and thereby communicating the disease to the animals.

The investigations carried on by the Sleeping Sickness Commission of the Royal Society established beyond dispute that a specific organism (the *Trypanosoma gambiense*) and a specific fly (the *Glossina palpalis*), which acted as a carrier, were the essential causes of the epidemic of sleeping sickness in Uganda.

THE TRYPANOSOMA GAMBIENSE.

This was the name given to the organism by Dutton, and the discovery happened in this way. On May 10, 1901, Forde received under his care at the hospital, Bathurst, Gambia, a European, aged 42, the captain of a steamer on the river Gambia. The man suffered with symptoms which were regarded as malarial. Examination of the blood did not reveal malarial parasites but *worm-like bodies* concerning the nature of which Forde was undecided. A little later the patient was seen by Dutton in conjunction with Forde, and the former recognized that these *worm-like* bodies were trypanosomes. Dutton gave an excellent description of this organism, which he called *Trypanosoma gambiense.* The patient died on January 1, 1903. Dutton and Todd, in their first report of the trypanosomiasis expedition to Senegambia, 1902, described further cases of human trypanosomiasis. Of 1,000 cases examined in Gambia, six natives and one quadroon showed trypanosomes in their blood. It subsequently was shown that this trypanosome found in Gambia was identical with that found in Uganda. Dutton, Todd, and Christy, in their report upon trypanosomiasis upon the Congo, state that the organisms in the blood of individuals (whether showing signs of sleeping sickness or not) are identical, and there is no reason to suppose that the trypanosome observed on the Congo differs from the *Trypanosoma gambiense.* Moreover, the pathogenic action upon animals is the same. Thomas and Linton have made a comparative study of the human trypanosomes derived from different sources: (1) Trypanosomes brought from Gambia by Dutton and Todd;

(2) trypanosomes sent by Bruce and Nabarro from Uganda; (3) trypanosomes of Dutton Todd, and Christy from the Congo (from the cerebrospinal fluid of sleeping-sickness patients and from the blood of patients showing no signs of this disease). Thomas and Linton inoculated the trypanosomes from these various sources into a large number of animals, and they found that the pathogenicity was almost the same in all cases. (Nabarro). Laveran has confirmed these researches by experiments upon animals with three different strains of the human trypanosome.

Plimmer concluded from certain experiments which he made upon rats that *Trypanosoma gambiense* and *Trypanosoma ugandense* are quite distinct and separate, but Thomas and Breinl (of the Liverpool Tropical School) made experiments on a large number of rats using several strains of human trypanosomes, including the two strains used by Plimmer, and they obtained results similar to those of Thomas and Linton and Laveran. Consequently it may be affirmed that the *Trypanosoma gambiense*, originally discovered and described by Dutton, is the specific organism of sleeping sickness, whether it be acquired in the Congo State, Senegambia, Uganda, or Portuguese West Africa. In the early stages of infection the lethargy characteristic of the disease does not occur, and the case recorded by Manson, of a European lady missionary, at first exhibited only the symptoms of trypanosomiasis and was described as a typical case of infection by *Trypanosoma gambiense*; subsequently, and for a few months prior to death, she developed the characteristic lethargy. Low and I examined the tissues of this patient and found the characteristic lesions which I had previously described. This case and many others which have died since show that the name "negro lethargy" had to be entirely abandoned, for Europeans, and in fact any human being, may be infected and die of sleeping sickness: and there is no racial immunity against sleeping sickness. It may be mentioned that in the "Bulletin of the Sleeping Sickness Bureau" just published there is a synopsis of fifty European cases; of these, one lived three and a quarter years and one case six years.

But the name "sleeping sickness" also should be abandoned for that of "human trypanosomiasis." There are at present in England three cases being treated for infection by *Trypanosoma gambiense*:[1] two of these I have recently seen, and neither shows any lethargy although trypanosomes are present in the blood and the patients are subject to irregular paroxysms of fever. One case, owing to the high fever and sweating that followed, was thought (as in the original case of Forde) to be suffering with malaria, until examination of the blood revealed the true cause. It would be better therefore to follow the convenient classification of the French Sleeping Sickness Commission. They distinguish *cas en bon état*, persons who have no symptoms whatever of trypanosome infection; *cas suspect*, persons who have symptoms which lead to further investigation; and *cas cliniques*, cases which can be diagnosed from the symptoms alone. A more accurate division, which they make when it is possible, depends on the result of lumbar puncture (Bagshawe). If invasion of the subarachnoid space has taken place, it is regarded as evidence of the patient having passed into the second or third stage; if not, he is in the first. But I shall have occasion to refer to this matter more fully later.

---

[1] I am indebted to Dr. Daniels, of the Tropical School, for his courtesy in allowing me to see these cases.

The evidence therefore points to the important fact that the changes found in the central nervous system—which I shall describe fully later—are due to the infection of the subarachnoid space by the parasites. It would be interesting to know if other forms of trypanosomes enter the subarachnoid space or whether it is only the *Trypanosoma gambiense*, because I have never found any lesions like those of sleeping sickness in any other form of trypanosomiasis, although sections of the brain may show swarms of trypanosomes in the blood vessels. I am inclined to think that it is the invasion of the subarachnoid space by the *Trypanosoma gambiense* which renders it so incurable. Bruce doubts whether a case is ever cured (but *vide* p. 17). The various drugs—e.g., atoxyl, soamin, mercury, trypanroth, and antimony—certainly cause the trypanosomes to disappear from the blood, but whether they will attack the organism when once it has infected the cerebrospinal fluid is the important question to be ascertained. I should doubt it, for these drugs do not pass from the blood into the cerebrospinal fluid. Seeing that the organism can easily penetrate the walls of the delicate capillaries of the central nervous system, once the subarachnoid space is invaded, there is always a reservoir for reinfection of the blood and lymph streams. Although the cerebrospinal fluid is not (owing to the absence of proteid) a suitable medium for development, yet by the production of inflammation of the meninges the fluid acquires proteid substances, and the organism obtains thereby a nutrient medium; moreover, the reaction engendered by their presence is lymphocytic, and not polymorphonuclear phagocytic.

The question arises whether the reappearance of trypanosomes in the blood may not be due to latent endocellular forms. It will be remembered that Schaudinn affirmed that trypanosomes may pass through states of endoglobular development. Since then there has been a good deal of discussion upon the relations which exist between intracellular parasites and trypanosomes, notably concerning Leishman-Donovan bodies. Carini, in a recent paper from the laboratory of Mesnil in the Pasteur Institute, describes and figures trypanosomes undergoing endoglobular development in the blood of Leptodactylus. In the examination of the tissues of a large number of cases of sleeping sickness, dourine, and other trypanosome infections, I have found and described cells which presented appearances *suggesting* the possibility that there were endocellular forms; and Salvin Moore and Breinl described what seemed to be somewhat similar cell forms as occurring in the spleen, lungs, and bone marrow of rats inoculated with *Trypanosoma gambiense*: these forms they regarded as a resistant form of the trypanosome.

I will now pass on to the part played by the fly, the carrier of the disease.

THE GLOSSINA PALPALIS.

Since the publication of the Report of the Sleeping Sickness Commission in 1903, it was known that *Glossina palpalis* was the transmitter of the disease, but only recently, owing to the researches of Kleine, published in March, 1909, have we learnt that the fly after the ingestion of trypanosomes (*Trypanosoma brucei*) remains non-infective for eighteen to twenty days, but after that period it is able to infect susceptible animals. This important observation regarding the *Trypanosoma brucei* was confirmed by Bruce for the *Trypanosoma gambiense*, and the researches of Bruce and his colleagues make it highly probable that some flies may remain infective for the rest of their lives. Bruce and his colleagues made the following interesting experiment: They inoculated a monkey subcutaneously with a small droplet of fluid obtained from the gut of a fly that seventy-five days previously had been fed on an animal infected with *Trypanosoma gambiense.* The droplet of fluid prior to injection was found on examination to be swarming with trypanosomes. When the blood of the monkey was examined eight days after, trypanosomes were found, showing that it had been infected (*vide* fig. 1).

Kleine has also investigated the existence of *Trypanosoma gambiense* in the alimentary canal of flies reared from the pupa which were first fed on animals infected with that parasite, and subsequently on healthy animals. Kleine's figures exhibit a marked difference in form. The slender, red-coloured flagellates, poor in plasma, with dark nucleus he considered to be male forms. The plump, blue-stained, possessing one, two, three, or more nuclei, he considers to be female forms. He asserts, with reason, that a sexual increase may occur even in the resting stage. Kleine, moreover, found parasites in the salivary glands, but he regards their presence as accidental, and not as playing an important part in the transmission of the disease. He found no evidence of hereditary or germinal transmissions, as Leishman has shown to occur with the spirillum of tick fever. A most important step forward has been made by these researches of Kleine, confirmed by Bruce, for we now know that some kind of development of the trypanosomes takes place in the fly after ingestion; but whether a sexual process occurs, as Kleine shows, or whether there is merely such a multiplication as occurs in cultures is not at present decided with certainty.

It was previously believed that the fly only retained its infectivity for forty-eight hours, consequently it was thought that it would be possible to stamp out the disease in an island by one day clearing out its infective population, and a few days later re-stocking it with healthy natives. Bruce remarks that it is known by experiment that the fly can retain its infectivity up to eighty days; indeed, it is probable that after a fly has become infected it will harbour the trypanosomes for the rest of its life, but what the duration of this is under natural conditions is unknown. Further experimental investigations by Bruce and his colleagues are of interest, although they by no means afford a positive solution of the question. The lake shore was cleared of the native populations in December, 1907, and had been deserted for nearly one year when the experiments began. Flies in this district were caught and allowed to feed upon monkeys, with positive results; it was therefore concluded that the *Glossina palpalis* on the uninhabited shores of Victoria Nyanza can retain its infectivity for a period of at least two years after the native population had been removed. The practical importance of this continued infectivity of the flies is undeniably great, but what the cause is, and how it can be prevented, is another matter. The experiment does not, in my judgment, prove that the flies live two years, or that there is germinal transmission. From a conversation I have had with several Europeans now in England suffering with infection of *Trypanosoma gambiense*—notably Mr. Grimes, an elephant hunter—it is impossible to control the movements of the native, and numerous means of re-infection of the flies of the district are possible; in fact, Bruce himself points out the possibility of the flies having fed on natives who frequent the lake shore in spite of prohibition; or it might be explained by the fact that the natives who were employed in collecting the flies were the subjects of trypanosomiasis, but Bruce asserts that it cannot be by infection of the flies by natives, because precautions were taken in respect to fly-boys and canoe-men employed by the committee; or, lastly, it is possible that the mammals and birds along the lake shore have been infected, and so act as a reservoir of disease. It will be remembered that Koch thought the crocodile might be a host for the sleeping-sickness parasite.

Bruce sums up: "There remain then the two theories—long duration of life of the fly, and a local reservoir. The former at present cannot be answered, and there is no experimental proof of the latter, since the injection of the blood of the lake-shore birds and mammals into susceptible animals has always up to the present given negative results." It appears to me that the main difficulty in accepting Bruce's conclusions is the fact that the natives may, and probably do, frequent the lake shore in spite of the prohibition.

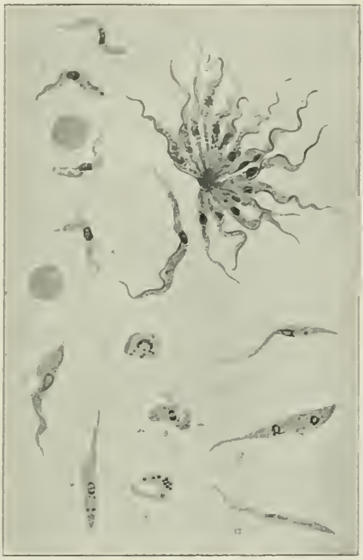


FIG. 1.

(1) Rosette form from the mid-gut (× 1,000). (2-5) *Trypanosoma gambiense* from the blood of the monkey into which a tiny drop of the contents of the mid-gut of fly had been injected (× 1,000). (6-13) Trypanosomes from the fore-gut of fly stained by Giemsa (× 1,000).—Report 10, Sleeping Sickness Commission.

It may certainly now be assumed that there is a period of time in which the fly does not transmit the disease after biting an infected person; and experiments appear to show that there is a period in which it more certainly transmits the disease, and this is correlative to the finding of large numbers of trypanosomes in the alimentary tract; and, as Todd suggests, this is an indirect support of the sexual stage, which Kleine's observations so clearly indicate. Kleine found that of 410 flies which had fed on animals infected with *Trypanosoma gambiense*, 22 became infected (5 per cent.). It seems therefore necessary that both males and females should be ingested in order that the flies may become infective. Bruce was formerly of the opinion that mechanical transmission played an important part, but recent experiments published in the *Proceedings of the Royal Society*, August, 1910, led him to the conclusion that mechanical transmission plays a much smaller part (if any) in the spread of sleeping sickness than has been supposed. The researches of Bruce and his colleagues show that "cattle may act as a reservoir of the virus of sleeping sickness, and that healthy animals may be infected from them by means of *Glossina palpalis*. It has also been proved that cattle in the fly area do naturally harbour *Trypanosoma gambiense*. It is therefore possible that the cattle and antelopes living in the fly area may act as a reservoir, and so keep up the infectivity of the *Glossina palpalis* for an indefinite period."[1] The fact that the geographical distribution of sleeping sickness corresponds with that of the geographical distribution of this particular fly *a priori* is against another source of transmission, although this does not necessarily follow, for the two modes may be coincident. *Glossina morsitans*, the fly that carries the *Trypanosoma brucei*, has not as yet been shown to act as a carrier to the *Trypanosoma gambiense*.

We may next inquire what other possible means there are of the transmission of the infective organism. Seeing that a scratch in making an examination of an infected rat probably led to Lieut. Tulloch being infected with *Trypanosoma gambiense*, and his death six months later, it is conceivable that sporadic cases of infection may arise from other causes than the bite of the specific fly, and the members of the Sleeping Sickness Commission in the French Congo bring forward evidence in favour of the transmission of the disease by biting insects other than glossina; they think that mosquitoes and other biting insects are important auxiliaries conveying infection in each hut from person to person during the night. They say that in regions where the natives nurse their sick in their own houses the disease spreads with much greater rapidity than in those where they drive them away. They give instances of one member of a family after another becoming infected, and quote instances in which a native was saved from infection, in their belief, by the use of a mosquito net. They found sleeping sickness extremely prevalent in marshy districts where mansonia abounded, and the Commission attributes to this method of infection, in some instances, the annihilation of whole villages.

---

[1] *Proc. Roy. Soc.*, B., 1910, lxxxii, p. 484.

Bagshawe, having thus stated the French Commission's observations, remarks that in nearly all cases there was a palpalis area near at hand or within a few miles. Moreover, why is not sleeping sickness endemic in the southern negro states of America and the West Indies, and why have there been no epidemics there? Certainly in the old slave days numbers of infected negroes must have been conveyed there. Again, the disease is endemic or epidemic only on the shores of the palpalis-haunted lakes and rivers. In Uganda, inland from the Victoria Nyanza and out of the reach of tsetse flies, no instance of infection from a sporadic case has come to light. I am informed by Dr. Grimes that sleeping sickness occurs nowhere in Rhodesia except on the borders of Lake Tanganyiki and the Loupopo River flowing from it. He also informed me that six cases of human trypanosomiasis existed at Broken Hill, and one European had died of sleeping sickness. No cases, however, had occurred in southern or north-western Rhodesia, and in those regions there is no *Glossina palpalis*.

Another possible means which had been put forward is coitus. We know that a trypanosome disease affecting equines (especially in mule-producing countries) is the *mal de coit* or dourine. In this disease a trypanosome has probably acquired the habit of direct transmission from one individual to another by multiplying in the mucous discharges of the sexual organs, and thus has arisen a new means of perpetuating the species. Koch and Kudicke put forward the hypothesis that sexual coitus explained the occurrence of the disease in women who lived in a palpalis-free area and who said they had never left it.

Kudicke's experiments support this hypothesis, likewise those of Manteuffel. The former introduced into the vagina of an uninfected monkey, taking care to avoid any injury, the blood of another monkey infected with *Trypanosoma gambiense*, and thereby infected it. The latter showed that blood containing trypanosomes placed upon a small patch of unshaven sound skin, allowed to dry and then covered with collodion, was followed in the greater number of animals by infection. These observations certainly suggest the possibility of infection by coitus. But as regards the explanation of the forty-four female patients treated by Kudicke, Hodges offers the following suggestions: "When the epidemic first swept through the lake-shore district the men, whose employment naturally carried them into the greatest danger, usually suffered first. As the men weakened, their occupations, such as canoeing, were in part taken up by the women, who then ran greater risks and more often contracted the disease, while many of their husbands by this time had either died from it or showed obvious symptoms of its presence."

If sporadic infection does occur by one or other of the "auxiliaries" described it must be unusual; for if we are sure of one thing in the ætiology and geographical distribution of this disease in Uganda, it is coincidence with the habitat of the *Glossina palpalis*. All the facts therefore point to the conclusion that something more than mere mechanical transmission of infection is necessary—viz., an intermediate host—and we owe to Kleine's investigations the proof of this, which the following experience in Uganda practically substantiates. In the progress report of the sleeping-sickness camps in Uganda, 1909, it is stated that "hundreds of sick have for long periods been collected in places in which *Glossina palpalis* is absent, and the disease has in no case spread either to the attendants or in the neighbourhood. This has materially assisted to impress on the natives—a task at first so difficult—the truth of that which they have been taught concerning the connexion between the fly and the spread of the disease."

I have endeavoured to give a résumé of our knowledge of the ætiology of this remarkable disease—a disease not only of great interest to the medical profession, but also to the general public.

CLINICAL STUDY OF HUMAN TRYPANOSOMIASIS.

My experience clinically is limited to several negroes, including the three in which I first described in 1899 the changes in the central nervous system which accounted for the principal clinical phenomena observed during life; also of several Europeans, including the first case, whose nervous system was examined post mortem. But besides these cases of negroes which have been brought to England and the Europeans who have returned from Africa and died in England, I have had forwarded to me the material from twenty-two cases that have died in Uganda, as well as material from monkeys and other animals which have been inoculated experimentally. An account of the pathological findings in this material formed the subject of the seventh report of the Sleeping Sickness Commission.

Description of the Disease.

The following account of the clinical symptoms is based upon my own experience, the notes of the cases of which I have examined the nervous system and which died in Uganda, together with knowledge acquired by reading the admirable reports of the Portuguese Commission and French Commission, the account given by Nabarro in his valuable translation of Laveran, and Mesnil's work on trypanosomiasis, as well as a recent important progress report on the Uganda sleeping-sickness camps by Hodges :—

First Stage : Local Infection followed by Generalized Blood and Lymph Infection.—A history obtained from Europeans shows that there is a period of incubation extending from the time of infection by the bite of the fly to the time that the parasite becomes generalized in the blood. At the point of inoculation a painful red papule, which may develop into a furuncle, appears; the nearest glands may become enlarged and painful. Twelve days may pass before the constitutional symptoms manifest themselves in the form of fever and sweating. This may be accompanied by swelling of glands, but not necessarily ; there is always fever, irregular, intermittent, sometimes high fever and sweating, which occur at the end of the rise of temperature. The patient usually thinks he has contracted malaria ; quinine, however, affords no relief, and examination of the blood, instead of showing malarial parasites, reveals the trypanosomes. This is often associated with nervous excitation, insomnia, headache as well as prostration, weakness, and emaciation. The respiration and pulse-rates are accelerated apart from the febrile attacks. Localized œdemas, especially of the face and ankles, puffiness of the eyelids, and evanescent, congested, or erythematous patches on various parts of the body, make their appearance. These symptoms may considerably improve and even end by completely disappearing at the commencement of the second period, which marks the invasion of the subarachnoid space and the presence of trypanosomes in the cerebrospinal fluid.

In negroes the above-mentioned symptoms are usually unobserved or even absent ; consequently the diagnosis could only formerly be made by the examination of the blood. The parasites, however, are often very scanty and difficult to discover in the blood. Having been struck with the frequency of glandular enlargement, I suggested to Greig and Gray that they should examine the fresh juice of enlarged glands obtained by excision or puncture. This method for diagnosis was performed by them, and found most useful when the organisms could not be seen in the blood. Moreover, it shows that possibly the lymphatic glands may harbour the parasites and lead to reinfection of the blood-stream and the infection of the subarachnoid space when the paravertebral glands are infected. Recent work confirms the observations of Dutton and Todd that the larger the glands the more likely are trypanosomes to be found in them. These observers, in a comprehensive investigation, have found that cervical gland enlargement without obvious cause in a native who has been exposed to the risk of infection is almost certainly due to trypanosomiasis, and should be regarded as such until the contrary is proved. Nabarro remarks: "The observations of Dutton and Todd in Gambia, and of Bruce, Nabarro and Greig in Uganda, show that in negroes this first stage is accompanied as a rule by no obvious signs of disease except the glandular enlargement above referred to." The French Commission, however, found that whereas under treatment some glands diminished in size, others did not; they concluded that the enlargement of these, though they often contained trypanosomes, was due to other causes. There are, moreover, cases in which the swelling of the glands is never very great. This was notably so in the case of a Persian who died of sleeping sickness, and whose tissues I examined. Heckenroth mentions the case of a boy who had slight suborbital œdema, and in whose blood trypanosomes were found. Not until a year later did any glands become puncturable. Heckenroth considers œdema as valuable an early sign as gland enlargement. The French Commission point out that the catamenia ceases in women and sexual desire is lost in men.

Second Stage: Sleeping Sickness, Lethargy.—The chief symptoms are fever and nervous manifestations. There is a pronounced expression of hebetude, which, once it has been seen, can immediately be recognized. The patient is indifferent to his surroundings, there is apathy and tendency to sleep or drowsiness, from which, however, he can be aroused to answer questions and for a brief period of time take an interest in what is said; but fatigue readily occurs, and, as a rule, answers are only obtained in monosyllables. He is paretic and unsteady, shuffling or oscillating in his gait. There is unsteadiness on standing, which increases on closing the eyes; there is nearly always tremor of the tongue and later of the hands when they are held out, and sometimes even when this measure is not resorted to; but there is no intention tremor. The knee-jerks are increased, and neither ankle clonus nor Babinski's sign can as a rule be obtained. The speech is slow, and sooner or later only monosyllables are uttered; but there is no slurring or elision of syllables, as in general paralysis, although there is some intellectual deficiency, as shown by weakness of memory, will power, and attention. Questions are comprehended as a rule, and the answers given are rational. There is no grandiose delirium, and hallucinations are but seldom noticed. There is no tendency to grandiose delusions, and the autocritical faculty is not wanting, for they keenly realize their hopeless condition. There is no optic neuritis or changes in the fundus, and the Argyll-Robertson pupil is invariably absent. The patient may remain in this condition, gradually getting more lethargic and feeble for three to six months, and then the terminal period commences in which there is profound lethargy, intense tremors and muscular weakness, loss of control over sphincters, and tendency to bed sores.

The resistance to microbial invasion is so lowered by the trypanosomiasis that the patients readily fall victims to pneumococcal and streptococcal infections resulting in bronchopneumonia and septicæmia; it is not surprising, therefore, that pneumococcal or streptococcal meningitis complicates the clinical picture in the terminal stages of the disease, and hastens its fatal termination. In the majority of the cases of which I have examined the tissues, I have found diplococcal or streptococcal infection either of the lymphatic glands or of the central nervous system, generally both.

In the excellent clinical study published in the French report, attention is drawn to the fact that " since our arrival in the Congo the cerebral form of the disease has attracted our attention, and we have observed numerous cases of insanity and hallucinations. This acute form of the disease is known to the natives." From their observations on twenty-four whites who were patients at the Pasteur Institute, the members of the Commission conclude that there is a cerebral and medullary form. The former they divide into diffuse and circumscribed. The diffuse is manifested by mental and meningeal symptoms of a subacute character, accompanied by loss of the intellectual faculties. The circumscribed form is characterized by localized cortical irritation and destruction, causing epileptiform convulsions and paralysis. The medullary forms are manifested by paraplegia, with some sensory troubles and bladder affection. The cerebral forms are incurable; the spinal progress slowly and respond to treatment. The French report also states that the mental complications of trypanosomiasis belong to the category of organic mental alienation, and are characterized before all by intellectual decadence. It may happen that the intellectual decadence is preceded by a period of slight exaltation. Mental confusion is more or less profound, and is characterized by stupor, confusion of ideas, amnesia, and disorientation, to which may be added visual and auditory hallucinations and non-systematized delusions. The evolution of these symptoms is always rapid, in some weeks the intellectual decadence becomes very profound, and the stupor appears. These symptoms remind one rather of Korsakow's psychosis, and it may be asked whether the treatment by arsenic may not have had something to do with them. Slight optic neuritis was mentioned. Possibly owing to the patients being kept alive longer by the treatment, a deeper affection of the nervous system may have taken place. Against this, however, I may state that I found the most profound change throughout the whole central nervous system in four of the cases that died in England. The most profound changes were found in an uncomplicated case that was under Dr. Stephen Mackenzie in the London Hospital in 1890; the patient lived six months after admission, and the only symptoms noted up to a few days of death were progressive lethargy, paresis, and tremors. Three days before death he could be aroused, and could answer when spoken to in monosyllables or simple phrases, such as "Good morning"; the same occurred in the two other cases which were under my care in the Charing Cross Hospital.

Again, on looking through the notes of the twenty-one cases of which post-mortem material was forwarded to me for examination, I can find no mention of paralysis or epileptiform convulsions; in fact, the main symptoms recorded pointing to the affection of the central nervous system were progressive paresis, tremors, and lethargy. Many of them, although in an advanced stage of the disease when admitted to the hospital, were able to tell where they came from, and give an account of their previous life. Most of these cases showed an advanced meningo-encephalitis.

Hodges, whose experience is based upon 5,081 cases received at the four sleeping-sickness camps at Uganda, thus comments on the effects of modern treatment:—

"Speaking generally of the effects of treatment, it must be said that atoxyl and its allies—though possessing a marked, if transitory, trypanocidal action—have not proved to be suitable for routine administration to all classes of cases, and that no considerable number of cures can be expected to result from their administration by the methods in use. *But it cannot fail to be noticed, by those who have been familiar with the natural course of sleeping sickness before the use of modern* *remedies, that this course is, if not cut short, at any rate considerably modified by the administration of the organic compounds of arsenic.*"

Paralysis, paresis, and epileptiform convulsions, which among untreated cases occurred in a small percentage, are now commonly met with and are often the precursors of sudden death, which itself was very exceptional before the use of organic arsenic. Sudden or rapid death, in fact, generally preceded by cerebral symptoms, would appear now to be almost the rule among such cases as have received full courses of organic arsenic, while the prolonged lethargic stage which almost invariably marked the end of untreated cases is either scarcely noticeable or absent. It would seem probable therefore that, owing to the prolongation of the course of the disease by treatment, the nervous lesions are afforded time to become more pronounced and eventually to kill the patient, and that this may happen even though all trypanosomes may have been eliminated from the system.

If this be so, and these nervous lesions are in no way due to the treatment itself, it of course follows that, when once the disease has reached a certain stage, the lesions then existing are likely to be progressive, apart from the toxins produced by trypanosomes, and that treatment by trypanocidal drugs during or after that stage will probably be useless. When this stage actually occurs is not known, and it is doubtful whether there would be any clinical symptoms by which it could be recognized, though it has long been agreed that it is necessary to begin treatment at as early a stage as possible.

I have recently examined the tissues of the central nervous system of a case of human trypanosomiasis who was treated so energetically with atoxyl that, owing to neuritis and mental confusion and dullness, the administration of the drug could not be continued. It had been given intermittently for eighteen months, and apparently it effected a cure, for the man lived for three and a half years after treatment had been suspended, and trypanosomes, which had, early in the disease, been found in the glands and blood, could no longer be found. Death from pneumonia occurred, the man for several years having been in excellent health. I could find no evidence of the characteristic perivascular infiltration of lymphocytes and plasma cells in the brain. It is probable that the trypanosomes had never entered the subarachnoid space. A full account of the case will be published shortly in the *Proceedings of the Royal Society*. I have seen a well-marked perivascular infiltration of the subcortical tissues in a case that died nine months after infection.

Before describing fully the changes in the central nervous system in sleeping sickness, it will be advisable to consider the changes in the lymphatic glands. We shall then be in a position to compare the same with those found in the central nervous system and elsewhere.

The occurrence of irregular remittent pyrexia, in cases of infection by *Trypanosoma gambiense*, with erythematous urticarial eruptions, suggests association with paroxysmal elaboration of a poison or the multiplication of the parasite. The great frequency with which the lymphatic glands become enlarged may be associated with the paroxysms of fever and the presence of the protozoa in the glands. It has been shown that this enlargement is not due to microbial infection; it must therefore be due either to trypanosomes or a toxin engendered by them irritating the gland and causing proliferative hyperplasia of the cell elements. It is a matter of speculation whether the degenerative changes occurring in the neoplastic formation produce cytotoxins or not; probably by analogy they do not.

Histological Changes in the Lymphatic Glands.

A lymphatic gland in the first stage of swelling shows the following changes: Active proliferation of the lymphocytes in the germ-centres so that they are very densely packed together. In the lymph-cords and sinuses a very active cell-proliferation can also be observed. The oval staining nuclei of the endothelial cells lining the lymph-channels can be seen greatly increased in numbers and proliferating. Numbers of large mononuclear cells can be seen; these are round, with a deeply-stained round nucleus. They differ from the small mononuclears by the more abundant cytoplasm. Others are plasma cells of Marscholko containing a nucleus with a wheel-like arrangement of chromatin, and all stages can be traced up to the formation of a typical plasma cell and its final granular degeneration. The origin of these different types of plasma cells has always been a matter of dispute. Moreover, it is a question whether the nuclei seen in the body of branching retiform cells belong to endothelial plates, or are nuclei of branched connective-tissue cells. There is no doubt that these nuclei, when subjected to irritation, are excited to hyper-nutritive activity and proliferate, and produce mononuclear cells. There is increased vascularity of the gland, and not infrequently hæmorrhages; in fact it presents the appearance of chronic inflammation, and we must suppose that the cell proliferation is a defensive reaction to a noxious agent. The cell proliferation goes on

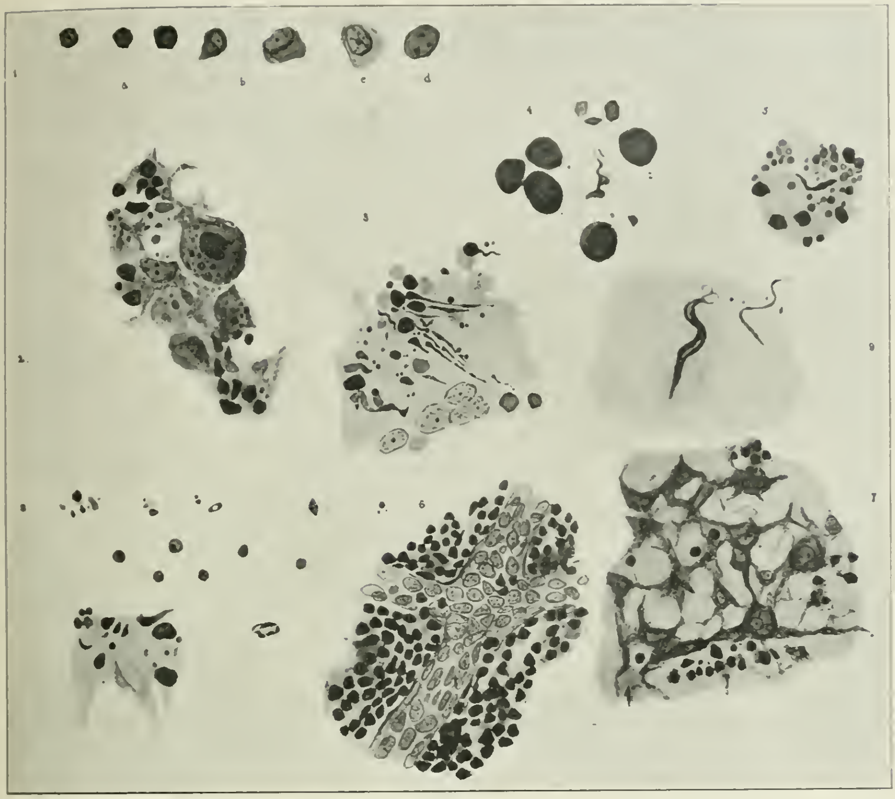


PLATE I.

CHANGES IN THE LYMPHATIC GLANDS.

FIG. 1.—Lymphocytes in all stages of transition to plasma cells.

FIG. 2.—Lymphocytes, plasma cells, and endothelial cells in all stages of granulo-aqueous degeneration. (× 375.)

FIG. 3.—Thread-like bodies and granules, deeply stained, seen in section of lymphatic gland. (× 750.)

FIG. 4.—Trypanosome in smear of fresh lymphatic-gland juice. Several lymphocytes and micronuclei. (× 750.)

FIG. 5.—Trypanosome in a section of lymphatic gland amidst disintegrated cell products.

FIG. 6.—Section of lymphatic gland from a fatal case of sleeping sickness. The glands in this case were not much enlarged. There is marked proliferation of the endothelial nuclei of the lymph channel. (× 375.)

FIG. 7.—Proliferation of the connective-tissue cells of the reticulum of a lymph sinus. Marked proliferation of the nuclei of the endothelial cells seen. This change closely accords with the change observed in the perivascular lymph spaces of the central nervous system. (× 375.)

FIG. 8.—Various granules and products of cell and (trypanosome?) degeneration seen in the perivascular cell infiltration of the central nervous system. (× 375.)

The preparations were stained with Leishman or Giemsa stain, and the glands used for preparation of specimens illustrated by figs. 1 to 5, inclusive, were removed during life in the first stage of the disease, before the invasion of the central nervous system had occurred.

until, automatically, an increase in numbers deprives the cells of sufficient nutrition, or they are destroyed by the virus and granulo-aqueous degenerative changes occur. These necrobiotic changes may be observed in the glands which are sterile by cultural tests for micro-organisms. In this stage there is only very occasionally evidence to be found in sections of the existence of trypanosomes. I have rarely in a very large number of sections seen any evidence of trypanosomes or their degenerated remains. Occasionally I have found the dead parasite in the form of attenuated thread-like forms, or macronuclei or micro-nuclei. According to Greig they can always be discovered in the fresh juice of the enlarged glands, but Thomas and Anton Breinl consider that they are not more numerous in the glands than in the blood.

In the third stage of very chronic cases, a few of which I have examined (one removed during life and sterile), the products of degeneration had been in great part absorbed, and the gland had become dense and fibrous. This is the final sclerous change that occurs in other chronic neoplastic formations, the fibrous conjunctival elements preponderating over the cellular elements. As a rule, in sleeping-sickness cases, death occurs before this can take place.

Morbid Changes in Lymphatic Structures.

All the observers from the earliest times have noticed the enlargement of the lymphatic glands; and Greig, at my suggestion, punctured the glands and examined the fresh juice. He is of opinion from his observations that this is an easier and more reliable mode of determining the existence of *Trypanosoma gambiense* than examination of the blood or cerebrospinal fluid. Dutton and Todd came to the same conclusion working in the Congo State. Many natives in Uganda and the Congo State have, however, enlarged glands, and yet are not the subjects of sleeping sickness. They may be, however, and probably in nearly all cases are, candidates for the disease.

Do the trypanosomes get into the glands and multiply there, setting up a chronic inflammatory process which terminates in fibrosis? The glands may be inflamed and enlarged and yet be sterile as regards micro-organisms. It is probable that trypanosomes infect the lymphatic glands by escaping from the ruptured capillaries, or they may have become infected by the cerebrospinal fluid when this secretion contains trypanosomes. Similarly by capillary hæmorrhage the trypanosomes may infect the cerebrospinal fluid and the lymphatic structures of the central nervous system. If the trypanosomes can set up chronic inflammatory changes in the lymphatic glands (as there is no doubt they do), and microscopic examination of sections reveals but occasional and scanty evidence of their presence, it is quite reasonable to suppose that they can similarly produce chronic inflammatory changes in the lymphatic structures of the central nervous system. We do not know if the trypanosomes produce this chronic irritation by their mere mechanical presence, which seems unlikely, seeing that vessels may be crammed with trypanosomes in nagana and surra without causing lymphangitis. There is, according to Plimmer, Thomas, and Anton Breinl, however, no experimental evidence that trypanosomes produce a chemical toxin; although that would seem the most probable cause of the chronic inflammatory change. The numbers of trypanosomes found in the cerebrospinal fluid are in no way proportional to the changes found in the central nervous system. Yet there is considerable evidence (*vide* Sleeping Sickness Reports, Royal Society) to show that not until trypanosomes are found in the cerebrospinal fluid does the chronic inflammatory change take place. If they existed in abundance instead of sparsely, we might consider that this fluid afforded a suitable medium for their propagation, and the absence, normally, of lymphocytes in this fluid might be counted a cause. On the other hand, the small quantity of proteids which the cerebrospinal fluid contains would not admit of suitable nutrition.

The posterior spinal ganglia always show some chronic changes, proliferation of the endothelium of the lymphatic capsules of the ganglion cells, together with interstitial lymphocyte accumulation, and these chronic changes may be due to the absorption of toxins from the neighbouring infected paravertebral glands.

In practically all cases of sleeping sickness the cervical glands are enlarged, and the most chronic change is found about the base of the brain. Hence a possibility that the chronic inflammation of the lymphatics spreads along the nerves, spinal ganglia and roots to the central nervous system, and especially along the lymphatics of the nerves and vessels entering the base of the skull. Examination of other tissues—e.g., the heart, pericardium, liver, alimentary canal, and testicles—shows, though generally speaking in far less degree, an infiltration and accumulation of lymphocytes in the lymphatics, suggesting a chronic inflammatory reaction of the lymphatics.

The Histological Changes in the Central Nervous System.

Observations of Bruce and his colleagues show that it is the invasion of the subarachnoid space by the organisms which cause this change, and my observations and experience show that there is a parallelism between the depth of the lethargy and the diffuseness and intensity of the lymphatic perivascular infiltration.

In the seventh report of the Sleeping Sickness Commission of the Royal Society I stated, as the result of an examination of a large number of sections stained by polychrome and eosin, Mallory and Heidenhain-eosin methods, that the meningeal-cell infiltration was the result of an irritative process affecting the pia-arachnoid serous membrane, which was manifested not only by a proliferation of the neuroglia cells but also by a proliferation of the endothelial-cell nuclei and an infiltration of the pia-arachnoid membrane with lymphocytes, which may become transformed into plasma cells. But sections do not show the mode of origin of these cells in such a clear and demonstrative manner as the following methods which I adopted. The pia-arachnoid membrane was stripped off small portions of brain from a number of cases of chronic sleeping sickness, including the European case under Dr. Bradford, which was of unusual value, because there was no terminal or secondary microbial infection, and because there was *no noticeable enlargement of the lymphatic glands.* Small portions of the stripped-off membrane were divided by tearing rather than cutting, so that the thin frayed edges could be examined under a high power of the microscope. They were stained by hæmotoxylin and eosin, Leishman's stain, polychrome and eosin, and Eisath's modified Mallory stain, and mounted in Canada balsam. Several interesting facts were observed. *The fibres forming the interlacing network were coarser than natural and much increased.* Many of the vessels were gorged with blood and there were *many capillary hæmorrhages.* A variable number in different cases of large cells containing blood-corpuscles or altered blood-corpuscles were seen, similar to those seen in sections. These cells were usually oval, sometimes round, with the oval or round nucleus pushed up to one end. Sometimes the cytoplasm contained discrete corpuscles, sometimes this *endothelial macrophage* had digested the corpuscles and the cytoplasm had assumed in consequence a uniform orange stain. Some of these cells containing blood-pigment had undergone nuclear proliferation; four or five deeply-stained, round nuclei could be seen in one cell. The adventitial sheath of the arteries can be distinctly seen, and there is often evidence of endothelial-cell proliferation shown by an increase in the number of large, pale oval nuclei, many of which could be seen undergoing mitosis and proliferation; they resembled the endothelial nuclei seen in the lymph sinus of the lymphatic glands, but the great mass of cell infiltration is in the meshwork of the pial trabeculæ of the subarachnoid space and its prolongation as a sleeve around the vessels entering the grey matter.

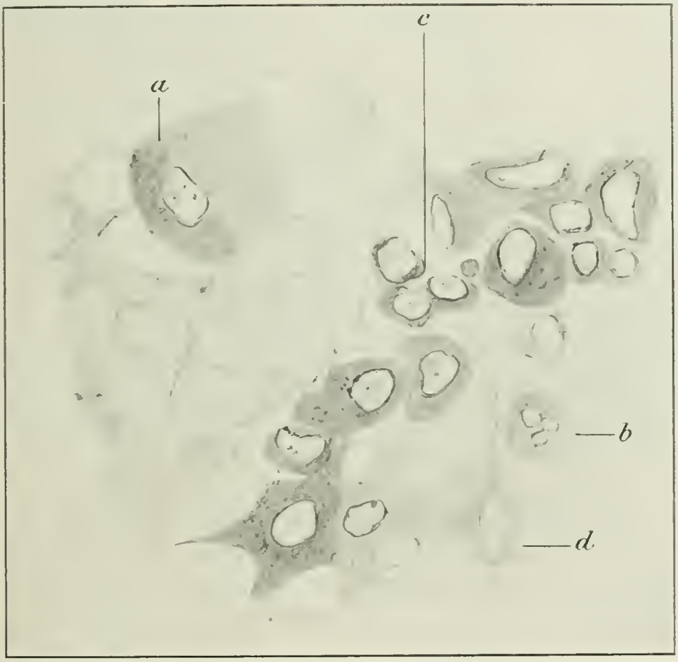


FIG. 2.
Small portion of pial-arachnoid tissue stained by Eisath's modified Mallory method mounted on the flat. (× 500.)

Preparations stained by logwood and eosin and Van Gieson's fluid exhibit two kinds of nuclei—viz., (1) large, pale, oval, less often roundish nuclei, with a delicate nuclear membrane and very fine intranuclear network, similar in all respects to the oval nuclei of a lymph sinus; (2) smaller round or irregularly shaped more deeply-stained nuclei with a narrow investing cytoplasm, also large cells containing similar deeply-stained nuclei, and not infrequently some cells, two or more, even as many as six, round nuclei, which are sometimes unequal in size and always uniformly, diffusely, and deep stained throughout. These cells are endothelial cells undergoing endogenous nuclear proliferation.

The endothelial cells of the lymphatic sheath of Robin and the endothelial plates lying upon the trabeculæ of the subarachnoid space and the pial sleeve of the vessels, as the result of the chronic irritation produced by the presence of the trypanosomes in the cerebrospinal fluid, undergo a progressive formative hyperplasia similar to that of the lymphatic glands.

In preparations stained by Eisath's modified Mallory stain, I have observed large flat endothelial cells without any processes exhibiting the following appearances of hyperplasia: (*a*) With the cytoplasm stained pink, and with an oval or round nucleus in the centre stained light yellow; (*b*) the same form of cell can be seen undergoing endogenous nuclear proliferation; (*c*) the same form of cell dividing or divided into small mononuclears in which there is only a relatively small surrounding pink-stained cytoplasm. Besides, we find cells which morphologically resemble the branched retiform cells of connective tissue of the lymphatic gland, with a large oval unstained nucleus. These nuclei appear to undergo division to form hyaline mononuclear cells which are seen proliferating in the inflamed lymphatic glands. The increase of the large and small mononuclear lymphocytes in the blood may be due to this cell hyperplasia in lymphatic structures. The meningeal and perivascular infiltration is due not only to active endothelial-cell proliferation *in situ*, but also to accumulation of the lymphocytes by conjunctival proliferation and consequent obstruction to the outflow of the lymph along the vessels, also obstruction to the escape of the cerebrospinal fluid from the craniospinal cavity. The infiltration is found especially around the vessels having a lymphatic and pial sheath; this sheath disappears on the smallest vessels, therefore we can easily understand why it is that the smallest vessels and capillaries show little or no investing sheath of cell infiltration. However, in chronic cases, lymphocytes, and especially plasma cells, can be seen closely applied to them. Do the branching processes of the *neuroglia cells* form a meshwork around the larger vessels and cause obstruction, or is the meshwork in which the mononuclear cells lie merely the thickened and proliferated trabeculæ of the connective-tissue cells of the lymphatic and pial sheath? My answer is that the infiltration around the large vessels and in the membranes entirely corresponds in appearance with the infiltration which I have described in patches around the vessels of the visceral layer of the

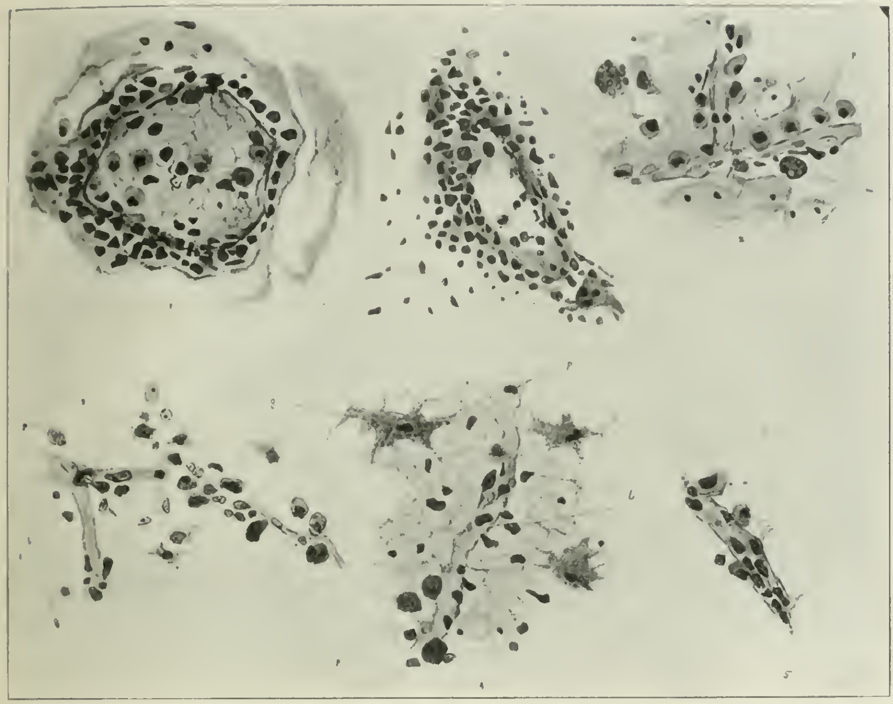


PLATE II.

CHANGES IN THE PERIVASCULAR LYMPHATICS OF THE CENTRAL NERVOUS SYSTEM, &C.

FIG. 1.—Transverse section of a small vessel of the medulla oblongata, showing perivascular infiltration with lymphocytes. Many of these have a hyaline appearance. The lumen of the vessel contains blood corpuscles, large and small mononuclear leucocytes, and a trypanosome is seen in the centre. This was the appearance presented by the vessels throughout the cortical and subcortical structures. The preparation was obtained from a very chronic case. (× 375.)

FIG. 2.—Small vessel with plasma cells (*p*) and morular cells (*m*). (× 375.)

FIG. 3.—Small vessel dividing into two capillaries, showing nuclear proliferation of the endothelial cells. In the neighbourhood are plasma cells (*p*), lymphocytes (*l*), and glia cells (*g*). (× 375.)

FIG. 4.—Three large neuroglia cells (*g*), their branches ending in a network around and upon a small vessel. In the meshes are lymphocytes (*l*) and plasma cells (*p*). (× 375.)

FIG. 5.—Small vessel with proliferation of endothelial nuclei and two plasma cells (*p*).

FIG. 6.—A transverse section of a vessel in a very chronic case of sleeping sickness in a European, showing marked perivascular infiltration with lymphocytes. (× 187·5.)

pericardium in the lymphatic spaces of the heart muscle and the perivascular lymphatics of the liver and the testis. Moreover, *I am unable to trace the processes of the neuroglia cells any farther than the outer sheath of the infiltration.* Again, no place shows the perivascular and meningeal infiltration better than the lymphatic and pial sheaths of the vessels in the soft membranes covering the cerebellum and their extensions between the folia, *yet there are no neuroglia cells seen in the adjacent cortex of the cerebellum,* although the neuroglia cells are seen in abundance in the white matter. The neuroglia proliferation is therefore not essential for the production of this characteristic perivascular cell infiltration. As a rule, the subcortical perivascular infiltrations are more marked and extensively diffused than the cortical, and this is especially evident about the base of the brain and around the perforating arteries; this corresponds in a way with syphilitic brain disease. Sometimes when there is but little evidence of cortical perivascularitis, there may be found very marked infiltration around the vessels of the base of the brain and the perforating arteries and their ramifications.

It is often difficult to distinguish lymphocytes from the proliferating nuclei of glia cells. We may distinguish three kinds of lymphocytes in transverse sections of blood-vessels and the surrounding tissues: (1) Hyaline forms, in which the nucleus is pale, staining poorly, irregular in outline or lobulated, and with a small amount of cytoplasm. (2) Small mononuclear cells in which the nucleus is irregular in outline or round, staining either deeply throughout, or the chromatin is arranged in the form of a wheel, with a central nucleolus, from which straight spokes pass out to a nuclear membrane ending in little knobs; there is hardly any surrounding cytoplasm. (3) Large mononuclears possessing phagocytic functions, the main difference from the smaller variety being the much larger amount of surrounding cytoplasm; they form the so-called plasma cells, and are developed from the proliferating endothelial plates, the same as the smaller lymphocytes; the latter can develop into them (*vide* Plate I, fig. 1). Whether an endothelial plate will form small or large mononuclear cells apparently depends largely upon the number of nuclei the original nucleus divides into.

In sections of vessels cut obliquely so that the outermost structure of the wall is shown—that is, the part in contact with sleeve of cerebrospinal fluid—I have seen endothelial cells lying like scales on the bark of a fir tree, or a tesselated pavement, and presenting all the appearances of the typical plasma cells of Marscholko. It appears to me a mere

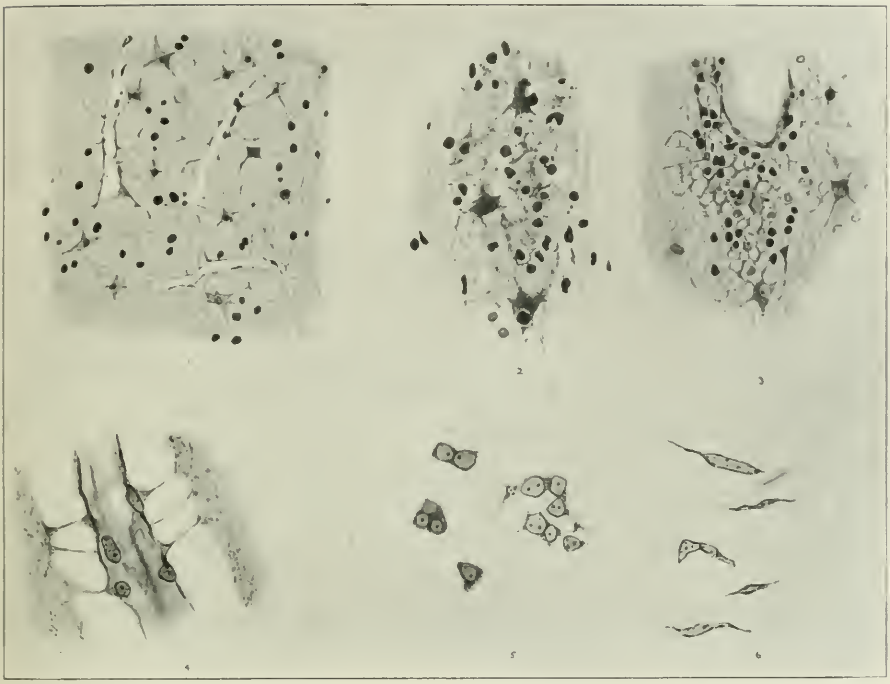


PLATE III.

NEUROGLIA, HYPERPLASIA, AND PROLIFERATION, &c.

FIG. 1.—Section of subcortical white matter of the brain of a monkey that died after infection by trypanosomes, caused by the bites of infected flies. There is little or no perivascular infiltration, but a considerable increase in size and number of the perivascular glia cells. The animal lived only eight months. (× 320.)

FIG. 2.—Section of subcortical white matter of a monkey that died eighteen months after infection, and showed the characteristic perivascular infiltration, with lymphocytes and plasma cells. The neuroglia proliferation is well shown, and in the meshwork of the branching fibres, which form a reticulum around a small vessel in longitudinal section, are numerous lymphocytes. (× 450.)

FIG. 3.—Transverse section of a blood vessel, with the pia-arachnoid and lymph-sheath much increased by proliferating cell elements. The lymphocytes are pale and unstained, and fill the connective-tissue meshwork. The neuroglia cells are seen externally, sending their processes inwards to join the connective-tissue reticulum of the pial sheath. (× 375.)

FIG. 4.—Small vessels of brain of a monkey in which experimental anæmia had been produced by ligature of all four arteries. This is to show the perivascular space filled with cerebrospinal fluid. The supporting trabeculæ are well seen passing across from the nervous matter to the wall of the vessel. It is easily to be understood that if this sheath is filled with cells entrapped in a thickened and proliferated network, the circulation of the lymph and cerebrospinal fluid will be interfered with. (× 750.)

FIG. 5.—Young neuroglia cells undergoing proliferation. (× 375.)

FIG. 6.—Red cells (Stäbchenzellen of Alzheimer), rarely met with, although occasionally appearances like this are seen. (× 450.)

quibble whether plasma cells are developed from lymphocytes or endothelial cells, since, in my opinion, there is sufficient evidence to show that both plasma cells and lymphocytes in chronic inflammatory conditions develop from endothelial cells of serous membranes and perivascular lymph structures, and from endothelial plates of the connective tissues.

The Neuroglia.

It is often a matter of some difficulty to distinguish young neuroglia cells from hyaline lymphocytes. By use of the modified Heidenhain and the polychrome and eosin stains, I was able to see all the changes which Watson described in juvenile general paralysis, and just as in that disease neuroglia-cell overgrowth is a leading histological characteristic, so it is of sleeping sickness and chronic trypanosome infections. The young neuroglia cells may be recognized by their pale-staining round or oval nuclei with a delicate intranuclear network containing one or two small nucleoli and a definite nuclear membrane. The chromatin substance is stained blue; surrounding the nucleus is a well-defined zone of protoplasm stained pink, of irregular quadrate or polygonal outline. These cells can be seen in groups and undergoing active division, especially in the neighbourhood of the ganglion cells. The various phases in the development of the neuroglia cells can be seen, viz.: (1) The nucleus, surrounded with an indefinite amount of cytoplasm, polygonal or irregularly quadrilateral in shape; (2) the protoplasm tending to form short spike-like processes, sometimes giving it a star-like appearance; (3) increase of cytoplasm around the nucleus and commencing formation and differentiation of the *darkly-stained* Weigert stiff fibrils; (4) further development of the Weigert fibrils and extension of one on to the wall of a vessel, there ending in a foot-like expansion; (5) further increase of development of the Weigert fibrils and differentiation from the pink-stained protoplasm on which they appear to lie; (6) the protoplasm is almost entirely differentiated into fibrils, and the nucleus is shrunken and stains deeply like the fibrils, so that the whole glia cell is stained a deep blue-black.

Distribution of the Glia Proliferation.

The distribution varies in different cases; it is almost entirely a primary interstitial overgrowth and not secondary to neural degeneration. It exists in a marked degree in cases which during life presented no very marked symptoms pointing to destruction of nervous elements. In some very chronic cases in which there have been many epileptiform seizures, there may have occurred sufficient degeneration in the pyramidal tracts to give rise to a secondary sclerosis, but this is exceptional.

The glia proliferation, which is not visible to the naked eye in sections of the spinal cord, becomes very manifest when examined with a low power, and there is a diffuse glia proliferation, as I first pointed out in the two cases which I first investigated. This diffuse subpial glial proliferation affects the periphery of the cord and spreads inwards along the septa; it is not only met with in the white matter, but is evident also in the grey matter. The situations in the brain where glia proliferation is most obvious in general paralysis are the situations in which it is most obvious in sleeping sickness. Thus it is well advanced in the most superficial layers of the cortex, where large branching cells with deeply-stained Weigert fibrils can be seen forming a subpial feltwork. The large branching cells with Weigert processes extending on to the small vessel walls cannot be seen so well amidst the columns of cells as in the subjacent white matter. From the examination of the brains of two monkeys that died of sleeping sickness after experimental inoculation, the glia-cell overgrowth and extension of processes on to the vessels appeared to be more marked than the perivascular mononuclear infiltration, as if this tissue was the first to respond to the irritation of the noxious agent (*vide* Plate III, fig. 1). However, examination of a case—Mrs. S.—in whom symptoms only existed for two months, did not show a glia proliferation in excess of the mononuclear infiltration; nor could I find any neuroglia proliferation or perivascular infiltration in a chronic case of infection by *Trypanosoma gambiense*, a native of Uganda, who died of pneumonia and pneumococcic meningitis after an illness of ten days, but who, prior to this illness, had displayed no symptoms of nervous affection.

The meningeal and perivascular infiltration with lymphocytes and *plasma* cells was regarded by Nissl as pathognomonic of general paralysis; but I pointed out that plasma cells as well as lymphocytes occurred in the perivascular infiltrations of sleeping sickness, and I figured the same on p. 289, vol. ii, *Archives of Neurology*, 1903. I mention this because an Italian observer claims to have first described plasma cells in sleeping sickness.

Changes in the Central Canal of the Spinal Cord.

Not only is there evidence of a chronic irritative action of the cerebrospinal fluid by the cell proliferation in the meningeal and perivascular lymphatics, but in all chronic cases the central canal of the spinal cord is filled up owing to a proliferation of the cells of the ependyma. I found that this had occurred in quite juvenile subjects. It was so in the little Congo negro boy who died in Charing Cross Hospital in 1898, and I was of opinion then that this fact afforded evidence of a very chronic nervous affection caused by some irritating agent. Such change denotes, then, a chronic process of considerable duration. Examined under a high power, the nuclei of the cells lining the spinal canal may often be seen undergoing active proliferation, and specimens stained with polychrome and Heidendain-eosin method exhibit large pale nuclei with a thin membrane and chromatin granules stained blue, surrounded by a pink cytoplasm, often with numerous processes. In some very chronic cases the glia proliferation had led to the formation of abundant Weigert fibrils. In the grey matter around the central canal numerous glia cells having a similar appearance can be seen.

I deem it of little importance whether the glia proliferation precedes mononuclear cell infiltration, or whether by its doing so it obstructs the flow of the lymph and entangles the mononuclear cells; nor do I regard it of much importance whether we speak of this formative cell hyperplasia as a chronic inflammatory process or not. The important fact to recognize is that this meningeal and perivascular infiltration is a hyperplastic reaction of fixed tissue elements to a noxious agent—*Trypanosoma gambiense.* So far we are on certain ground. It is, however, a matter of speculation whether this tissue reaction is due to (*a*) the relatively few trypanosomes which can be demonstrated in the fluid; (*b*) the elaboration of a toxin by them; (*c*) a transition to some hitherto undiscovered modified forms.

Changes in the Small Vessels and Capillaries.

The capillaries in the pia and in the brain tissue show the following changes, but these are not nearly so marked as in general paralysis of the insane.

The nuclei of the endothelial cells may undergo proliferation, and in the neighbourhood of the capillaries and small vessels there are often numerous lymphocytes, plasma cells, and glia cells sending a process on to the wall of a vessel; but I fail to find evidence of sprouting new capillaries as seen in general paralysis, nor can I but very rarely find any evidence of the Stäbchenzellen or rod-cells described by Alzheimer in general paralysis (*vide* Plate III, fig. 6).

The marked proliferation of the vascular endothelium with hyaline degenerative changes of the small vessels so frequently met with in general paralysis is hardly ever seen in even the most chronic case of sleeping sickness, nor can I find any evidence of endarteritis so generally met with in all cases of syphilitic brain disease. There may be a granulo-aqueous degeneration of the lymphocytes and plasma cells in the perivascular spaces, but I have never seen caseation nor have I seen tumour formation. This looks as if the trypanosome when it was surrounded by cells in the perivascular space did not undergo division and multiply because it requires a fluid medium, but was walled in by cells and killed unless it had escaped into the free cerebrospinal fluid. Whereas the growth starting in the meninges and spreading inwards along the pial sheaths as well as superficially suggests that the spirochæte multiplies at the expense of the cells, resulting from the chronic irritation of the endothelial and connective-tissue cells spreading thereby and setting up fresh cell hyperplasia with the formation of lymphocytes and plasma cells; but inasmuch as the walls of the arteries participate in this cell hyperplasia, endarteritis occurs, and this, in conjunction with the rapid neoplastic formation, leads to necrobiosis of the older central portions of the tumour.

Vascular (usually capillary) hæmorrhages are met with in all forms of trypanosome disease, and probably are the result of obstruction by the organism. Hæmorrhages may occur in syphilis, but these are due to arterial degeneration, as a rule, with thrombosis or rupture of the vessel.

Changes in the Neural Elements.

Although the meninges are in many cases obviously thickened and the convolutions flattened (indications of some intracranial pressure), yet there is no naked-eye wasting of the brain. The depth of the grey matter of the cerebral cortex is not appreciably diminished, although the vessels both in the grey and white matter may appear somewhat congested.

I have not observed granulation of the ependyma of the ventricles, so characteristic of the meningo-encephalitis of general paralysis of the insane. Moreover, the marked wasting of the grey matter of the cerebral cortex, so characteristic of the disease, is not met with in sleeping sickness. The convolutions are broad and of normal size, and the sulci tend to be obliterated in sleeping sickness, whereas in general paralysis the convolutions are shrunken from atrophy of the neural elements, cells and fibres, and the sulci are consequently broad and deep. In both diseases there is thickening of the leptomeninges and septal and perivascular changes, but here, it seems to me, the similarity ends. But this statement becomes more apparent and convincing when the microscopic changes are described. Moreover, a comparison of the size of the remaining structures of the central nervous system shows that in general paralysis there is a primary neuronic atrophy which does not occur in sleeping sickness. Thus to the naked eye the spinal cord in the latter disease may appear normal as regards amount of grey and white matter, whereas in general paralysis the cord is often much reduced in size and there is very obvious neuronic atrophy.

The naked-eye appearances therefore point especially to a primary parenchymatous degeneration in general paralysis with chronic interstitial and meningeal inflammation, whereas in sleeping sickness the morbid change is primarily interstitial and with some secondary parenchymatous atrophy (*vide* figs. 3, 4, 5).

Microscopic Examination of the Nerve Cells and Fibres.

In uncomplicated cases that have died within six months of the onset of the lethargy untreated by organic arsenical preparations, I have found a widespread diffuse infiltration of the meningeal and perivascular lymphatics, especially of the subcortical structures, with comparatively little distortion of Meynert's column or atrophy of cells. In a European who was treated and lived one year, having epileptiform seizures for weeks prior to death, there was a considerable atrophy and destruction of cell elements; but the perivascular and glia change, as compared with general paralysis, was out of all proportion to the neuronic atrophy.

Cells.—The changes in the ganglion cells may be considered as due (1) to the primary lymphangitis, and (2) to secondary microbial toxæmia. It is difficult to differentiate the cells which are affected by the one cause from the other. I consider, however, that the chronic change is indicated in those cells in which (1) there are appearances of atrophy of the dendrons, the protoplasmic processes being either attenuated or broken off; (2) there is a perinuclear chromatolysis, the cytoplasm still exhibiting some remnants of a pattern of Nissl granules in the circumference of the cell and on the dendrons; (3) the nucleus is large and clear, and often eccentric. Sometimes a dead ganglion cell may be seen being devoured by phagocytes. The cells of the spinal cord usually show much less change than the cells of the medulla oblongata and the cerebral cortex. The cells of the posterior spinal ganglion usually show chromatolysis, but not destruction (*vide* Plate IV). The appearance of the cells in acutely fatal trypanosome affections—e.g., surra and jinga in animals—could be accounted for by the anæmia caused by the blood change and the obstruction of the small vessels by the trypanosomes. In the brain of a rabbit dying of surra one month after inoculation, the ganglion cells all showed a shrinking of the cytoplasm, a marked chromatolysis and disappearance of the Nissl granules and swelling of the nucleus, and a change not unlike that observed in some forms of experimental anæmia.

Fibres.—In cases uncomplicated by terminal microbial infection, there is a certain amount of fibre atrophy proportional to the cell atrophy described. This atrophy is most obvious in the tangential layer of the cortex cerebri, where the fibres in places are greatly diminished, or even absent. There may also be some diminution of the fibres in the super-radial and inter-radial systems, especially in chronic cases. There is, however, in the brain as in the spinal cord, no definite system tract sclerosis, the result of atrophy of a neuronic system. Generally in the lateral columns corresponding to the pyramidal systems some degenerated fibres can be seen by the Marchi method, but the glia proliferation tends to follow the distribution of the septa rather than to accord with any definite atrophy of a system of nerve fibres. By the Marchi method, the cerebrum, cerebellum, spinal cord, and spinal ganglia were examined in a number of cases. In most instances the results were unsatisfactory, owing to a generally diffuse blackening of the myelin sheaths and the deposition of black granules. I consider that this change was probably the result of acute changes in the myelin, brought about by terminal microbial toxæmia, fever, &c. Some few of the cases, however, did not show this generalized change in the myelin, and a certain number of fibres showing Wallerian degeneration were found. These changes we may regard as definite, and indicative of neuronic decay.

EXAMINATION OF NERVOUS TISSUES OF ANIMALS EXPERIMENTALLY INFECTED BY TRYPANOSOMES.

Experimental Evidence.—Animals inoculated with *Trypanosoma gambiense* usually die before the characteristic lesions of the nervous system can occur. I have examined the tissues of nine animals (monkeys) which were inoculated at Entebbe in one way or another with *Trypanosoma gambiense.* They were all said to have exhibited the characteristic lethargy, but it is very difficult to differentiate (according to my experience) between a monkey that sits moping when profoundly ill and an animal which exhibits a lethargy on account of the brain lesion.

The tissues of the brains of all the animals sent to me, with the exception of two, showed no characteristic change. The vessels of the brain were empty and there was no meningeal or perivascular infiltration. Several of these animals had survived the infection (as proved by the existence of trypanosomes in the blood) one year. One was subsequently infected with diplo-streptococci from a case of sleeping sickness; yet there was no sign of the meningo-encephalitis met with in every case of human sleeping sickness. This was the experience, apparently, of Ayres Kopke.

The tissues of two monkeys inoculated with *Trypanosoma gambiense* showed, however, the characteristic lesion of human sleeping sickness. I have examined portions of the tissues, and find that there is a very marked neuroglia proliferation of the perivascular lymphatics, endothelial cell proliferation and lymphocyte accumulation, and a few plasma cells around the vessels of the brain in all the situations examined. In fact, the lesion in no respect differs essentially from that of human sleeping sickness.

A correlation of the clinical notes of thirty cases of sleeping sickness before the treatment by organic arsenical preparations was introduced, with a microscopic investigation of the changes in the central nervous system, shows that there is a parallelism between the intensity of the chief nervous symptoms—viz., drowsy lethargy, mental enfeebleness and fatigue, paresis and tremors—and the generalized chronic diffuse meningo-encephalitis. The general intense perivascular infiltration with lymphocytes and plasma cells must interfere with the circulation of the ambient fluid of the neurons, whereby they suffer from an insufficiency of oxygen. The fluid which circulates in the perivascular lymphatics is the ambient fluid that takes oxygen from the blood to hand it over to the nerve cells. This progressive, universal, and intensely inflammatory state of the perivascular lymphatics would interfere with its flow and lead to deficient oxygen supply. There is an interference with the outflow of the cerebrospinal fluid, but not sufficient to produce a choked disk, so that, although this may tend to produce cerebral anæmia, it cannot be so important a cause of the functional defect of the neurones, and yet it may take part in the production of the symptoms. The principal cause of

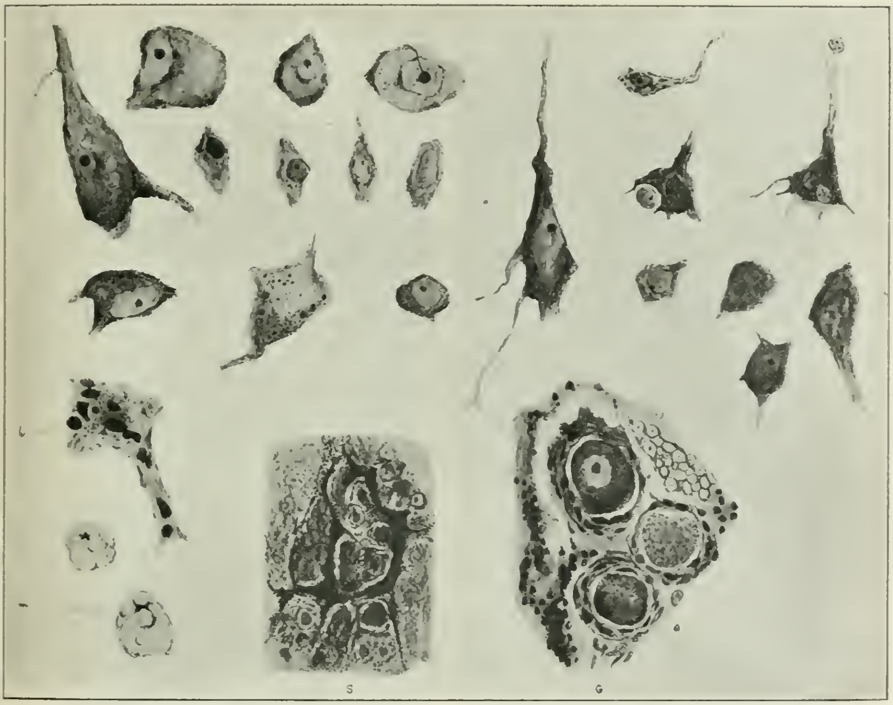


PLATE IV.

Changes in the Cells of the Central Nervous System.

Appearance of various large and small pyramidal cells of the cerebral cortex in advanced cases of sleeping sickness, showing various degrees of chromatolysis, eccentric position of the nucleus, breaking off and disappearance of the processes.

The cell (*h*) is obviously dead and being devoured by phagocytes. Below this are two granule cells (Körnchenzellen). (× 375.)

Fig. *S* is a section of a posterior spinal ganglion, showing an intense interstitial lymphatic-cell infiltration with lymphocytes. (× 90.)

Fig. *G* shows a portion of the section of the same more highly magnified. Not only can the interstitial lymphatic-cell infiltration be observed, but there is a proliferation of the endothelial cells of the capsule. (× 375.)

the lethargy, in my judgment, is the perivascularitis. Moreover, a vicious circle is established, for the more these lymphatics become obstructed by the actively growing young cells, the more the oxygen that may be in the fluid will be snapped up by them and the less will be at the disposal of the neurones. Consequently, the oxygen supply necessary for functional activity of the nerve cells becomes progressively less and the drowsy stupor deepens proportionally. The experiments of Verworn prove the importance of oxygen storage by the nerve cells and

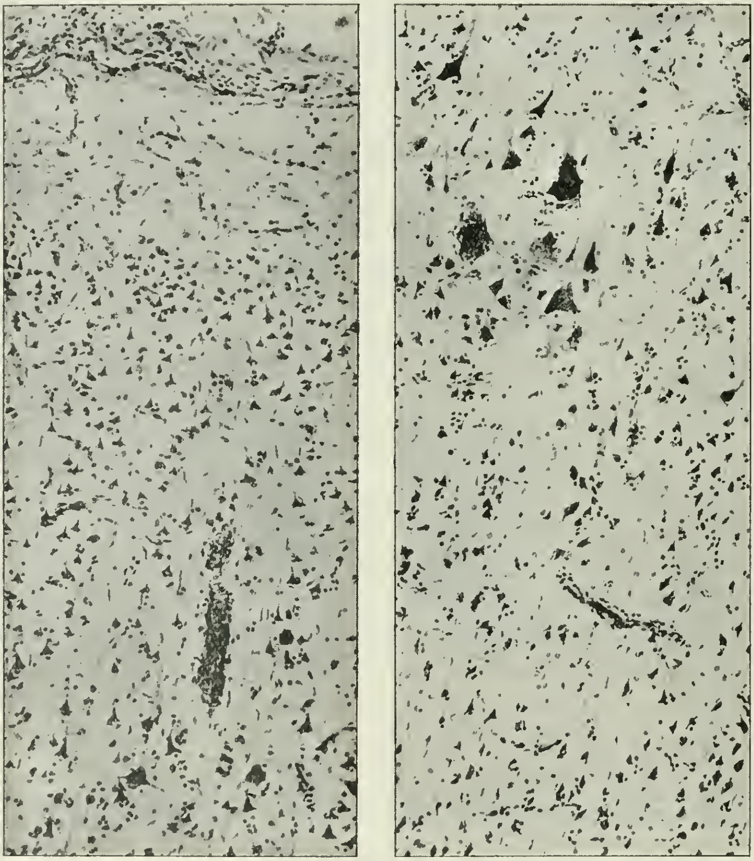
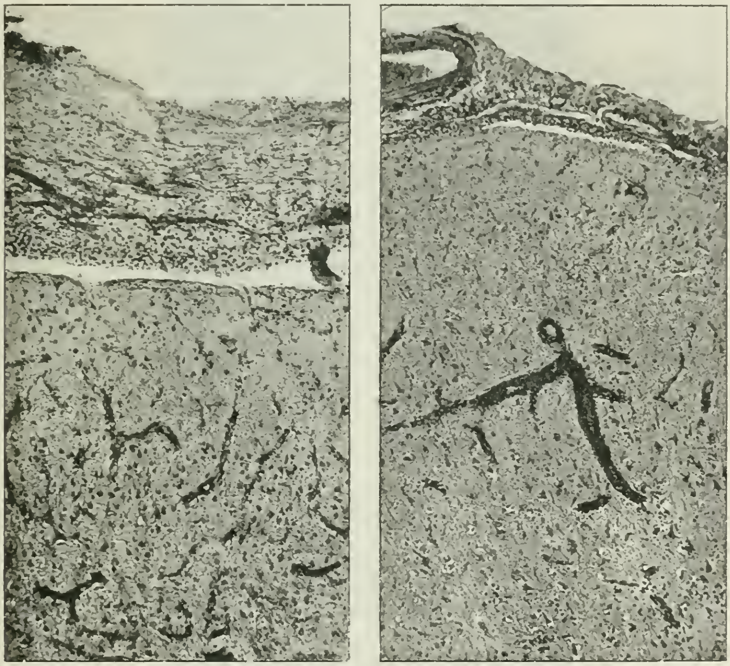


FIG. 3.

Photomicrograph of the cortex from a case of uncomplicated very chronic sleeping sickness. The pyramidal cells are not destroyed; there is not any increased vascularity; there is some perivascular and meningeal infiltration, not nearly so marked as in the subcortical tissue. The columns of Meynert are not disorganized; this accords with the fact that the main symptoms were paresis, tremors, drowsy lethargy, but no epileptiform seizures or mental disturbance beyond enfeeblement. (× 150.)

the necessity of its supply for functional activity. In widespread generalized syphilitic meningitis and perivascularitis a drowsy stupor is a frequent symptom ; in general paralysis the perivascularitis may be very intense in the cortex, but never so diffuse and intense in all the subcortical structures as in sleeping sickness. The progressive dementia which is the characteristic of the former disease is proportional to the atrophy and wasting of the cortical substance—a condition which is not usually met with in sleeping sickness. In juvenile general paralysis the primary parenchymatous change is more manifest because convulsive seizures are less frequently met with. I have observed two forms of change in the cells in general paralysis—viz., an atrophic change and an acute swelling accompanied by chromatolysis similar to that observed in experimental anæmia. This change is doubtless due to vascular stasis and accounts for the fact that after prolonged unilateral convulsive


a FIG. 4. *b*

Two sections of the cortex in general paralysis ; thickening and infiltration of membranes, perivascular infiltration, formation of new vessels and marked atrophy of cortical substance, especially in *b*, the frontal region ; *a* is the post-central region. (× *a*, 120 ; *b*, 75.)

seizures one hemisphere will be found to weigh very much less than the other, and microscopic examination will exhibit acute destruction of nervous elements in the hemisphere opposite to the seizures. If we regard the parasyphilitic affections, tabes and general paralysis, as being due to premature decay of neurones, it is not to be expected that drugs which benefit by killing the organisms will be of any service. Seeing that the spirochæte has never been demonstrated either in the tissues or cerebrospinal fluid in these affections, whereas it has in gummatous meningitis, we can understand why mercury, antimony, and arsenic

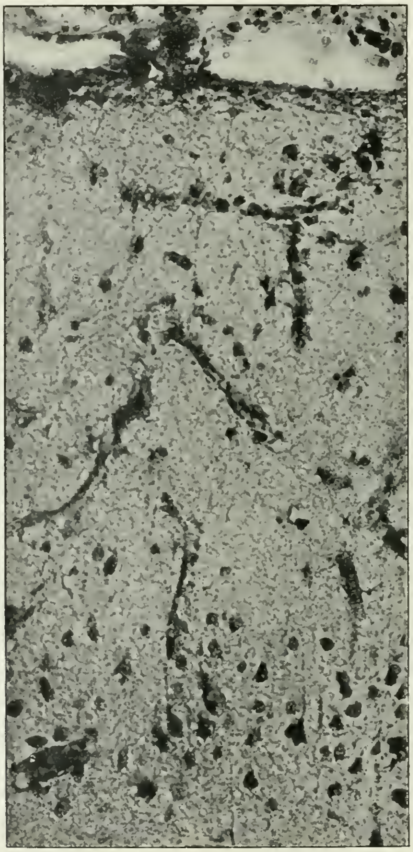


Fig. 5.

Section of the brain of a case of advanced general paralysis, showing the absence of cells in the superficial layer of the cortex. Numbers of vessels are seen to which many glia cells are attached. There is a complete absence of fibres and destruction of the superficial cortical cells (stained by van Gieson method). (× 262.)

compounds will, by killing the organism, cure syphilis of the nervous system and will not cure general paralysis or tabes. But why do these drugs have no influence in sleeping sickness? In syphilis the organism has not escaped into the free fluid, for it has never been demonstrated there. To rid the nervous system of the syphilitic organism it is therefore probably not necessary for the drug to get into the cerebrospinal fluid.

BIBLIOGRAPHY.

Alzheimer. "Histologische Studien zur Differenzialdiagnose der Progressiven Paralyse."

Bagshawe. "Recent Advances in our knowledge of Sleeping Sickness" (Trans. Soc. Trop. Med. and Hygiene), *Journ. Trop. Med. and Hyg.*, 1909, xii, p. 341.

Idem. Bulletin of Sleeping Sickness Bureau.

Bettencourt, Kopke, Gomez de Rezende, Correia Mendes. "La Maladie du Sommeil," Lisbon, 1903.

Bosc. "Les Maladies bryocytiques (Maladies à Protozoaires)," *Centralbl. f. Bakt.*, Jena, Abt. 1, 1906, Orig. xlii, p. 705.

Bradford and Plimmer. "*Trypanosoma brucei*, the Organism found in Nagana, or Tsetse-fly Disease," *Quart. Journ. Micro. Sci.*, 1902, xlv, pp. 449-471.

Bruce. "Discussion on Human Trypanosomiasis," *Brit. Med. Journ.*, 1910, ii, p. 864; *Sleeping Sickness Bureau: Bulletin*, 1908, i, No. 6, p. 209.

Bruce and Commissioners. "Reports of the Sleeping Sickness Commission":—

No. I, 1903 (1) Castellani. "Presence of Trypanosoma in Sleeping Sickness," p. 3.
(2) Bruce and Nabarro. "Progress Report on Sleeping Sickness in Uganda," pp. 11-88.
" II, 1903 (3) Christy. "The Distribution of Sleeping Sickness, *Filaria perstans*, &c., in East Equatorial Africa," p. 3.
(4) Castellani. "Adult Forms and Developmental Forms of the Trypanosome found in Sleeping Sickness," p. 11.
(5) Low and Castellani. "Report on Sleeping Sickness from its Clinical Aspects," pp. 14-63.
" III, 1903 (6) Christy. "The Epidemiology and Etiology of Sleeping Sickness in Equatorial East Africa, with clinical observations," pp. 3-32.
(7) Theobald. "Report on a Collection of Mosquitoes and other Flies from Equatorial East Africa," p. 33.
" IV 1903 (8) Bruce, Nabarro and Greig. "Further Report on Sleeping Sickness in Uganda," pp. 3-87.
" V, 1905 (9) Austen. "A Provisional List of Diptera," p. 3.
(10) Nabarro and Greig. "Further Observations on the Trypanosomiases (human and animal) in Uganda," pp. 8-47.
" VI, 1905 (11) Greig and Gray. "Continuation Report on Sleeping Sickness in Uganda," pp. 5-273.
(12) Greig. "Report on Sleeping Sickness in the Nile Valley," p. 273.
(13) Austen. "The Distribution of Tsetse Flies," p. 278.
(14) Gray and Tulloch. "The Multiplication of the *Trypanosoma gambiense* in the Alimentary Canal of the *Glossina palpalis*," p. 282.

Bruce, Hamerton, Bateman, and Mackie. "Duration of the Infectivity of the *Glossina palpalis* after the removal of the Lake-shore population," *Proc. Roy. Soc.*, B, 1909-10, lxxxii, p. 56.

"Trypanosome Diseases of Domestic Animals in Uganda," *Journ. Trop. Med. and Hyg.*, 1910, xiii, p. 265.

Calkins. "Pathogenic Protozoa," *Pop. Sci. Monthly*, New York, 1906-7, lxix, p. 409.

Castellani. "Researches on the Etiology of Sleeping Sickness," *Journ. Trop. Med. and Hyg.*, 1903, vi, p. 167.

Dutton. *Journ. Trop. Med. and Hyg.*, 1902, v, p. 363.

DUTTON and TODD. "First Report of the Trypanosomiasis Expedition to Senegambia, 1902," Liverpool, 1903.

Idem. "Gland Puncture in Trypanosomiasis," "Thompson Yates and Johnston Laboratories' Report," 1905, Memoir xvi, p. 97.

FORDE. *Journ. Trop. Med and Hyg.*, 1902, v. p. 261.

HARVEY. "Report on a Case of Experimental Sleeping Sickness in a Monkey," *Journ. of Roy. Army Med. Corps*, 1905, iv, p. 621.

HODGES. "Progress Report, Uganda Sleeping Sickness Camps," 1909.

KLEINE. *Deutsch. med. Wochenschr.*, 1909, xxxv, pp. 469, 470, 924, 925, 1257-1260.

KLEINE. "Weitere Untersuchungen uber die Aetiologie der Schlafkrankheit von Stabsarzt. Leiter der Schlafkrankheitsbekampfung in Deutsch Ostafrika." *Deutsch. med. Wochenschr.*, 1910, xxxvi, p. 1400.

KOPKE. "Investigacoes sobre a Doença do Somno," *Arch. de Hyg. e Path. Exocitas*, 1905, i, fasc. 1, pp. 1-65.

LAVERAN. *Compt. Rend. Acad. Sci.*, 1906, cxlii, p. 1065.

MANSON. "Lectures on Tropical Diseases" (Lane Lectures, 1905). (Constable, London.)

MANSON and MOTT. "African Lethargy, or the Sleeping Sickness," *Trans. Path. Soc. Lond.*, 1900, li, p. 99.

MARSHALL. "Trypanosomiasis or Sleeping Sickness," *Rev. of Neur. and Psychiatry*, 1906, iv, p. 112.

MARTIN, LEBŒUF, et ROUBAND. "Rapport de la Mission d'Études de la Maladie du Sommeil au Congo français," 1906-8.

METCHNIKOFF et ROUX. *Annales de l'Inst. Pasteur*, 1904, xviii, pp. 1, 657; 1905, xix, p. 673; 1906, xx, p. 785.

MOTT. The Oliver Sharpey Lectures on "The Cerebrospinal Fluid," *Lancet*, 1910, ii, p. 81.

Idem. "The Changes in the Central Nervous System of Two Cases of Negro Lethargy," sequel to Dr. Manson's Clinical Report, *Brit. Med. Journ.*, 1899, vi, p. 1666.

Idem. "A Case of Negro Lethargy," by STEPHEN MACKENZIE, with a Note on the Histological Changes of the Nervous System, by F. W. MOTT, *Trans. Path. Soc. Lond.*, 1900, li, p. 118.

Idem. "Observations on the Brains of Men and Animals infected with various forms of Trypanosomes," *Proc. Roy. Soc.*, 1905, lxxvi, B, p. 76.

Idem. "System of Syphilis," 1910, iv, pp. 20-36.

Idem. "Histological Observations, &c." Report VII of the Sleeping Sickness Commission, 1906, p. 3.

MOTT and LOW. "The Examination of the Tissues of a Case of Sleeping Sickness in a European," *Brit. Med. Journ.*, 1904, i, p. 1000.

MOTT and STEWART. "Some further Observations on the Cell Changes in Dourine and Sleeping Sickness," *Brit. Med. Journ.*, 1907, ii, p. 1327.

NABARRO. Translation of LAVERAN et MESNIL'S "Trypanosomes et Trypanosomiases," 1907.

NOVY. *Journ. of Infect. Dis.*, Chicago, 1905, ii, p. 250.

PLIMMER. "Note on the Effect produced on Rats by the Trypanosomata of Sleeping Sickness," *Proc. Roy. Soc.*, 1905, lxxiv, p. 388; 1907, B. lxxix, p. 95.

ROGERS. "Preliminary Note on the Development of Trypanosoma in Cultures of the Cunningham-Leishman-Donovan Bodies of Cachexial Fever and Kala-azar," *Lancet*, 1904, ii, p. 215.

SALVIN MOORE and BREINL. *Lancet*, 1907, i, p. 1219.

SCHAUDINN. *Münch. med. Wochenschr.*, 1905, lii, p. 1028.

SICARD et MOUTIER. "Recherche bactériologique et histol. dans un cas de Maladie du Sommeil chez un blanc," *La Presse méd.*, 1905, xiii, p. 422.

"Sleeping Sickness in Whites," Sleeping Sickness Bureau, Bulletin No. 20, 1910.

THOMAS and ANTON BREINL. "Trypanosomes, Trypanosomiasis, and Sleeping Sickness," Report, 1905, vi, part 2 (Thompson Yates and Johnston Laboratories, University of Liverpool).

THOMAS and LINTON. *Lancet*, 1904, ii, pp. 1337-40.

TODD and CHRISTY. *Brit. Med. Journ.*, 1904, i, p. 186.

WARRINGTON. "A Note on the Condition of the Nervous System in a case of African Lethargy," *Brit. Med. Journ.*, 1902, ii, p. 929.

WATSON. "The Pathology and Morbid Histology of Juvenile General Paralysis," *Archives of Neurology*, 1903, ii, p. 621.

WINTERBOTTOM. "An Account of Native Africans in the Neighbourhood of Sierra Leone;" to which is added an account of the present state of Medicine among them. 2 vols., 1803.

Pathological Section.

November 15, 1910.

Dr. Lazarus Barlow in the Chair.

The Bentley-Taylor Method of Mounting Mosquitoes, with Demonstration of Specimens.

By Arthur Powell (Bombay).

The methods hitherto employed in the mounting and preservation of mosquitoes and other small insects have in the hands of the writer proved most unsatisfactory. My observation of the mosquitoes exhibited in European museums and schools of tropical medicine proves that these methods are equally disappointing in the hands of the more expert entomologists. Specimens mounted flat under cover-slips either become too transparent to be of any practical value, or the scales become displaced or lost, while pinned specimens become shrivelled and curled into mere caricatures of the living insect. In damp and tropical climates the inclusion of creasote, naphthol, camphor, and other "preservatives" in the case, as a rule, fails to ward off the attacks of mould and destructive mites and insects, so that when one is desirous of demonstrating to a colleague or a class some rare specimen, all he finds is a mere wreck of the insect, or possibly only the bare pin. All the experts (including Colonel Alcock, F.R.S., Dr. Daniels, Dr. Lieper, and the staff of the London School of Tropical Medicine) who have seen these specimens have expressed their approval or admiration and are adopting this method of preparing them. The credit of inventing this is due to Captain Taylor, I.M.S., and Dr. Bentley, of Bombay. My thanks are due to them for permission to demonstrate it. The method is extremely simple and rapid. The twelve specimens shown were all mounted in twenty-three minutes. No specimens were lost or spoiled in the process, which is as follows:—

(1) Prepare a solution of 1 per cent. celloidin in absolute alcohol.

(2) A solution of celloidin in absolute alcohol of the consistence known as "thick" in ordinary histological work. I believe that used for these insects was 5 per cent.

It is essential that no ether be employed in the solution, as it makes the scales transparent.

(3) Catch the mosquito.

(4) Chloroform it.

(5) When dead or narcotized, place a drop of solution No. 1—thin celloidin—on a cover-glass.

(6) Place the insect back downwards on the cover-glass. In the majority of cases wings and legs spread themselves out in the orthodox exhibition position. If they do not do so, the solution remains fluid sufficiently long enough in Bombay at a temperature of 85° F. to 90° F. for from three to five minutes to permit them to be adjusted with a needle.

(7) When the thin solution has become "tacky," to use the language of the motorist—i.e., in about eight or ten minutes from the commencement of operations—place a drop of the thick solution, No. 2, over the insect.

(8) Invert the cover-glass over a hollowed slide, to which it may be fixed by a ring of balsam.

The specimen is now complete, and in this condition both ventral and dorsal surfaces can be examined under the microscope.

If it be thought desirable to employ a white background, as in the specimen shown, instead of manœuvre (8) proceed thus:—

(8a) Suspend some oxide of zinc in ordinary mounting Canada balsam. Shake or stir well immediately before using; fill the cell or hollow of the slide with this emulsion; invert the cover-glass with the mosquito and press rather firmly into the emulsion.

(9) Clean off any of the emulsion that spreads beyond the edge of the cover.

Specimens prepared by this latter method will, of course, display only one surface.

The Biological Characteristics of Bacillus Typhosus, with especial Reference to the Fermentation of Dulcitol and Arabinose.

By BURTON BRADLEY.[1]

THE following work resulted from a more or less chance observation when investigating the biological properties of an anærogenic organism isolated from fæces. It was noticed that this organism, which in its original state on glucose, mannitol, and dulcitol peptone water gave an acid reaction, could be further induced to ferment lactose and saccharose if left sufficiently long. As the action on glucose, mannitol, and dulcitol occurred in twenty-four hours, whereas the action on lactose and saccharose did not occur until over a week, it seemed to me that in the lactose and saccharose cultures we had quite different conditions from those in glucose, mannitol, or dulcitol. In the latter case the organism readily ferments the sugar, whereas in the former some time of preparation is necessary. It seemed as if only when the organism was acclimatized to the new sugars, or else when *faute de mieux* it needs must use these strange sugars to preserve its life, that they were attacked and broken up. Also it seemed likely that the organism, once having acquired the property, might show it if reinoculated into fresh peptone-water solutions of the same sugars it had "learnt" to ferment. Acting on this assumption, I inoculated three sets of sugars: (1) From the original agar culture; (2) from the saccharose peptone water; and (3) from the lactose peptone water. I append the result in tabular form. It will be seen that from the saccharose culture I have a modification of the original organism fermenting saccharose in two days, while from the lactose I have a modification fermenting lactose in two days.

TABLE I.—ORGANISM FROM CASE OF PSEUDO-TYPHOID.

	Glucose	Mannitol	Dulcitol	Lactose	Saccharose
Original agar culture	A	A	A	—	
Cane-sugar peptone-water culture ...	A	A	A	—	A
Lactose peptone-water culture ...	A	A	A	A	—

Incubated for two days.

---

[1] From the Laboratory of the Government Bureau of Microbiology, Sydney, N.S.W.

I did not pursue the subject further with regard to the above organism, which was merely an interesting specimen weeded out from numerous other types in the fæces, but resolved to test whether the same modification in biological properties could be brought about in an organism, the identification of which is a matter of supreme importance, the *Bacillus typhosus*.

TECHNIQUE.

The only point necessary to mention in this connexion is the formula of the "sugar" media. The "sugar," which name I have used as a general term for the various carbohydrates, alcohols, &c., used, is dissolved or suspended in litmus peptone water. The medium has the following formula :—

Peptone (Witte's)	2·0
Sodium chloride	0·5
Litmus solution (saturated watery)	10·0
Sugar	1·0 (0·5)
Distilled water	up to 100·0

The glucose, mannitol, lactose and saccharose media contained 1 per cent. of respective "sugar." The remainder contained 0·5 per cent. The "sugars" were supplied by Baird and Tatlock, except the saccharose, which was ordinary brewers' crystals.

EXPERIMENT I.—THE BIOLOGICAL CHARACTERISTICS OF BACILLUS TYPHOSUS.

As a preliminary, experiments were carried out to see if the biological characteristics of different strains of the typhoid bacilli were identical. The results of many experiments allow me to draw the following conclusions :—

(1) The reactions of the strains tested give identical reactions in twenty-four hours and remain constant and identical up to four days.

(2) Glucose, mannitol, maltose, galactose, and sorbitol are fermented with the production of acid within twenty-four hours.

(3) No change occurs in lactose, saccharose, inulin, amygdalin, salicin, raffinose, erythritol, and adonitol up to four weeks.

(4) No change occurs in dulcitol or arabinose up to four days. Both are gradually fermented with the production of acid, usually after the first week, at times varying with the strains and also on different occasions of testing. Typhoid grey L. I. P. M. is an exception; dulcitol was never affected by it.

TABLE II.—SHOWING BIOLOGICAL CHARACTERISTICS OF SIXTEEN CULTURES OF BACILLUS TYPHOSUS. ALL INOCULATIONS MADE FROM TWENTY-FOUR-HOUR AGAR CULTURES. COMPILED FROM FOUR EXPERIMENTS.

No.	Morphology	Motility	Gram	Gelatin (20 days)	LITMUS MILK			Glucose	FOUR DAYS' INCUBATION																Remarks
					1 day	6 days	15 days		Mannit.	Dulcit.	Lactose	Saccharose	Maltose	Dextrin	Galactose	Inulin	Amygdalin	Salicin	Arabinose	Raffinose	Sorbit.	Erythrit.	Adonit.	Indol	Agglutination	
M 1	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	? source
M 2	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From a water supply
M 3	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Pasteur Institute, 1902
M 5	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Lister Institute, 1903
M 6	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Lister Institute, 1898
M 7	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Dr. Dick, 1900
M 8	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Lister Institute, 1902
M 9	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Kral, 1902
M 10	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From French, 1902
M 11	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From case of pyosalpinx, 1907
T. Grey	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Lister Institute, 1908
T. Syd. I.	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From blood of patient at Sydney hospital, 1909
Ty. Syd. II	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From blood of patient at Sydney hospital, 1909
Wright	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Lister Institute, 1908
T.A.W.	b	+	—	—	a	a	a	A	A	—	—	—	A	—	A	—	—	—	—	—	A	—	—	—	+	From Dr. Ainley Walker
Control	.	.	.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	

b, bacillus; +, positive result; —, negative result; a, slight acidity; A, marked acidity.

(5) All strains had a *similar* morphology, were motile, Gram-negative non-gelatine liquefiers, did not produce indol, produced slight permanent acidity on litmus milk and were agglutinated well by anti-typhoid serum (prepared from No. 3) in dilutions not less than $\frac{1}{60}$.

It is not possible here to give full details of the four experiments on which these conclusions are formed. The essential facts are, however, shown in Tables II, III, IV.

TABLE III.—TIME TO FERMENT DULCITOL ON THREE OCCASIONS.

M 1	13 days	13 to 19 days	12 days
M 2	7 to 9 ,,	12 ,,	12 ,,
M 3	5 ,,	8 to 10 ,,	6 to 8 ,,
M 5	7 to 9 ,,	12 ,,	15 ,,
M 6	7 to 9 ,,	8 to 10 ,,	10 ,,
M 7	13 ,,	13 to 19 ,,	17 to 19 ,,
M 8	14 ,,	13 to 19 ,,	15 ,,
M 9	7 to 9 ,,	Over 19 ,,	8 ,,
M 10	10 ,,	11 to 12 ,,	12 ,,
M 11	7 to 9 ,,	? 19 ,,	19 ,,
Ty. Grey	—	—	Over 27 ,,
Ty. Syd. I	11 ,,	Over 19 ,,	15 ,,
Ty. Syd. II	—	—	16 ,,
Ty. Wright	7 to 9 ,,	8 ,,	8 ,,
T. A. W.	10 to 11 ,,	11 to 12 ,,	14 ,,

TABLE IV.—TIME IN DAYS TO FERMENT ARABINOSE ON TWO OCCASIONS.

	Second trial	First trial
M 1	10 to 12 days	10 days
M 2	15 ,,	Over 15 ,,
M 3	Over 26 ,,	10 ,,
M 5	10 ,,	10 ,,
M 6	6 to 8 ,,	10 ,,
M 7	14 ,,	Over 15 ,,
M 8	10 ,,	Over 15 ,,
M 9	8 ,,	Over 15 ,,
M 10	13 ,,	Over 15 ,,
M 11	10 to 12 ,,	9 ,,
M Wr.	9 ,,	6 ,,
Ty. Grey	10 ,,	10 ,,
Ty. Syd. I	26 ,,	8 ,,
Ty. Syd. II	10 ,,	Over 15 ,,
T. A. W.	23 to 26 ,,	Over 15 ,,

Table II is compiled from four experiments (in the course of which most of the work has been repeated at least once), and shows the reaction of fifteen strains of *typhosus* up to the end of four days.

Table III, from three of the experiments, shows the times at which the dulcitol was fermented in three trials.

Table IV, from two of the experiments, shows the times at which the arabinose was fermented in two trials.

In one of the above-mentioned experiments the fifteen cultures were inoculated on to the seventeen sugars and results noted on: First, second, third, sixth, eighth, ninth, tenth, twelfth, thirteenth, fourteenth, fifteenth, sixteenth, seventeenth, nineteenth, twentieth, twenty-first, twenty-second, twenty-third, twenty-fourth, and twenty-sixth days.

On the fourteenth day and on the twenty-sixth day the dulcitol and arabinose tubes were examined by means of Gram-stained films. Nothing but Gram-negative bacilli were detected. Controls of the various sugar media showed no acidity in any case.

Having been able to show that 14 of the 15 cultures would ferment dulcitol under twenty-seven days, and all would ferment the arabinose, experiments were designed to show whether a culture, by a sojourn in dulcitol- or arabinose-containing media, would become more active in fermenting these "sugars."

EXPERIMENT IV.—CAN THE DULCITOL FERMENTING POWER OF BACILLUS TYPHOSUS BE ACCENTUATED?

The two agar cultures, T. Wright and T. Syd. Hosp., were inoculated into glucose, mannitol, dulcitol, lactose, and sorbitol, and incubated during twenty-four days, being examined at intervals. Glucose, mannitol, and sorbitol were fermented in twenty-four hours. The dulcitol was not fermented under five days in either case :—

Time to produce acid in dulcitol from T. W.	5 to 9 days
,, ,, ,, ,, T. Syd. Hosp.	9 to 16 ,,

From the original agar cultures, and from the dulcitol peptone water, fresh dulcitol media were inoculated :—

Time to ferment dulcitol from T. W. (agar culture)	8 to 9 days
,, ,, ,, T. W. (dulcitol peptone water) ...	1 to 2 ,,
,, ,, ,, T. Syd. Hosp. (agar culture) ...	Over 26 ,,
,, ,, ,, T. Syd. Hosp. (dulcitol peptone water)	3 ,,

Having, in the above, used cultures of varying age and media, it was next tried whether fresh agar cultures from the dulcitol peptone waters, compared with fresh agar cultures of the original cultures, would still show the difference in rate of fermentation. Accordingly the following experiment was arranged :—

EXPERIMENT V.—CAN THE INCREASED DULCITOL FERMENTING POWER BE TRANSFERRED TO AGAR CULTURES?

Forty-eight hours' agar cultures from T. W., T. Syd. Hosp., T. W. (dulcitol), and T. Syd. Hosp. (dulcitol) were planted on glucose, mannitol, dulcitol, lactose, saccharose, raffinose, and sorbitol. Glucose, mannitol, and sorbitol were fermented within twenty-four hours; lactose, saccharose, and raffinose were unaffected. The dulcitol was fermented as shown below:—

TIME TO FERMENT DULCITOL.

T. W.	9 days
T. W. D.	3 to 4 ,,
T. Syd.	11 to 12 ,,
T. S. D.	6 ,,

Though not quite as great a difference is here manifested, the property acquired of fermenting dulcitol is nevertheless possessed much more strongly by the agar cultures after their sojourn in dulcitol peptone water.

EXPERIMENT VI.

The following experiment demonstrates the same facts both for dulcitol and arabinose. Ten cultures from Experiment I, which had been inoculated on to various media and the sugars and examined daily, were used. After twenty days the results conformed to those of Table II, but, in addition, the dulcitol and arabinose had fermented on the fourteenth, and again on the twenty-sixth day. All the original dulcitols and arabinoses had been examined for contamination by making Gram-stained films. There was no evidence of this, all containing only Gram-negative bacilli. On the twentieth day agar cultures were made from the dulcitols and from the arabinoses. These were, after twenty-

TABLE V.

No.	TIME TO FERMENT DULCIT			TIME TO FERMENT ARABINOSE		
	Original	After passage through dulcit	After passage through arabinose	Original	After passage through dulcit	After passage through arabinose
	(days)	(days)	(days)	(days)	(days)	(days)
M 1 ...	12	5 to 6	Over 7	11 to 12	Over 7	1
M 2 ...	12	1	,,	15	,,	2
M 5 ...	15	5 to 6	,,	10	,,	2
M 6 ...	10	1	,,	7 to 8	,,	1
M 7 ...	18 to 19	3	,,	14	4 to 5	2
M 9 ...	8	1	,,	8	,,	2
M 10 ...	12	1	,,	13	,,	2
M 11 ...	19	3	,,	11 to 12	,,	2
Wright ...	8*	1	,,	9	,,	1
Syd. II ...	16	3	,,	10	,,	1

* The original Wright gave a slight acid reaction on dextrin on the ninth day, probably due to contamination, as in other experiments this has not occurred. Unfortunately, the culture was not further examined for impurity.

four hours' growth, re-inoculated on to all the sugars, and examined for seven days. The results may be expressed as follows:—

(I) In the ten original cultures no dulcitol was fermented under eight days. No arabinose was fermented under seven days.

(II) In the agar culture from dulcitol all dulcitols were fermented under six days; eight of them under three days, five under twenty-four hours. One arabinose (M 7) was fermented between the fourth and fifth days; no other was fermented up to seven days.

(III) In the agar cultures from arabinose no dulcitol was fermented up to seven days. All arabinoses were fermented in two days.

(IV) In none (except possibly M 7) of the cultures after sojourn in dulcitol or in arabinose was there evidence of any increased activity towards any other "sugar" than the dulcit or arabinose respectively.

Table V shows these results in detail.

EXPERIMENT VII.—DOES THE INCREASED DULCITOL FERMENTING POWER PERSIST?

This experiment was designed to show if the increased dulcitol fermenting properties manifested by agar cultures of *Bacillus typhosus* which had had a sojourn in dulcitol persisted through several generations on solid media. To this end, ten successive subcultures were made over a period of a month of T. Wright (T.W.), and T. Wright after sojourn in dulcitol peptone water (T.W.D.), and of T. Sydney Hospital No. 1 (T.S.) and T. Sydney Hospital No. 1 after sojourn in dulcitol peptone water (T.S.D.). These successive subcultures were examined for dulcitol active properties. The table below is self-explanatory:—

TABLE VI.

Date of making agar culture	Date of inoculation of dulcitols	TIME TO FERMENT DULCITOL			
		T.W.	T.W.D.	T.S.	T.S.D.
		(days)	(days)	(days)	(days)
May 5, 1910	May 25, 1910	8	2	13	Between 5 and 6
,, 24, ,,	,, 25, ,,	8	2	13	7
,, 26, ,,	,, 27, ,,	8	Between 2 and 3	10	6
,, 27, ,,	,, 28, ,,	9	Between 1 and 2	12	6
,, 28, ,,	June 1, ,,	12	3	14	7
,, 31, ,,	,, 1, ,,	12	3	15	7
June 1, ,,	,, 7, ,,	10	3	12	7
,, 7, ,,	,, 8, ,,	11	Between 4 and 5	12	11
,, 8, ,,	,, 9, ,,	10	4	15	14
,, 9, ,,	,, 10, ,,	12	5	13	13

From the above, although there is some irregularity in the working of the experiment, it can be seen that the property, though persisting for some time, tends to die out. In the case of T. Sydney it is practically abolished in less than a month of growth in agar, frequently subcultured. In the case of T. Wright the difference is less apparent, the experiment being complicated by the fact that the control cultures seem also to be varying in their power of producing acid in dulcitol.

On another occasion dulcitol active cultures which had been stored for one month still showed increased dulcitol activity.

EXPERIMENT VIII.

Plates were made from *Bacillus typhosus* Wright (June 13, 1910). Ten colonies were picked off (June 14, 1910) and put on agar. All proved to be Gram-negative bacilli. They were inoculated into glucose, mannitol, dulcitol, lactose, saccharose and sorbitol (June 15, 1910). Afterwards, as far as possible every day, agar cultures were made from the dulcitol peptone water-tube, and these agar cultures tested by inoculation into fresh dulcitol (in most cases after twenty-four hours' growth) for their power to ferment dulcitol.

As regards the results on sugars of the original ten cultures:—

In twenty-four hours acid was produced in every case on glucose, mannitol, and sorbitol, and in no case in twenty-four hours on dulcitol, lactose, or cane sugar.

No change occurred in the lactose or cane sugar up to July 14, 1910 (twenty-nine days).

TABLE VII.—WRIGHT. TIME TO FERMENT DULCITOL (IN DAYS).

No.	Original	Agar cultures made from original dulcit on							
		1st day	2nd day	3rd day	5th day	6th day	7th day	8th day	20th day
1	13	11	7	7	3	3	2	3	3 to 4
2	9	14	7	7	3	3	2	2	No growth
3	11	18	8	7	4	4	3	3	3 to 4
4	11	Between 16 and 17	23	7	4	3	2	3	3 to 4
5	Between 18 and 19	28	Between 15 and 16	11	5	4	4	7	4
6	21	26	8	6	5	4	3	3	3 to 4
7	6	9	4	6	2	1	1	1	Over 13
8	10	11	7	9	11	5	4	3	4
9	12	26	7	Between 13 and 14	5	3	3	3	4
10	Between 18 and 19	10	8	9	5	3	3	3	5

The dulcitol inoculated from the original cultures fermented at various times, as shown in the table along with the results of the successive subcultures on agar of these dulcitols.

Agar cultures made from the original dulcitol peptone water-tubes were agglutinated in a 1 in 60 dilution with antityphoid serum, prepared by inoculating a horse with a strain from the Pasteur Institute (No. 3 of Table II).

The agar cultures made on the third day and on the twentieth day were not tested by inoculation on dulcitol till forty-eight hours; all the remainder were tested in *twenty-four hours*.

From Table VII it may be seen:—

(1) That the individual sub-colonies from a culture all undoubtedly *typhosus* (all agglutinating with antityphoid serum) show marked variations in respect to their power of fermenting dulcitol.

(2) That at first, more or less *pari passu* with the length of sojourn in dulcitol, the agar cultures made from the dulcitol show increased power of fermenting this substance. After a certain length of time further length of stay in the dulcitol does not appear to increase the fermenting capabilities.

The lactose and saccharose cultures, which showed no change up to twenty-nine days, were on the sixteenth day inoculated into glucose, mannitol, dulcitol, lactose, and saccharose, and grown for thirteen days.

(1) No power to ferment either lactose or saccharose was manifested.

(2) From the saccharose tubes no dulcitol was fermented under nine days.

(3) From the lactose tubes no dulcitol was fermented under six days.

From which it may be concluded that the increase in fermentative activity manifested by cultures of Bacillus typhosus after a sojourn in peptone-water solutions of dulcitol is not due to growing in peptone water; that growth for a long period on peptone-water solution of lactose or saccharose does not tend to make the typhoid organism acquire the property of fermenting either of them.

CONCLUSIONS.

(1) The fermentation reactions of *Bacillus typhosus* are identical up to the fourth day.

(2) *Bacillus typhosus* ferments glucose, mannitol, galactose, and sorbitol with the formation of acid within twenty-four hours. Dulcitol[1]

---

[1] One culture, T. Grey L. I. P. M., never fermented dulcitol.

and arabinose are fermented more slowly, acid being produced usually during the second or third week. Lactose, saccharose, dextrin, inulin, amygdalin, salicin, raffinose, erythritol, and adonitol are not fermented. Gas is never produced by *Bacillus typhosus.* Litmus milk is turned faintly acid generally within twenty-four hours, and remains thus permanently.

(3) The time taken to ferment dulcitol or arabinose varies widely even in the same culture tested repeatedly, and also amongst daughter colonies from a single-plated culture.

(4) The time taken by a given strain of *Bacillus typhosus* to ferment dulcitol or arabinose is very markedly quickened for that strain by a sojourn in media containing dulcitol or arabinose respectively.

(5) Within certain limits the increase in (dulcitol) fermenting powers varies with the length of sojourn in dulcitol-containing medium.

(6) The increased activity as regards fermentation of dulcitol persists through several generations of agar cultures, but eventually tends to die out. It also persists on stored agar cultures for over a month if this culture be sub-cultivated and tested.

(7) No other modifications either as regards agglutination, fermentation reactions, or cultural characteristics are seen in the agar cultures after passage through dulcitol (or arabinose) containing media other than the increased activity towards the corresponding "sugar." The cultures after passage through dulcitol become "dulcitol active," and after passage through arabinose "arabinose active." The "dulcitol active" cultures do not show any increased activity towards arabinose, nor do the "arabinose active" cultures show any increased activity towards dulcitol.

(8) Mere passage through peptone water or through other sugars —e.g., mannitol, lactose, or saccharose—does not increase the fermentative activity towards dulcitol or arabinose.

(9) It was not found possible to make *Bacillus typhosus* ferment lactose or saccharose.

(10) By means of passage through appropriate sugars it was found possible to develop two modifications of an anærogenic coliform organism, each active toward a new sugar not easily affected by the original strain.

(11) This work shows that the fermentation activity of *Bacillus typhosus,* while one of remarkable constancy in quality, can be increased in quantity, and suggests that certain work of classification amongst colon organisms requires reconsideration.

When the above work was nearly completed, I found a paper by Twort,[1] in which the following conclusions run closely parallel to mine: He says that he has been able to make several microbes ultimately attack sugars which, according to universal bacteriological opinion, they do not attack under normal conditions. He says he has been able to make *Bacillus typhosus* ferment lactose and dulcitol, and has shown that the dulcitol activity persisted on agar cultures through several generations, and also after passage through animals. *Bacillus paratyphosus*, *Bacillus dysenteriæ* Kruse, *Bacillus dysenteriæ* Flexner, &c., are also able to be made in a similar way to attack new sugars.[2]

The Influence of Diet upon the Formation and Healing of Acute Ulcer of the Stomach.

By CHARLES BOLTON.

THE present research is one of a series which was initiated in order to inquire into the exact part played by the gastric juice in the production and healing of acute gastric ulcer. For many years the hypothesis has been prevalent that the gastric juice plays an important rôle in the pathology of gastric ulcer, mainly because ulcers having the characters of those found in the stomach are almost entirely limited to the parts of the alimentary canal which are directly exposed to the action of the gastric juice. Many experiments indirectly bearing upon this problem have been performed in order to substantiate this hypothesis, but, it must be confessed, without much success. I have no intention in this short communication of discussing the relative merits of these experiments, because I have already referred to most of them in various other papers, and because they have no particular bearing upon the subject of this discourse. In all my experiments acute gastric ulcers have been produced in animals by the injection of gastrotoxic serum, the preparation of which I was able to demonstrate some years ago [1]. By means of this method of producing gastric ulcer I have arrived at the following definite conclusions, which have been already published in various papers:—

---

[1] Twort, F. W., *Proc. Roy. Soc.*, B, lxxix, 1907, p. 329.

[2] The author's attention may be directed to the more recent literature on the subject of variation and mutation by Reiner Müller, Burk, Massini, Penfold, and Wilson.

(1) That acute ulcer fails to appear if the gastric juice be put out of action, although the animal may die from the effects of the poison.

(2) That the ulceration produced in the presence of hyperacid gastric juice is much more extensive than that produced in a stomach secreting juice of the normal acidity [2].

(3) That the gastric juice is able to attack the gastric cells and produce an ulcer, although the cells are not actually killed by the poison. It is merely necessary to damage the cells to some extent.

(4) That hydrochloric acid of the various strengths found in the condition of hyperacidity in the human subject is able to act as a poison for the gastric cells.

(5) That acute gastric ulcer heals equally well, whether the gastric juice be increased or diminished in acidity to the extent usually found in man, provided that the stomach empties itself in the normal time [3].

(6) That acute ulcer is more easily produced in the digesting than in the resting stomach, and that in the former case it is much more extensive in character.

(7) That undue retention of food in the stomach delays the healing of acute ulcer for at least twice the normal time, because the prolonged action of the gastric juice irritates the base of the ulcer, and may cause necrosis of the granulation tissue in the early stages and excessive formation of fibrous tissue in the later stages. The growth of the new mucous membrane over the base of the ulcer is thus delayed, and there is produced more inflammatory thickening than usually occurs in the condition of normal healing [4].

Since motor insufficiency of the stomach prolongs the period of healing of an ulcer, it follows that food which is retained for a considerable time in the stomach, and which excites a copious secretion of gastric juice, should be more unfavourable to the healing of ulcer than food which leaves the stomach rapidly and excites only a moderate flow of gastric juice. This is a question of great importance to the clinician, as I think most physicians will agree that rectal feeding is inadvisable in any case in which it can be avoided, so that it is necessary to have the question definitely settled with regard to what diet is the most suitable for a case of gastric ulcer. In all the present experiments cats were used. The ulcer was in each case produced on the anterior wall of the stomach midway between the cardiac and pyloric orifices by the local injection of gastrotoxic serum, obtained by immunizing the goat with the gastric cells of the cat, as described in a former paper [5]. The two diets chosen were meat and milk respectively. After a meal of 100 grm. to 120 grm. of meat, the cat's stomach is usually not empty till a period of about twelve hours has elapsed, whilst after a meal of 6 oz. of milk it is empty in three hours. The stomach of a 3,000-grm. cat will hold when fully distended a little over half a pint, but the animal will only drink about 6 oz. at once. There is the mechanical irritation of the meat also to be taken into account; but perhaps this is not of so much importance, because small clots are formed in the milk which will act in a similar manner. It was first demonstrated by Griffini and Vassale [7] that after removal of a portion of mucous membrane by the knife the new mucous membrane was regenerated by proliferation of the glandular epithelium at the edge of the lesion, the cells growing over the raw surface, which they cover in a single layer. The glands are subsequently developed from this single layer of cells.

In my experiments on motor insufficiency I found that the delay in healing occurred in the early stages before the single layer of cells had developed, but that when once the base of the ulcer was covered with epithelium the regeneration of the mucous membrane was chiefly a question of time, but was also to some extent dependent upon the density of the fibrous base of the ulcer. From the clinical point of view, therefore, the important point in promoting the healing of ulcer is to exercise special care in the early stages of the healing so that the single layer of cells covers the base of the ulcer as rapidly as possible, and when once that is accomplished the chief difficulty is over. I have observed the healing of acute ulcer in twenty-six cats up to the stage at which the base of the ulcer is covered with a single layer of epithelial cells, because, for the purposes of my inquiry, it was sufficient to observe the healing up to this stage. Thirteen cats were placed on a meat diet and thirteen on milk. The animals were killed at various stages, and the ulcers of twenty of these animals examined microscopically. It was not necessary to examine the remaining six with the microscope, because they were quite in an early state and the condition obvious to the naked eye. The exact time of healing, of course, depends upon the size of the ulcer. The size of the ulcer varies in proportion to the strength of the serum, which in its turn depends upon the stage of immunization which the goat has reached. In the present instance all the ulcers were produced by injection of the same dose of serum obtained from a goat at the same time, so that all the ulcers were of the same size. The serum which is injected so as to cause a local œdema of the stomach wall soaks into the mucous membrane and other tissues. The portion of stomach wall so exposed to the action of the serum is digested by the gastric juice and converted into a slough which separates, leaving a clean ulcer.

The formation of the ulcer varies according to the condition of the contents of the stomach. When the animal is on a meat diet, so that the walls of the stomach are exposed to the prolonged action of the gastric juice, the slough is rapidly formed and has separated by the fourth day in most cases, a clean ulcer resulting. In the case of animals fed on milk, however, the period of formation of the ulcer is longer. In two of the cases the sloughs had not separated on the seventh day, and in two other animals killed on the eighth and eleventh days respectively a small slough was still adherent to the centre of the base. Out of a large number of animals fed on a meat diet in previous experiments I have never yet encountered an unseparated slough like this at so late a date. There is no doubt whatever that the condition of the stomach contents exercises a great influence upon both the formation and extension of an ulcer in whatever way it is produced, and we know that in the human being acute ulcer is initiated in several ways, but in all cases, by the action of the gastric juice, the different lesions are eventually converted into precisely the same kind of ulcer. It is by no means uncommon for a sudden hæmorrhage or perforation to occur after a meal, and this is not altogether due to the stretching and movements of the walls of the stomach, but also to digestion by the gastric juice. These experiments support the result which I gave at the beginning of this paper—namely, that an ulcer is produced more easily in the digesting than in the resting stomach. On account of the delay in the separation of the slough on a milk diet, I placed six further animals on meat for the first four days, and subsequently on milk, so that the experiments would be more strictly comparable with those in which the animals were fed on meat all the time.

The microscopical examination of a lesion on the third day shows the overlying mucous membrane completely necrosed, the tissue being shrunken and unstained, and at the junction of the dead and living tissue an infiltration with round cells, chiefly leucocytes. The muscularis mucosæ has a hyaline appearance and stains badly. The submucous and muscular coats are thickened, infiltrated with hæmorrhages, and contain much necrotic tissue having a hyaline appearance. There is commencing infiltration with cells around the edge, which are invading the dead tissue.

After the slough has separated, the base of the ulcer is formed of the remains of the muscular coat or the subperitoneal tissue infiltrated with round cells. Towards the end of the second week the deeper layers of the base have become fibrous and the peritoneum is thickened, the superficial layers being formed of granulation tissue. At the end of the third week the whole base is formed of fibrous tissue in which strands of muscular tissue can be seen. This blends with the muscular and submucous layers at the edges of the ulcer.

Until the epithelium has grown over the base the most superficial layer is formed of more vascular and cellular tissue. This is the invariable course of events in every animal fed on milk, but in those fed on meat the granulation tissue on the surface of the base and extending to various depths is liable to become necrotic. It may be only in a part of its superficial extent or in the whole of it. In this case the growth of the epithelium over the base is completely prevented, and when the whole superficial extent is involved, the epithelium has merely commenced to grow at the edge of the ulcer and the whole is completely unhealed at the end of three weeks. The size of such an ulcer is always much smaller than the original ulcer owing to contraction of the fibrous tissue in the base. This necrosis of the granulation tissue of the base of the ulcer occurred in three out of five animals which were fed on meat, and which were killed from the fourteenth to the twentieth day. The ulcers of eight animals fed on milk were examined during the same period, and in not a single case was there any sign of such necrosis.

This is the essential difference between the healing in the two cases: the granulation tissue of the base is unable to withstand the prolonged action of the gastric juice and undergoes necrosis, with the result that the healing is delayed, and the base of the ulcer becomes more dense from increased formation of fibrous tissue.

I will next consider the regeneration of the mucous membrane. As soon as the edges of the mucous membrane are separated from the slough, and all the necrotic portions of glands have been removed by the gastric juice, the cells lining the ducts and bodies of the glands, which are apparent on the raw surface, commence to proliferate and cover this surface with a single layer of at first flattened and later cubical cells. In this way the edge becomes smooth and rounded. On section this edge is composed of short, irregular glands separated by a considerable cellular stroma, the surface being covered by a layer of cubical cells, which is continued in a single layer on to the surface of the ulcer. Not uncommonly the edge has been inverted by retraction of the muscularis mucosæ, and in this case the epithelial cells are compelled to grow round the angle between this inverted edge and the base of the ulcer. This, however, appears to offer little or no obstacle to their growth. By the tenth or eleventh day in the case of milk-fed, and by the thirteenth or fourteenth in the case of meat-fed animals, the layer of epithelial cells has extended a little way over the base of the ulcer. On the twentieth day the base of the ulcer was completely covered in four milk-fed animals, and almost completely in three others. Of the animals fed on meat, the layer of cells had only extended a short distance over the base on the fifteenth day, and of three cases examined on the twentieth day, one had a large uncovered area in the centre, and in the other two the base was completely uncovered owing to its superficial layers having undergone necrosis. The earliest time at which I have seen primitive glands develop is the tenth to the fifteenth day. They appear at first as little pouches invaginated from the layer of cubical cells.

These results, therefore, confirm those of my experiments dealing with motor insufficiency, and show that the delay in healing in that condition is merely an exaggeration of that which occurs under normal circumstances, when a diet is administered which remains for a long period of time in the stomach, the fault being in both cases in the base of the ulcer rather than in the overgrowing epithelium.

The conclusions to be drawn from these experiments are as follows:—

(1) The theory with regard to the part played by gastric juice in the production of gastric ulcer receives further confirmation, because ulceration is the more rapidly produced in proportion as the gastric juice is allowed a longer period of contact with the wall of the stomach.

(2) The epithelium grows over the base of an ulcer more rapidly when the animal is given a milk diet than when it is given a meat diet. In the case of a milk diet the base of a moderately-sized ulcer is usually completely covered up by the twentieth day, whilst in the case of a meat diet the same sized ulcer would in most cases be uncovered in the centre at that time.

(3) Frequently in the case of meat-fed animals the ulcer is completely uncovered on the twentieth day, the granulation tissue of the base of the ulcer having become necrotic. Such an ulcer may be only one-sixth of the size of the original ulcer owing to the contraction of the fibrous tissue in the base, although healing has only commenced at the edge.

(4) In the treatment of a case of ulcer of the stomach, the following principles should be observed: (*a*) During the early stages of the healing

of acute ulcer the patient should be given a food which does not stay long in the stomach, and which does not excite a copious flow of gastric juice. (*b*) The period of treatment in bed should be at least three weeks. (*c*) The starvation diet of the older physicians is not necessary, because the general nutrition suffers too much, and because ulcers heal well on some diet such as the above. (*d*) In the case of acute ulcers which are extending, or chronic ulcers, healing cannot be expected to occur in three weeks, because the ulcer must first be got into a suitable condition for healing, and then, owing to its size and thickness, the healing must take some weeks longer to be completed ; so that the treatment in bed is to be conducted like that of simple acute ulcer, but extended over a period at least twice as long. (*e*) Since in many cases of gastric ulcer there is hyperacidity of the gastric juice, and when the gastric juice is acting destructively, hyperacidity increases this destructive tendency, the high degree of acidity should be controlled by the administration of alkalies. This is not so necessary in acute ulcer as in the more chronic forms, because the few estimations that have been made of the gastric secretion in acute ulcer show that it is not hyperacid, and I have found experimentally that the effect of acute ulcer is to diminish the secretion in the early stages, and that the latter becomes normal as the ulcer heals [6].

REFERENCES.

[1] BOLTON. *Proc. Roy. Soc.*, 1904, lxxiv, p. 135.
[2] *Idem.* Loc. cit., 1907, B, lxxix, p. 533.
[3] *Idem.* *Proc. Roy. Soc. Med.* (Path. Sect.), 1908, ii, p. 54.
[4] *Idem.* *Proc. Roy. Soc.*, 1910, B, lxxxii, p. 233.
[5] *Idem.* *See* Reference 4.
[6] *Idem.* *Journ. Path. and Bact.*, Camb., 1909-10, xiv, p. 418.
[7] GRIFFINI und VASSALE. *Beitr. zur path. Anat.*, Jena, 1888, iii, p. 425.

Atypical Chloroma.

By A. C. D. Firth and J. C. G. Ledingham.[1]

During the last few years the occurrence of certain forms of chloromatous growth differing widely from the typical picture of chloroma has led to considerable modification of our views regarding the position of this disease as an independent entity, and there appears to be an increasing tendency to bring it into line with the primary diseases of the hæmopoietic system, and especially with the acute leukæmias. In its typical form chloroma is a disease occurring mostly in young subjects and characterized by the presence of green tumour-like infiltrates connected principally with the periosteum of the bones of the skull, sternum, ribs, and vertebræ. The blood-changes described in various cases vary considerably, but the differential blood-picture has been found to resemble that of the acute leukæmias, which may or may not be associated with any very marked increase in the total leucocyte count.

Although chloroma is rare, the literature of the subject is fairly extensive, and it would serve no useful purpose to refer to it here. We shall confine ourselves to some account of the variants of the chloroma picture and discuss these in the light of an interesting case observed at the Victoria Hospital in February, 1909, which in our opinion supplies another link in the chain of atypical cases connecting the acute leukæmias with chloroma.

The following are brief notes of the clinical history and post-mortem examination:—

The patient, F. G., male, aged 12 months, was admitted to the Victoria Hospital on January 22, 1909. No family history was obtained, as the child was illegitimate and had been sent out to nurse. No illness had been recorded until fourteen days before admission, when signs of bronchitis appeared. A rash on the chest was noticed on January 21, and was attributed to poulticing.

On admission, the child was well nourished. He had a frequent short cough and some nasal obstruction. Coryza was well marked, the nasal discharge being purulent in character. A few small conjunctival hæmorrhages were present in both eyes. The skin had a faint yellowish tinge, and on the forehead were the faded remains of a scattered macular rash. On the chest the rash was purpuric, some of the spots being of the size of a split-pea, but nowhere confluent. The abdomen, back, arms, and legs were quite free from purpura. The post-occipital and cervical glands were markedly enlarged and those in the axilla and groin palpable. The tongue was covered with thick white fur. Two small ulcers were visible on the hard palate. The gums were normal and no Koplik's spots were to be seen on the buccal mucosa. The tonsils were swollen and red, but no membrane was present. Examination of the chest revealed a considerable amount of bronchitis, but no other abnormality. The abdomen was distended and the liver and spleen were not palpable. All joints seemed perfectly healthy and no signs of rickets were observed. The urine was normal.

---

[1] From the Victoria Hospital for Children and Lister Institute.

Course of the illness: For the next few days the condition of the child improved. The purpura on the chest began to fade and the bronchitis diminished. Cultures from the nose and throat negatived any idea of diphtheria. The nasopharynx was filled with adenoid vegetations. Four days after admission a mottled purpuric rash appeared on the abdomen and slowly spread round to the flanks. On the same day a gland beneath the angle of the jaw on the right side was more swollen and tender. Until January 29 the temperature did not exceed 100·4° F., but on that morning it rose to 104° F., and careful examination revealed no cause for this. For the next ten days little change took place in the general condition; the purpuric rash was fading, but the glandular enlargement persisted. The urine at this time contained a trace of albumin. On February 6 fluctuation was obtained in the right preauricular gland, and about a drachm of pus was evacuated. After this the other glands about the head and neck diminished a little in size, and the temperature returned to its previous range between normal and 100° F. The nasal discharge ceased and the stools, which on admission had been loose, green, and offensive, became normal. On February 19 a change in the condition of the child was evident. He was becoming puffy about the eyes and a curious greyish tint appeared on the scalp and forehead. The glandular enlargement slowly increased and the upper ends of tibiæ and lower ends of femora were markedly tender on percussion. The sternum and ribs were not. A day or two later similar tenderness was observed on percussing the cranium. On February 28 a definite enlargement of the lower end of each femur and the upper end of each tibia was observed. On March 4 the facial features had entirely changed, owing to the solid œdema of the scalp and skin. The eyes were nearly submerged. The upper part of the face and scalp were shining and of a greenish-grey tint, the skin elsewhere presenting a dirty yellow colour. The popliteal glands had become palpable, while the bony swellings of the legs were more definite. A few purpuric spots appeared on the chest and abdomen about this time (March 4), and similar spots continued to appear until the termination of the illness. From that time the condition of the child remained unchanged, except that the green tint about the head became more marked. On March 12 vomiting occurred and the temperature rose to 103° F. Death took place next day, March 13, the temperature before death rising to 106° F.

A blood examination carried out on March 9 gave the following result: Hæmoglobin, 49 per cent.; red cells, 2,920,000; leucocytes, 40,000. Differential count of leucocytes: Small lymphocytes, 48·2 per cent.; large lymphocytes, 41·7 per cent.; polymorphonuclears, 8·2 per cent.; myelocytes, ·25 per cent.; eosinophils, 1·0 per cent.; mast cells, ·5 per cent.

The red cells varied greatly in size and a slight degree of poikilocytosis was present. Here and there a red cell showing punctate basophilia was found. In a count of 400 leucocytes, three nucleated red cells were met.

Abstract of protocol: Body is poorly nourished. The cranium appears large and bossy, especially over the temporal, orbital, and malar regions. The skin over scalp has a decided greenish tinge, but elsewhere presents no discoloration. Round the orbits and malar bones the skin is much swollen and evidently thickened. Thoracic organs present no pathological lesions, and the bronchial glands are not enlarged. There was no enlargement of the liver or spleen. Kidneys appeared healthy, and the mucosa of intestinal tract showed no changes. Along the descending aorta to the brim of the pelvis was a string of slightly enlarged glands. A few were also present in the groins. Cranium: While removing the skull-cap, the skin over the scalp was found to be greatly thickened and fleshy, but showed no greenish colour on section. Over the orbital and malar regions the skin was similarly thickened. Underneath the thickened scalp bare bone was found. The dura mater was enormously thickened and intimately adherent to the cranial vault. After stripping it off, its periosteal surface presented a reddish appearance with irregular warty protuberances. The bony vault was deeply set with osteophytic spicules which had been lodged in the thickened dura (*see* fig. 1). This condition of the dura was confined to the vertical and parietal regions. The brain itself presented no obvious lesions. Long bones: The periosteum of the long bones, tibiæ, femora, and humeri, was much thickened, especially in the neighbourhood of the epiphyses. Section through the thickened periosteum presented an appearance similar to that of the dura mater. The underlying bone shaft

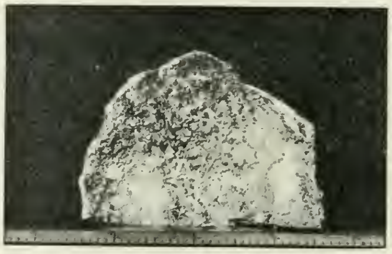


FIG. 1.

Inner surface of parietal bone showing osteophytic spicules which projected into the underlying subdural infiltrate.

was quite hard and was studded with spicules which had penetrated the subperiosteal infiltrate. There were no discrete tumour-like masses in connexion with the periosteum. The change consisted in a more or less uniform thickening of the periosteal tissue over the whole shaft of the bone (*see* fig. 2). Lymph glands: A few soft enlarged glands were present near the tragus and in the parotid region. The external cervical glands also showed some enlargement, and a few were found in the popliteal space. The bone marrow of the long bones was red and fleshy. Cancellous bone appeared to be largely absorbed, leaving merely a hard outer shell. There were no prevertebral infiltrates.

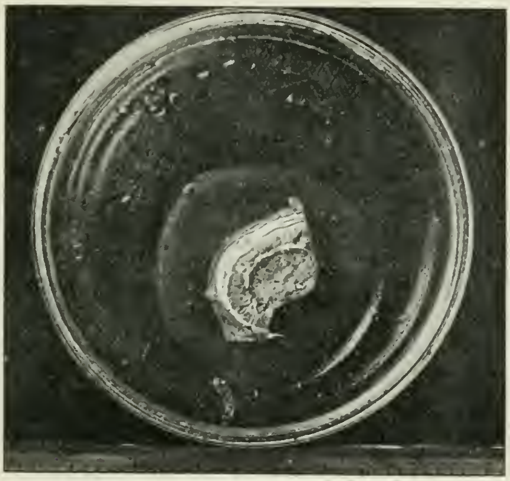


Fig. 2.

Cross section of femur shaft showing subperiosteal infiltrate as a sheath round the bone.

Histological Examination.

Section of periosteal infiltrate of femur in a plane perpendicular to the shaft (*see* fig. 3): Three cell layers are readily distinguishable, viz.: (*a*) Subperiosteal tumour layer in immediate contact with the shaft and containing bony spicules embedded in it; (*b*) connective-tissue layer representing the periosteum proper; and (*c*) extra-periosteal areolar and fatty tissue layer.

Layer (*a*) (*see* Plate I) consists of parallel rows of tumour-cells, the great majority of which have undergone karyorrhexis. The morphology and staining reaction of the degenerated cells differed so considerably from that of the healthy tumour-cell that some difficulty was experienced at first in recognizing the relationship of the two elements. The normal tumour-cell varies little in size and corresponds in dimensions to the large lymphocyte of the blood. The cytoplasm is pale and non-granular, and the nucleus is, as a rule, large and very deficient in chromatin (*see* Plate II, *D*). The cell which has undergone karyorrhexis is much smaller and shrunken, its cytoplasm stains intensely with acid stains ("Licht-grün" in the Twort mixture), while the nucleus is broken down into two or three rounded deeply pyknotic portions. The acidophilia of the cytoplasm was explained by the finding here and there of mitotic cells presenting at each pole a deeply-stained chromatin body and a cell-body showing the acidophilia accompanying division. The extraordinary degree of karyorrhexis was therefore the result of speedy degeneration following division (*see* Plate II, *A*, *B*). Lying amongst the karyorrhectic cells were enormous numbers of pyknotic nuclear particles, either free or included in the phagocytic cells, some of which attained very large dimensions (*see* Plate II, *C*). Occasionally the normal tumour-cell contained in its cytoplasm an included karyorrhectic cell.

Layer (*b*) consists of connective tissue infiltrated with tumour elements. Here the karyorrhectic tumour-cells lie in no regular order, but are squeezed between the connective-tissue fibres where their nuclei are drawn out into long processes of bizarre shape. Frequently rows of karyorrhetic cells are found lying in the perivascular lymphatic vessel, but never in the blood-vessel itself.

Layer (*c*) consists of loose areolar tissue and fat spaces with blood-vessels. The perivascular spaces are all stuffed with tumour elements forming dense aggregations, and the karyorrhexis and pyknosis are most marked in this layer. The muscular attachments were also infiltrated, but there was no evidence of destruction of the muscle tissue.

Section of thickened dura mater: The dura mater requires no special mention, as the arrangement and structure of its layers exactly resemble that found in the periosteal infiltrate.

Section of skin of scalp: The epidermis is thinned out owing to the enormous accumulation of tumour elements beneath. Among the broken-down cells are numerous phagocytes with included nuclear débris. In the subcutis the areolar and fatty tissues are densely infiltrated. No osteophytic spicules from the external table penetrated the infiltrate.

Bone-marrow of femur: Films of the bone-marrow taken at autopsy and stained with Giemsa showed accumulations of nuclear débris, round or crescent-shaped, and lying either free or surrounded by portions of acidophilic cytoplasm. There were also intact tumour-like cells with vesicular nuclei. No nucleated red cells, or granular cells, or giant cells were seen. In sections stained with Laurent's eosin-methylene-blue mixture it is seen that the fat cells have completely disappeared and the marrow, as a whole, presents a similar structure to that of the subperiosteal infiltrate already described. The tissue is much congested with red cells and a very few normoblastic foci remain. Granular cells (polymorphonuclears, myelocytes and eosinophiles) are very scarce and no giant cells are present. A few thin, bony lamellæ persist.

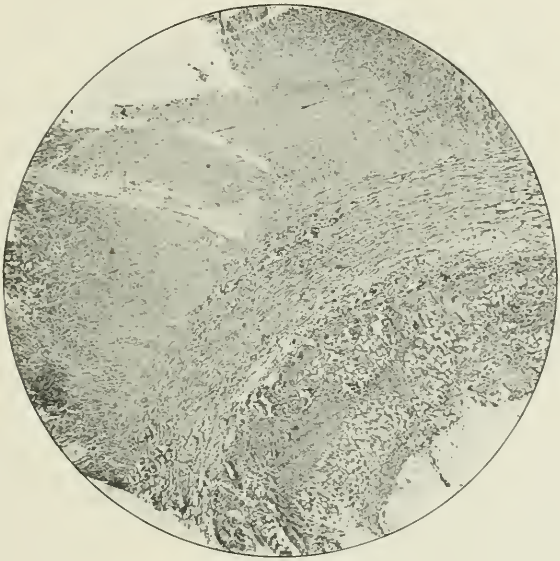


Fig. 3.

Section through subperiosteal infiltrate of femur, periosteum, and extraperiosteal tissue. The long white areas in the subperiosteal layer are osteophytes. (× 15.)

Spleen : This organ, which was not enlarged, presents on section no evidence of infiltration. There are numerous large endothelial cells, but they contain no nuclear débris. The cells of the pulp are, in the main, of the large non-granular type. A few small nodes are present, but their germ centres are frequently broken down.

Lymphatic gland from cervical region : The normal gland structure was replaced almost entirely by accumulations of cells similar to those in the infiltrates elsewhere. The periglandular tissue was also invaded ; only a few small nodes remained. Lymphatic glands in other regions presented a similar appearance.

DESCRIPTION OF PLATE.

Figure I.

Leitz obj. 6; Oc. 4. Stain: Licht-grün, neutral-red (Twort).

Section of subperiosteal layer showing the parallel arrangement of the infiltrating cells; numerous cells with karyorrhectic nuclei; phagocytes containing nuclear débris; osteophyte on the left.

Figure II.

Leitz obj. $\frac{1}{12}$ oil immersion; Oc. 4. Stain (Twort).

A.—Cells undergoing mitosis.
B.—Karyorrhexis of nuclei.
C.—Large phagocytes containing nuclear débris.
D.—Normal cells of infiltrate.
E.—Phagocytosis of nuclear fragment by a single tumour cell.

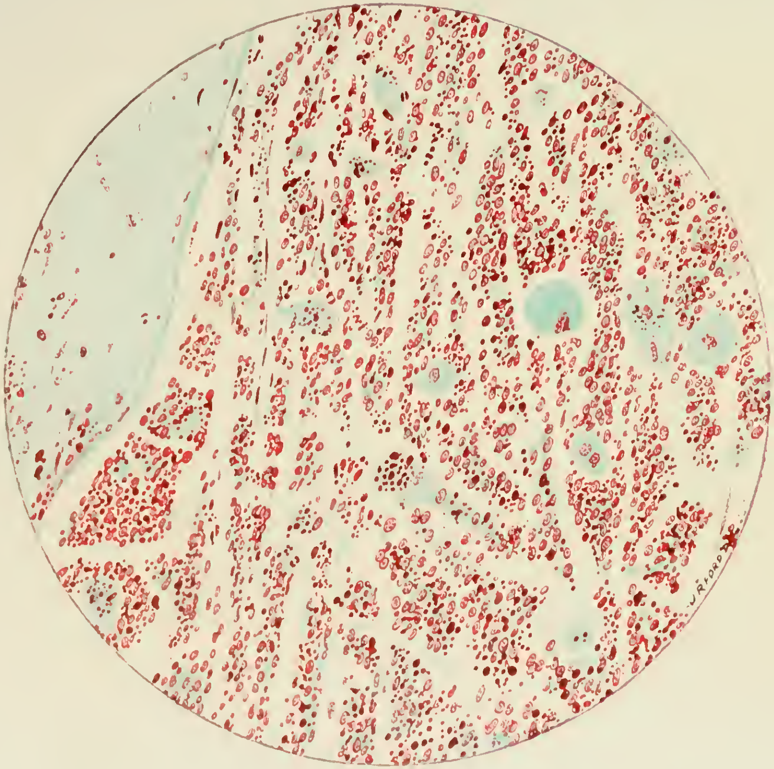


Fig. 1.

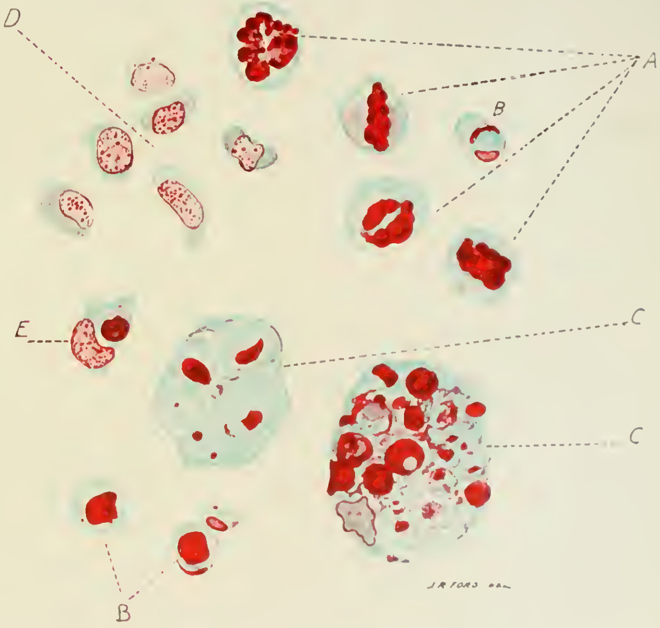


Fig. 2.

FIRTH & LEDINGHAM: Atypical Chloroma.

Liver: The central portions of the lobules had undergone fatty change. Infiltration of the interacinar capillaries with the tumour cells was very little marked, but here and there cells showing mitosis were met with.

Kidney: The only portion of the kidney which showed evidence of infiltration with tumour cells was the tissue immediately subjacent to the epithelial lining of the calices.

No micro-organisms were found in any of the tissues examined.

We have therefore before us a case which presented many of the features of chloroma, notably the distribution of the infiltrates in the orbital region, scalp and meninges, with, in addition, the presence of similar infiltrates connected with the periosteum of the long bones. The presence of infiltrates in the latter situation has hitherto been regarded by all writers as of rare occurrence. Essex Wynter (1909) has, however, recently described to a Section of this Society a case of chloromatous growth in which infiltrates in the neighbourhood of the ends of the long bones were also found. In our case there was no green colouration whatever of the infiltrates, and in no situation did these infiltrates take on an obviously tumour-like character. If the green colouration of the growth is essential before one can enrol a particular case in the category of chloroma, then the evidence in our case rests solely on the greenish colouration of the skin of the scalp and orbital regions which appeared in the late stages of the disease. It would appear, however, that in many of the recorded cases the green tint may be confined to the infiltrates in certain situations and wanting in others. Recently Lehndorff (1910), in discussing variant forms of chloroma, has described two cases of great interest from the point of view of the colour of these tumours. Like our case, both his cases were brought to hospital on suspicion of septic diphtheria. His first case showed progressive ulcerative processes in the tonsils, hard palate, gums and uvula, with swollen cervical glands; but at autopsy all the lymph glands were found to be enlarged, while those in the neck only, were green on section. There were no infiltrates connected with periosteum. The blood during life showed an aleukæmic stage, followed by a marked increase from 5,100 to 85,000 leucocytes per cubic millimetre, the increase affecting principally the myelocytes and myeloblasts. The designation "myeloid chloroleukæmia" would therefore be applicable to it. His second case also showed necrotic ulcers in the throat and tonsils, while the leucocytes, of which 95 per cent. were large lymphocytes, reached a total of 118,700 per cubic millimetre. At the autopsy the spleen was enlarged, the kidneys showed diffuse infiltration, while the cervical glands only, were bright green on section. Had it not been for the green colour of the cervical glands, therefore, the case would have been described as an acute lymphatic leukæmia. There were no periosteal infiltrates. The changes in the blood-forming organs were not lymphomatous, as might have been expected from the blood-picture, but myeloid; so that the second case might be defined as a myeloblastic chloroleukæmia. These two cases, although they possess one important feature of the chloroma picture—viz., the green colour, confined, however, to the cervical glands—are in other respects far removed from the typical picture of chloroma; but, nevertheless, in view of the colouration, they must be considered as links uniting the acute leukæmias to the chloromas; and, further, they show that the green colour, although the most striking feature of chloroma, is not necessarily of first importance. Treadgold (1908) has also reported two very similar cases in which the cervical glands showed a green tint on section, while other features of typical chloroma were absent. Again, we find in a case reported by Johannson and Moritz (1908) that green areas may be solely confined to the bone-marrow, with entire absence of periosteal infiltrates. The great majority of the leucocytes in their case consisted of lymphocytes, and they consider it questionable whether it is always possible to distinguish sharply the lymphatic form of chloroma associated with large lymphocytes from the myeloid form associated mainly with myeloblasts. In our case the cells of the infiltrates and of the bone-marrow resembled more closely the large lymphocytes than the myeloblasts, and, further, the cells of the granular series were extremely scarce in the bone-marrow, which was almost entirely occupied by the non-granular cells similar to those found in the infiltrates elsewhere.

Coming now to the distribution of the infiltrates in chloroma, we find that in no sense can they be regarded as features peculiar to this disease. In a number of cases of acute leukæmia, collected by Glinski (1903) where no green colouration was present, infiltrates have been found in situations similar to those of chloroma—e.g., in dura mater and scalp, ribs, and prevertebral region. Glinski's own case of acute lymphatic leukæmia presented infiltrates at the costochondral junctions of the ribs, in the epicardium, where they also invaded the heart-muscle fibres, and also, what is exceedingly rare, in the leptomeninges. Naegeli has in fact spoken of Glinski's case and other similar cases as "Ungefärbte Chloromen." With regard to the osteophytic formations which are so prominent a feature of our case, we have been unable to find accounts of similar bone changes in other cases. There have certainly been erosions of the vault where it has come in contact with chloroma infiltrates, but reactive changes have apparently not been noted. These bone changes must, nevertheless, be held to constitute still another link between the chloromas and the leukæmias, as the occurrence of osteosclerosis in association with myeloid leukæmia and rarely with lymphatic leukæmia (Assmann, 1907) is now well known.

There remains one other point for discussion—viz., whether the chloromas are to be classed with the leukosarcomatoses or with the true leukæmias. The former is the view upheld by Sternberg, who looks upon the infiltration or aggressive character of the growth in chloroma as essentially suggestive of the leukosarcomatoses. It has now become impossible, however, to draw any clear distinction on this ground in view of the fact (as stated by von Domarus, 1908) that cases of chloroma have occurred (e.g., case of Fabian, 1908) in which there has been no trace of malignancy, and, on the other hand, there have been cases of leukæmia in which marked infiltration of the neighbouring parts—e.g., periglandular tissue—has been noted as the result of careful histological examination.

Finally, there remains the question of ætiology, on which it must be confessed that the profound morphological researches, which have dominated hæmatology for many years, have thrown little light. The bacteriological questions involved in the acute leukæmias do not appear to have been seriously tackled, but certainly, in view of the frequent association of ulcerative processes about the mouth and throat with cases of acute leukæmia and chloroma, a wide field is open for bacteriological research, provided a sufficient quantity of suitable material is placed at the disposal of the investigator.

BIBLIOGRAPHY.

Assmann. *Beitr. zur path. Anat.*, Jena, 1907, xli, p. 565.
von Domarus. *Fol. Hæmat.*, Leipz., 1908, vi, p. 337.
Glinski. *Virchows Archiv*, Berl., 1903, clxxi, p. 101.
Johannson und Moritz. *Fol. Hæmat.*, 1908, vi, p. 243.
Lehndorff. *Fol. Hæmat.*, 1910, ix, p. 309.
Pappenheim. *Fol. Hæmat.*, passim and more particularly, 1909, vii, p. 439.
Treadgold. *Quart. Journ. Med.*, Oxford, 1908, i, p. 239.
Wynter. *Proc. Roy. Soc. Med.* (Child. Sect.), 1909, iii, p. 96.

The Experimental Production of the Carrier-State by Feeding.

By G. F. Petrie and R. A. O'Brien.[1]

It is somewhat striking that very little experimental work on the production of intestinal carriers by feeding has been reported, although the literature dealing with the subject of human intestinal carriers is already extensive and is rapidly growing. The only observations that have been recorded, so far as we are aware, are some published recently by Zwick and Weichel (1910), who carried out similar experiments to those we are about to describe.

We have succeeded in experimentally producing the carrier-state in guinea-pigs by feeding them with cultures of an organism of the "paratyphoid" group which is indistinguishable from *Bacillus aertryck* and *Bacillus suipestifer*. Our experiments were suggested by the results of an inquiry into a severe epizootic in a stock of guinea-pigs at the Lister Institute. In animals dying during the epizootic this organism was frequently isolated from the contents of the small intestine, from the organs and from the fæces before death. Moreover, a small number of the survivors of the epizootic were found to be excreting this bacillus in their fæces for several months, although apparently quite healthy. The strain isolated from the epizootic animals was highly pathogenic to guinea-pigs by subcutaneous inoculation, but of low pathogenicity when given in moderate doses by feeding. The details of the experiments we bring forward here are arranged in the tables.

It will be seen from Table I that six animals were fed with moderate doses—i.e., 5 cc. of a broth culture on two or three occasions. With the exception of one animal, which died eight days after the first feeding, the animals remained apparently healthy during the period of observation, and from the fæces the bacillus was isolated at intervals—in the case of two animals as long as sixty-three days after the first feeding. Some of the animals apparently showed an intermittent excretion.

Thirty-two days after the first feeding the agglutinin content of the sera of the five surviving animals was estimated, with the result that the sera of four animals agglutinated the bacillus in dilutions varying from 1 in 50 to 1 in 200. The serum of the fifth animal did not agglutinate in a dilution of 1 in 20, which, as will be seen from Table III, corresponds to the usual results obtained with normal guinea-pigs' sera. Two of the positive sera were found to agglutinate a strain of *Bacillus paratyphosus* (*B*) in a dilution of 1 in 1,000.

---

[1] From the Lister Institute, London.

In Table II are recorded further experiments with four animals which were fed on one occasion only, the bacillus being recovered from the fæces in each instance nine days later. The agglutination limit, determined on the forty-second day after feeding, was found to be 1 in 20 in the case of two of the animals, and 1 in 100 in the others. The agglutination figures noted in this table and in Table I are similar to those obtained in the spontaneous carriers above referred to. In order to discover, if possible, whether these agglutination results were related to some slight infection of the organs (spleen or liver) by the bacillus, four of the fed animals (see Table II) were killed and examined six days after the agglutination tests were made. In every case the organs appeared healthy, and the cultures from the contents of the small intestine, the spleen, the liver, and the heart-blood proved sterile.

These results raise the interesting question of the mechanism of agglutinin production in the apparent absence of even a mild infection. It is possible, of course, that a transitory invasion of the organs by the bacilli from the intestinal canal had occurred, and that the agglutinins were called forth in response thereto; but there is also the possibility that no such migration had taken place and that the agglutinins were evoked by a reaction on the part of the intestinal epithelium to the bacilli present in the intestinal contents. In this connexion it is known that killed cultures of bacteria given by the mouth may stimulate the production of specific agglutinin, but in a very irregular and inconstant fashion. It must be confessed that the whole question is at present obscure. It is noteworthy that Zwick and Weichel, in their experiments in which they produced healthy carriers by feeding mice on cultures of "paratyphoid" bacilli, obtained no evidence of the presence of specific agglutinins in the sera of their carrier animals. In other respects the experiments of these observers agree with those described above.

Our experiments may be summarized as follows:—

(1) Healthy intestinal carriers (guinea-pigs) of a bacillus of the "paratyphoid" group (*Bacillus aertryck or Bacillus suipestifer*) can readily be produced experimentally.

(2) Some of the carriers gave evidence of an intermittent excretion of the bacilli in the fæces.

(3) The agglutinin content of the carriers' sera was in excess of that of normal control animals, although, so far as the experiments go, there was no evidence of a somatic infection.

TABLE I.

No. of animal	First dose	1 day	2 days	4 days	5 days	11 days	30 days	Agglutination limit 32 days	43 days	63 days
1	1 broth tube 48 hours	+	Same dose		+	+	+	1 in 50	+	+
2	,,	+	,,	Same dose	+	+	+	1 in 100*	−	
3	,,	+	,,	,,	−	+	−	− 1 in 20	+	−
4	,,	+	,,	,,	Dead in 8 days; small intestine and liver +					
5	,,	+	,,	,,	+	−	+	1 in 200*	−	−
6	,,	+	,,	,,	−	−	−	1 in 100	−	+

+ = Bacillus recovered from fæces. * = 1 in 1,000 with *Bacillus paratyphosus* (*B*).

TABLE II.

No. of animal	Dose	Examination of fæces 9 days	Agglutination limit 42 days	Killed and examined 48 days; cultures from spleen, liver heart-blood, small intestine
1	¼ of 3 agar slopes	+	1 in 20	0
2	,,	+	1 in 20	0
3	,,	+	1 in 100	0
4	,,	+	1 in 100	0

TABLE III.

	Agglutination with normal guinea-pigs' serum
1	− 1 in 20
2	
3	
4	
5	
6	
7	+ 1 in 20 − 1 in 50

BIBLIOGRAPHY.

O'BRIEN, R. A. *Journ. of Hyg.*, 1910, x, p. 231.
PETRIE, G. F., and O'BRIEN, R. A. *Journ. of Hyg.*, 1910, x, p. 287.
ZWICK and WEICHEL. *Arb. a. d. Kaiserl. Gesundsheit.*, 1910, xxxiii, p. 250.

Experiments with Air-dried Mouse Cancer: (I) The Lethal Effect of Drying upon Mouse Cancer; and (II) The Inability of Air-dried Mouse Cancer, or of a Centrifugalized Extract, to produce Prophylactic or Curative Immunization.[1]

By S. G. Shattock and L. S. Dudgeon.

Although the inoculation of mice with dried mouse-carcinoma has already been carried out by Jensen (1903), and later by Bashford, and by Bridré (1908), the subject nevertheless appeared to us of sufficient importance to merit re-investigation, partly because of its bearing upon the theoretical possibility of the transference of carcinoma from one individual to another, partly because the possibility of using material, dried by simple exposure, in order to produce prophylactic or curative immunization, had not been inquired into.

I.—The Lethal Effect of Drying upon the Carcinoma Cell.

Amongst the factors reducing the chances of the transmission of carcinoma from one individual to another must be reckoned the lethal effect which drying has upon the cancer cell.

In the following experiments our purpose was to dry the carcinomatous material as débris might dry under natural circumstances—i.e., without the aid of a calcium-chloride or other dehydrating chamber, and at no higher temperature than that of the body. The particular tumour selected for the whole of the work recorded in the present communication is one which is in general use at the Imperial Cancer Research, and which is numbered as 63. The original of it arose "spontaneously" in the mamma of a mouse.

Experiment I.

July 23, 1909: The tumour used for drying had been raised by subcutaneous inoculation, carried out on July 5—i.e., it was of eighteen days' growth. The suprajacent skin was intact. The mouse was killed by decapitation, and the tumour removed after peeling off the skin. The whole of the procedures were in this, as in all other cases, carried out aseptically. The growth having been finely minced with curved scissors, the pulp was transferred to, and thinly spread over, the bottom of a Petri dish. The drying was carried out with the aid of Koch's plate-pouring apparatus, the water being at 37° C., and the apparatus being screened from sunlight to prevent the possibility of any deleterious action of light upon the cells.[1] The drying was allowed to proceed slowly, the lid of the Petri dish being partially raised. The material, when dried, was moistened with salt solution and remade into a pulp. Twelve mice were inoculated subcutaneously with a Bashford syringe, each animal receiving ·05 c.cm., the cannula being entered at the groin and carried as high as the axilla, and the material itself being slowly injected along the track of the cannula as the latter was withdrawn, according to the usual practice. Immediately before the material was moistened with the salt solution there remained in its centre *a small area*, no larger than the top of a pencil, which was *imperfectly dried;* this was spread to and fro over the dried portion around, yet a little of it was still sticky or moist when the salt solution was added and the pulp remade for injection. The drying was commenced about midday and the injections were carried out about 5 p.m.

---

[1] The material used for this investigation was obtained from the Imperial Cancer Research, where much of the work also was carried out; and we have to thank Dr. B. R. G. Russell for his ever-ready help in furnishing us with the tumours used; and Dr. E. F. Bashford, the Director, for imparting to us so freely the results of his experience in regard to many matters connected with the inquiry.

The animals were examined on August 4, and after that each week, the results being noted, and, when positive, graphically represented. This plan was adopted throughout the work, according to the general practice at the Imperial Cancer Research. The results are reproduced in the accompanying chart (*see* p. 75).

The chart shows that on August 4 two of the twelve mice presented a minute nodule of growth. On August 11 these growths had progressed and a third mouse presented a minute tumour. On August 23 the last-mentioned mouse died. On September 27 one of the other two positive results died, the growth being as large as a cherry. September 29: The only surviving positive result was now killed; the growth, at one time well developed, was in process of retrogression.

Experiment II.

December 8, 1909: The tumour used for drying was of twenty-eight days' growth. It was treated as already described, except that the lid of the Petri dish was completely removed and the material was repeatedly spread with a platinum loop in order to hasten the drying, which occupied about two hours, and was in this case complete throughout. The material was moistened with salt solution, remade into a pulp, and ·05 c.cm. injected subcutaneously into each of seventeen mice.

---

[1] The lethal action of sunlight upon carcinoma cells has already been demonstrated.

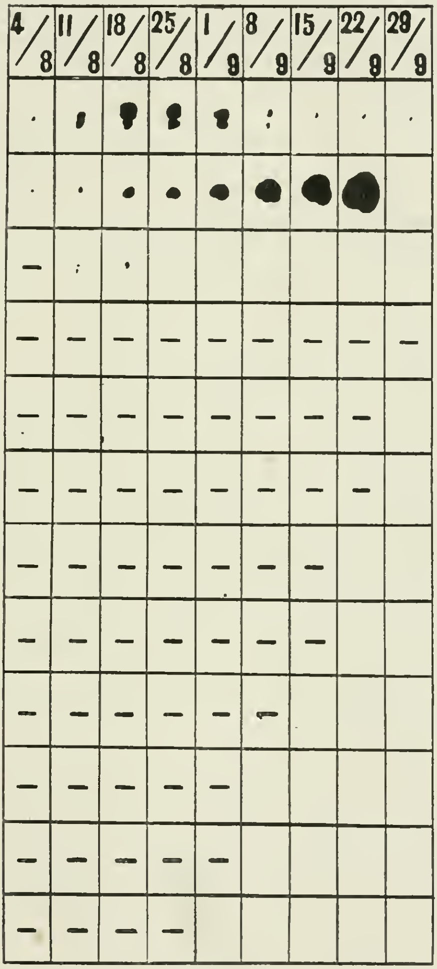


CHART I.

Showing the results of the inoculation of twelve mice with dried mouse-cancer. A small area remained incompletely dried; this explains the growth which occurred in the three mice at the top of the chart. The history of the animals reads horizontally from left to right. The dates of the examinations are given at the head of each column, beginning with August 4 and ending with September 29.

January 13, 1910 (thirty-six days after inoculation): No growth had taken place during this period in any of the mice. To-day twelve of these seventeen mice were inoculated (in a different position) with living carcinoma pulp to ascertain whether any prophylactic immunization had resulted from the insertion of the dried carcinoma; the other five were kept as controls.

February 4: One of the five controls died without having shown any sign of new growth.

February 28: A second of the five controls died without having shown any sign of growth.

March 8 (90 days after inoculation): The three remaining controls, having shown no growth, were now killed. Of the other twelve not one showed, at this date, any growth at the site of the insertion of the dried carcinoma, nor did they at any subsequent period. These twelve mice are again referred to under "Prophylactic Immunization" (III).

Experiment III.

February 14, 1910: The tumour used for drying was of sixteen days' growth. The drying was completed in one and a quarter hours. Twenty-four mice received, each, ·05 c.cm. of the dried material after it had been remade into a pulp with salt solution, the material being inserted subcutaneously along the left side of the thorax and abdomen.

February 28 (fourteen days after inoculation): No growth had appeared in any of the twenty-four mice. Twelve were to-day injected subcutaneously in another position with living carcinoma pulp to ascertain whether any prophylactic immunity had been established, and twelve were kept as controls.

March 21 (thirty-five days after inoculation): No growth had appeared at any time in the twelve controls, and they were killed to-day. Nor did growth occur at any time later, in the other twelve, at the site of the insertion of the dried material. These twelve mice are again referred to under "Prophylactic Immunization" (III).

Summary of the Results obtained by the Insertion of Dried Carcinoma.

In two of the three sets of experiments no tumours were raised from the dried material. In the third set, out of twelve mice inoculated, growths appeared in three. The latter result, however, in place of being at variance with the others, strikingly confirms them. For in this instance a small area of the pulp remained incompletely dried when the whole was moistened with salt solution and remade into a pulp for the purpose of the inoculation; and to the comparatively few cells which in this case remained unkilled by the drying the positive results are attributable.

In confirmation of these results we may add that in the series of experiments to be presently detailed, in which carcinoma pulp, similarly dried and remade with salt solution, was inserted subcutanously in order to test its possible prophylactic and curative effects, no growth ever arose from the material so treated.

II.—Attempts to transmit Mouse-carcinoma by means of Cancer Spray.

Substituting the living carcinoma cell for a bacterial micro-organism, could the cell be conveyed to the air-passages it is theoretically possible that it might gain a foothold and induce malignant disease. The following experiments were carried out in the endeavour to realize this theoretical possibility:—

The fluid for the spray was prepared in the following manner: The tumour was removed and finely minced with scissors in the usual way; salt solution was then added, and the material, after being well agitated, was at once passed through sterile muslin, transferred to a small test tube fitted with a rubber cork and bellows, &c., and used without delay. That such spray contains carcinoma cells we had by previous observation ascertained by spraying over a series of slides, allowing the films to dry, and staining them with carbol thionine. On such slides abundant groups of carcinoma cells are demonstrable. The mice were, one by one, held by the skin of the neck and back, turned over, and the nares kept closed by compression. The cancer spray was then forcibly driven into the mouth as soon as the latter was widely opened by inspiration. The tumours used were sub-cultures of No. 63, the original of which was a "spontaneous" mammary carcinoma, and they were taken at a stage of active growth. This particular tumour was chosen by reason of the extreme readiness with which it is inoculable upon other animals. The spray was administered on July 22, August 2, and October 7, 1910. Minute examination of the lungs showed no trace of disease. These experiments we are at the present time repeating.

Looking at the problem of air-borne carcinoma from the clinical side, there is little evidence that primary disease of the lung—e.g., in general—is due to such a mode of infection. The lethal effect of drying on the carcinoma cell makes the question at the outset altogether different from the common air-borne tuberculous infection of the same organ. Primary carcinoma of the lung is, speaking broadly, one of the rarest of lesions met with in morbid anatomy. We have not ourselves ever seen an example of it. In those that have been recorded, moreover, the possible occurrence of an antecedent and predisposing cirrhosis or bronchitis (some of the cases being of bronchial origin) must not be lost sight of, and this would reduce the air-borne contingency to a still finer margin; and, finally, the remoteness of such a possibility is emphasized by the fact that direct auto-infection of the lungs by way of the air-passages—in cases of carcinoma of the larynx, pharynx, tongue, or lip, where everything might be supposed favourable to its occurrence—is an event hardly known. Nevertheless, it is a remarkable fact, as Osler states,[1] that the workers in the Schneeberg cobalt mines are very liable to primary cancer of the lung; it is said that in this region a considerable proportion of all deaths in persons over forty are due to this cause. Here the pneumonokoniosis doubtless prepares the soil for the other disease, whether the latter is of endogenous or exogenous source.[2] The frequency of primary carcinoma of the *larynx* can be satisfactorily explained without having recourse to an air-borne theory of infection.

How far carcinoma may be conveyed from one individual to another is, nevertheless, a subject that still merits further investigation. Its contagiousness and its infectiousness, using the terms in their usual clinical meanings, are theoretical possibilities indicated by cancer houses, cancer villages, and cancer districts, and, possibly, cancer mines. One strong argument against the indications afforded by cancer houses is undoubtedly the fact that the histological form of the disease in the different individuals concerned is by no means always the same, and that the primary disease is not confined to the skin and mucous membranes (to which alone a graft would be accessible), but may affect such an organ, e.g., as the breast. The substitution of a sarcomatous growth, produced from the connective tissue of the host, in the place of a carcinomatous graft (an occurrence now so well established in the case of certain forms of mouse-carcinoma) indicates, nevertheless, the possibility that a parallel phenomenon *might* take place, were a carcinomatous graft implanted upon an epithelial surface furnished with an epithelium different in kind from that of the graft itself. The access of a graft, e.g., from a squamous-celled carcinoma, *might* induce the growth of a columnar-celled tumour from the tissues of the host, should the graft alight upon a surface furnished with an epithelium of the latter variety.

---

[1] "Medicine," 6th ed., p. 642.

[2] According to Wagner and others the disease in question is a lymphosarcomatosis of the mediastinal and bronchial glands, involving the lung (Borst: "Lehre von Geschwülsten," Wiesbaden, 1902).

Some of the discoveries, now so well known, in regard to the vitality of the carcinoma cell (in the mouse) go far to support the possibility of a transference of the disease from one individual to a second. When we recall the fact that mouse carcinoma may be successfully grafted into other mice as long as three weeks after the tumour itself has been removed from the body and kept (without being allowed to dry) in an ice-safe, and that tumours may be raised from the carcinomatous tissue after it has been frozen in liquid air, the theoretical possibility of the conveyance of carcinoma from one individual to another by means of débris derived from ulcerating and necrotic growths of external parts, or expectorated or otherwise voided from the mouth, in carcinomatous disease of the tongue, or lip, or larynx, will hardly be denied. Such débris might, theoretically, produce disease from any of the sites which obtain in the case of bacterial infections, the *contagium vivum* being the carcinoma cell in place of the bacterium. The living cancer cell might be implanted upon some part of the cutaneous system, or of the alimentary, or the respiratory, or the genito-urinary. Its indirect transference from the exterior to the lymphatic glands, without the production of a carcinomatous lesion at the site of entrance (an occurrence so well recognized in tuberculous infections), is scarcely conceivable, those cases of squamous-celled carcinoma of the inguinal lymphatic glands without recognizable cutaneous lesion, observed in chimney-sweepers, being more readily explained by the obsolescence of an unnoticed primary cutaneous lesion.[1]

Nevertheless, if the conveyance of carcinoma ever takes place from one individual to another, it must be very exceptional. And it would probably require the concurrence of a highly susceptible condition in the recipient, and a peculiarly resistent or "virulent" property in the carcinoma transferred. That these collateral factors would play a part in such a transference is clear from the diversity of results attending the subcutaneous grafting of mouse-carcinoma, for the success attending the grafting of different tumours of "spontaneous" origin varies widely; in the case of some it is altogether negative, and in the case of the rare tumours which are inoculable with the highest success a certain number of mice prove refractory—i.e., the graft retrogresses after having commenced to grow.

---

[1] Sir James Paget, "Surgical Pathology" (one case); Mr. George Lawson, *Lancet*, 1878, ii., p. 576 (two cases); Mr. H. T. Butlin refers to these several cases in his lectures on "Cancer of the Scrotum," *Brit. Med. Journ.*, 1892, i, p. 1341.

III.—THE SUBCUTANEOUS INJECTION OF AIR-DRIED CARCINOMA PRODUCES NO PROPHYLACTIC IMMUNIZATION TO THE GROWTH OF A SUBSEQUENT GRAFT.

Four sets of experiments were carried out to test the possible production of a prophylactic immunization by the subcutaneous insertion of carcinoma pulp, dried, as already detailed, by simple exposure, in a subdued light, at 37° C. In three of these sets a single insertion of the dried material was made previously to the inoculation with the living tumour; in the fourth two insertions were made, with an interval of fourteen days between them.

Experiment I.

December 8, 1909: Seventeen mice received each, subcutaneously, 0·05 c.cm. of dried carcinoma, remade into a pulp with salt solution. The tumour used was of twenty-eight days' growth, and is the same as that referred to under the second experiment, made to test the vitality of the dried material.

January 13 (thirty-six days after inoculation): Twelve of these seventeen mice were inoculated subcutaneously, each with 0·05 c.cm. of living pulp prepared from a tumour of twenty-one days' growth, the inoculation being made at another site than that of the first insertion—viz., the opposite side of the thorax and abdomen. The remaining five were kept as controls, and in these no growth at any time took place.

January 24: Small growths had appeared in eleven.

February 28: In five retrogression and disappearance of minute growths had occurred. In four there was a growth the size of a cherry; in one the tumour was twice this size; one died on February 23, with the largest growth of all, the size of a plover's egg.

March 7: The tumour was still progressing, and the experiment was closed. No growth at any time appeared at the site of insertion of the dried carcinoma.

Experiment II.

March 22, 1910: Twelve mice were inoculated, subcutaneously, with the dried and remade pulp of a tumour of twenty-one days' growth. The drying of the tumour was completed in two hours. Each received 0·05 c.cm. beneath the skin of the thorax and abdomen.

April 6: Each animal received 0·05 c.cm. of living pulp beneath the skin of the back.

June 24: In five no growth had appeared; in five inconsiderable growths appeared which retrogressed and vanished. In two the tumours progressed and were of large size.

The animals were killed on July 4.

Experiment III.

February 14, 1910: Twelve mice received 0·05 c.cm. of dried tumour remade into a pulp, subcutaneously, on the left side of the thorax and abdomen. The tumour was of sixteen days' growth.

February 28: Each received 0·05 c.cm. of living pulp of a tumour of twenty-four days' growth, the insertion being made in the right side of the thorax and abdomen.

March 16: One had shown no growth; one had died with a minute growth on March 3; in three the tumours were quite small; in seven they were well declared.

Experiment IV.

May 6, 1910: Twelve mice were inoculated beneath the skin of the back with dried carcinoma remade into a pulp as usual; each received, beneath the skin of the back, 0·05 c.cm. The drying of the pulp occupied three-quarters of an hour; the tumour was of thirteen days' growth.

May 20: A second injection was made from a tumour of fourteen days' growth, dried and made into a pulp; the drying was completed in three-quarters of an hour. Beneath the skin of the back, each of the same twelve mice received 0·05 c.cm.

June 3: Each mouse received 0·05 c.cm. of living carcinoma pulp beneath the skin of the thorax and abdomen; fourteen days, i.e., after the second insertion of the dried material.

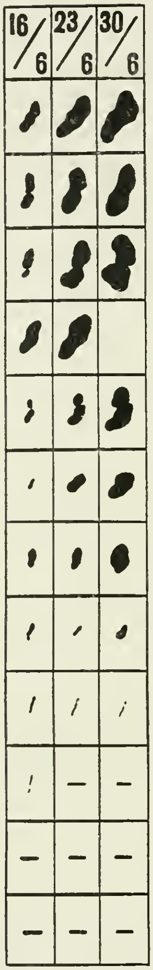


CHART II.

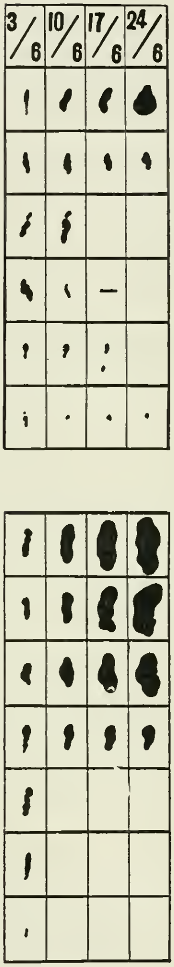


CHART III.

Chart II.—Showing the growths arising in twelve mice after inoculation of living cancer pulp, carried out fourteen days after the second of two previous subcutaneous insertions of dried carcinoma. No prophylaxis has been induced. The history of each animal reads from left to right; the first charting was carried out on June 16, and the last on June 30.

Chart III (referred to under Section V).—Showing that no prophylactic immunization is produced by centrifugalized extract of mouse cancer. The upper of the figures gives the course of growth of the grafts in six untreated mice. The lower gives that of the grafts, made at the same date, after the previous injection of centrifugalized extract of carcinoma. It will be apparent that the growth, in place of being retarded, has been accelerated by the previous injection.

June 30: In two no growth had appeared; in two a minute linear growth appeared which shortly afterwards vanished. In eight, progressing tumours of varying sizes followed; in one of these eight the growth was only of the dimensions of a large pea; in the other seven the tumours were of pronounced size—the largest, which was bilobed, was the size of two cherries.

The animals were killed a fortnight later. (Chart II.)

Summary of the Foregoing Results.

It is clear from these observations that the subcutaneous insertion of carcinoma cells killed by the simple process of drying in the air at 37° C. produces no prophylactic immunization. In three sets of experiments a single insertion of the dried material was made; in a fourth two insertions were made at an interval of fourteen days. The living cancer pulp was inserted, in one set, thirty-six days later, in another set fifteen days later, and in the two other sets it was inserted fourteen days later. These results accord with the others obtained at the Imperial Cancer Research, which show that no prophylaxis is producible by the insertion of dead carcinoma cells, however the death has been brought about, whether by the action of radium or by other means. In Experiment I some delay in the appearance of the growths after grafting occurred, as compared with what took place in a series of mice inoculated by Dr. Russell from a "sister" tumour to that used by us on January 13 (by a "sister" tumour is meant one raised in another mouse from the same cancer pulp as that used to raise the growth in the animal used for the experiment carried out on January 13). Delay in the appearance of the tumours after grafting, however, is a well-recognized phenomenon at the Imperial Cancer Research, and no significance can be attached to it in the present instance, especially as it was not observed in the other sets of similar experiments.

IV.—THE SUBCUTANEOUS INSERTION OF AIR-DRIED CARCINOMA PRODUCES NO CURATIVE IMMUNIZATION TO THE GROWTH OF A GRAFT.

In the two following sets of experiments the dried material was inserted after a growth had been raised by the subcutaneous inoculation of living carcinoma pulp, in order to see whether any curative retrogression of the tumours would result. The preparation of the dried material was carried out in the way already detailed.

Experiment I.

March 22, 1910: Four mice in which progressing tumours had been raised by inoculation on February 14, 1910, with a growth of sixteen days, received 0·05 c.cm. of dried carcinoma remade into a pulp. The first series of injections, with the living material, was made beneath the skin of the left side of the chest and abdomen; the second series, with the dried, was made beneath the skin of the right side. The tumours steadily progressed.

The experiment was closed on April 15.

Experiment II.

April 12, 1910: Five mice in which progressing tumours had been raised by the subcutaneous insertion of living carcinoma pulp on March 23 (from a growth of twenty-two days) received 0·05 c.cm. of dried carcinoma remade into a pulp. The first insertions were made on the left side, the second beneath the skin of the back. The tumour dried was of fourteen days' growth; drying was completed in one and three-quarter hours.

April 21: The five mice again received 0·05 c.cm. of dried and remade pulp, beneath the skin of the back; the tumour dried was of fifteen days' growth.

April 28: The five mice again received (third time) 0·05 c.cm. of dried and remade carcinoma pulp beneath the skin of the back. The drying of the tumour was completed in one and a half hours.

May 1: One mouse died, the tumour still increasing.

May 5: One mouse died, the tumour still increasing.

May 6: The other three mice were killed and the experiment closed. The tumours had steadily progressed and were of considerable size.

Summary.

It will appear from these data that no curative immunization to the growth of the graft was brought about by the subcutaneous insertion of dried cancer, and this notwithstanding the fact that in the second set of experiments three insertions of the dried material were made—viz., on April 12, April 21, and April 28, after the growth was declared.

V.—A CENTRIFUGALIZED EXTRACT OF MOUSE-CANCER PRODUCES NO PROPHYLACTIC IMMUNIZATION TO THE GROWTH OF A GRAFT.

In the preparation of the extract the method we first attempted was to filter the ground tumour through porcelain in order to remove entire cells and cell-débris. The filtration, however, occurred so slowly that the simpler method of clearing the suspension by centrifugalization was resorted to.

May 10, 1909: Four mice with tumours of twenty days' growth were decapitated. The tumours were removed and ground with a sprinkle of powdered and sterilized glass for an hour. Salt solution was then added and filtration through porcelain commenced. Only 2 c.cm. of fluid had come through in two and a half hours; no use was made of this. The rest of the unfiltered material was centrifugalized at high speed, three times in succession, the fluid being pipetted off after each centrifugalization into another series of tubes. The fluid finally obtained was quite turbid, with a thin whitish surface layer. A microscopic examination ($\frac{1}{12}$ oil immersion) showed the presence of myriads of fine fat droplets, but no cells. Four mice (A) received 0·5 c.cm. of the fluid beneath the skin of the back.

May 19: These four mice (A) were inoculated with 0·05 c.cm. of living cancer pulp prepared from a tumour of twenty-nine days' growth. Six control mice (C) received the same living cancer pulp.

May 29: Batch A—Tumours had grown in all. Batch C—Tumours had appeared; though taken all together the bulk of these was somewhat less than in A.

June 5: Batch A—Tumours progressing; they are obviously larger than those in the control series. Batch C—Tumours of fair size.

June 19: Batch A—One had died June 12 with a large progressing tumour. In two the growths were of large size and increasing; in the fourth mouse the tumour, one of moderate size, was retrogressing. Batch C—In two the tumours were of considerable size; in four they had retrogressed and disappeared. All the survivors were now killed.

The history showed some acceleration of growth in the mice treated with the extract; though when the animals were killed some of the tumours of the controls were as large as the others.

May 13, 1909: An extract was prepared by grinding four tumours, of twenty-three days' growth, adding salt solution, and centrifugalizing the fluid in order to get rid of the débris, as already detailed. Seven mice received each 0·5 c.cm. of the extract beneath the skin of the back (Batch B).

May 24: Batch B.—Each of the seven mice received 0·05 c.cm., subcutaneously, of living carcinoma pulp prepared from a tumour of thirty-four days' growth, the necrotic area being scraped away. Batch D.—Six mice were inoculated with the same living pulp to serve as controls.

June 3: Batch B.—Tumours had commenced to grow in all. Batch D.—Tumours had appeared in the controls. Taken generally, these were slightly less voluminous than those in Batch B.

Of Batch B one died on June 11, another on June 16, another on June 20; of Batch D one died on June 8.

June 25: All were killed to-day.

Of Batch B one mouse grew a minute linear tumour, which retrogressed and disappeared. In both batches the tumours, when the animals were killed, were of considerable dimensions, yet the growth had been accelerated by the injection of the extract, and at the date of death the general bulk of the tumours in the treated mice exceeded that of those in the others. No trace of new growth appeared at the site of the injected extract in these or any other of the experiments (*see* Chart III, p. 82). A similar hypersensitization was observed by Dr. Haaland[1] as a result of the injection of an extract obtained from material ground in a mortar with sand, and submitted to the action of a Buchner's press. The hypersensitization was induced by the fluid expressed not only from mouse-carcinomata, but, also, from the normal tissues of mice.

---

[1] *Proc. Roy. Soc.*, B, 1910, xxxii; also *Lancet*, 1910, i, p. 787; "The Contrast in the Reactions to the Implantation of Cancer after the Inoculation of Living and Mechanically disintegrated Cells" (Imperial Cancer Research).

Pathological Section.

January 17, 1911.

Dr. F. W. Mott, F.R.S., President of the Section, in the Chair.

The Incidence of Gall-stones and of Primary Carcinoma of the Gall-bladder and Biliary Passages in the Insane.

By J. P. Candler.[1]

The following paper is based upon the investigation of 2,228 autopsies conducted at the London County Asylum, Claybury, during the past ten years. The main points to which attention has been directed have been the investigation of the incidence of gall-stones, and the prevalence of primary carcinoma of the gall-bladder and biliary passages in the series of cases examined. The investigation was commenced at the suggestion of Dr. Mott who, while aware of the frequency with which gall-stones are found on the post-mortem table at Claybury, could not recall to mind meeting with a case of primary carcinoma of the gall-bladder. By reason, therefore, of the importance which is assigned to gall-stones in the causation of primary carcinoma of this organ, he considered that an investigation on these lines might yield some points of interest.

The Incidence of Gall-stones.—The total number of autopsies investigated was 2,228 ; of these 1,169 were females, and 1,059 males. Gall-stones were found in 315, a percentage for both sexes of 14·13. Of the 1,169 females there were 210 cases with gall-stones (17·85 per cent.), and of the 1,059 males, 105 cases (9·91 per cent.).

---

[1] From the Pathological Laboratory of the London County Asylums.

Reference to Hospital Statistics.—Dr. Hale White estimates from post-mortem records on Europeans of all ages, and of both sexes that gall-stones are present in from 5 to 10 per cent. Rolleston estimates the percentage incidence in post-mortem examinations to be between 6 and 10. Schroeder has found in Strasburg a percentage incidence of ·12 for both sexes. In his cases the proportion of female cases was 20 per cent., against 4·4 per cent. of males, an incidence for females in considerable excess of that obtained at Claybury.

Dr. Ticehurst [1] gives the following table on the incidence of gall-stones taken from the papers of Naunyn and Kelynack :—

Table I.

Observer	Place of observation	Percentage incidence of gall-stones
Poulson	Copenhagen	3·8
Peters	Kiel	5·0
Munk Rother Hiller	Munich (10 years)	6·2
Brockbank	Manchester (487 cases) ...	6·5
Fielder	Dresden	7·0
Schloth	Erlanger (24 years)	7·2
Voelcker	286 cases	9·4
Roth	Basle	10·0
Frank	Vienna	10·0
Schroeder	Strasburg (7 years)	12·0

Ticehurst, in his investigation based upon the post-mortem records of Guy's Hospital for twenty-five years (1876-1900), found 333 cases of gall-stones in 11,031 autopsies, a percentage of 2·08, or excluding patients dying under 20 years of age, 6·3 per cent.

From the figures above given it would appear that the incidence of gall-stones among the insane was greater than that found in ordinary hospital practice, to which fact attention has already been drawn by Dr. Beadles, who at Colney Hatch Asylum found gall-stones present in 27 per cent. of the female cases, and in 5 per cent. of the males. Beadles quotes Warnock's figures of 50 per cent. in females, and 11 per cent. in males dying in Peckham House Asylum (Private), to show that this excess is by no means confined to pauper lunatics.

The figures from Claybury appear to bear out this contention, but not so markedly as those of Beadles and Warnock; nevertheless, it is probable, from the evidence obtainable, that the percentage incidence of gall-stones is slightly higher among the insane than among the general population. The greater incidence at Colney Hatch may be due to the fact that it is the second oldest London County Asylum, and probably the average age of the inmates is higher than that of Claybury, which is comparatively a new asylum. Reference to Table II, compiled from the Claybury statistics, shows that, with the exception of the age-period 40—50 years, there is a gradual increase in the percentage incidence of gall-stones as age advances, and this percentage agrees fairly accurately with that given by Schroeder.

---

[1] "On the Mortality from Gall-stones." Thesis for M.B. Degree, Cantab. 1903.

TABLE II.

Age-periods	Total number of autopsies	Number of cases with gall-stones	Percentage	Schroeder's percentage
0 to 20	16	0	0	2·4
20 ,, 30	155	6	3·87	3·2
30 ,, 40	375	31	8·26	11·5
40 ,, 50	424	27	6·36	11·1
50 ,, 60	412	54	13·1	9·9
60 ,, 70	440	85	19·31	—
70 ,, 80	337	90	26·70	—
80	69	22	31·83	—
60 and over	846	197	23·28	25·2

The figures which I have recorded give the incidence of the gall-stones as regard hospital patients and the insane. It is of importance to compare the statistics afforded by these two classes, and to ascertain how far the figures can be used as an indication of the incidence of these affections among a general population.

The preceding tables show that the incidence of gall-stones among hospital patients varies considerably in the hands of different observers. No satisfactory conclusions can be given to account for these variations from a mere perusal of the figures, unless it be known that the statistics have been compiled under precisely similar conditions, especially as regards the number of cases dying at the various age-periods, the percentage incidence of gall-stones at these age-periods, and the number of cases dying before the age of 20. This last factor is of the utmost importance, for since gall-stones are exceedingly rare in cases dying before the age of 20, the inclusion of a large number of such cases before age 20 will materially affect the tables. This is well shown by the figures of Ticehurst—the percentage incidence of gall-stones on 11,031 autopsies was only 2·08; of these no less than 3,156 were under 20 years of age, and only one case with gall-stones was found amongst this number. The elimination of all cases dying before 20 increased the percentage incidence to 6·3.

The percentage incidence of gall-stones at Claybury Asylum was 14·13 on 2,228 autopsies. The number of cases dying under 20 years of age was only 16, and in none of these cases were gall-stones found.

There is a considerable difference between the incidence of gall-stones among the insane (as shown by the Claybury statistics and elsewhere) and that among hospital cases. Even after the elimination of all cases dying before 20 years of age, there is still a great difference between the Claybury figures and those from Guy's Hospital. The following table shows the total number of gall-stones found at the different age-periods at the two institutions :—

TABLE III.

Age-period	Number of gall-stones Guy's Hospital	 Claybury Asylum
10 to 20	1	0
21 ,, 30	11	6
31 ,, 40	36	31
41 ,, 50	85	27
51 ,, 60	91	54
61 and over	107	197

This table shows that out of an approximately equal number of cases with gall-stones, the Claybury figures provide a smaller number at age-periods 40—50 and 50—60, and a considerably greater number at 60 years and over, than at Guy's. I have been unable to obtain the number of cases dying at the different age-periods at Guy's for comparison with the Claybury figures, and, in the absence of these, I hesitate to form any definite conclusions. As, however, the incidence of gall-stones increases with advancing age, the inference is that a relatively greater number of deaths occurred at Guy's Hospital between the ages 40—60 and less from 60 onwards than occurred at Claybury Asylum.

Mayo Robson has suggested that the incidence of gall-stones among hospital patients is less than that in a general population because hospitals are patronized by the labouring classes, in whom the incidence of gall-stones is less. This may be so, but I am inclined to think that there is another explanation which may account for any lessened incidence of gall-stones in the hospital statistics, namely, that a relatively small number of the aged poor seek hospital assistance, but die from old age in their own beds or in the infirmaries, and thus fail to provide the hospitals with the necessary material for complete statistics.

Statistics obtained from asylums (at any rate as far as the relative incidence of gall-stones is concerned) would be more comparable with those obtained from a general population from which all cases dying before the age of 20 have been eliminated, by virtue of the fact that the population of asylums is not constantly changing as is that of hospitals. The patients are admitted solely for their mental condition, irrespective of any bodily ailments, and many of them on admission are in good health and ultimately die of senile decay after many years of residence in the asylum. They therefore provide a greater number of people in the later years of life than do hospitals. In the Claybury statistics, out of 2,228 autopsies, 1,258, or 56 per cent., had survived the age of 60; 406, or 18 per cent., survived the age of 70; and 69, or 3 per cent., survived the age of 80.

The death-rate in asylums differs from that in a general population in that there is a greater mortality in the mid-period of life from phthisis, general paralysis of the insane, pneumonia, and dysentery. The average duration of life, therefore, among the insane is probably shorter than among the general population who have survived the age of 20: and this should tend to *diminish* the relative incidence of gall-stones among the insane. But, in spite of this, the percentage incidence in the insane is estimated to be *higher* than in a general population. Various explanations have been put forward to account for this increased incidence in the insane, such as the greater prevalence of gastric disorders, of constipation, of melancholia and lethargic states, and of the breaking down of nervous tissue with a greater deposition of cholesterol in the bodily tissues. Thudichum, commenting upon the paper of Beadles, regarded the high percentage of persons dying in asylums with gall-stones as being evidence of some causal relationship to cerebral decay and insanity. No doubt this great authority on the chemistry of the brain considered the brain decay and resulting cholesterol production accountable for the higher incidence of gall-stones among the insane than among the general population.

The Incidence of Primary Carcinoma of the Gall-bladder and Biliary Passages as ascertained at Guy's Hospital.—Ticehurst found in 11,031 autopsies, with 333 cases of gall-stones, that there were 45 cases of primary carcinoma of the gall-bladder (13·5 per cent.), and 15 cases of primary carcinoma of the bile-ducts (4·5 per cent.), or, combining the two, a total of 60 cases of primary carcinoma of the gall-bladder and biliary passages (18 per cent.).

The Incidence of Primary Carcinoma of the Gall-bladder and Biliary Passages in the Insane.—Of the 2,228 cases examined at Claybury 315 were found to have gall-stones. In this series there were only two definite cases of primary carcinoma of the gall-bladder (one male and one female), gall-stones being found in the gall-bladder in each case. In other words, the incidence of primary carcinoma of the gall-bladder was 1 in 1,000 of the insane dying at Claybury during ten years; or, looked at from the percentage incidence of gall-stones, two cases of primary carcinoma of the gall-bladder out of 315 cases of gall-stones (0·65 per cent.). Both these cases were proved by microscopic examination. In addition to these two cases there was one case which occurred at the commencement of the series which was noted as (?) primary carcinoma of the gall-bladder, or primary malignant disease of the breast with secondary involvement of the liver and gall-bladder. The right breast had been previously amputated, and there was a malignant mass in the site of the scar. The liver was studded with malignant nodules. The gall-bladder was involved in a mass of cancer and contained one large stone the size of a pigeon's egg. The case was not microscoped. Even if this be conceded as a case of primary malignant disease of the gall-bladder, which is very doubtful, the percentage incidence is only 0·95.

Of primary carcinoma of the liver and biliary ducts apart from the gall-bladder there were only three very doubtful cases occurring very early in the series. In Case I, the growth in the liver was probably secondary to malignant disease of the ovary. In Case II it was probably secondary to carcinoma originating in the pylorus; and in Case III it was noted as probably secondary to primary malignant disease of the mediastinum. The gall-bladder was quite free from growth in each case, and there were no stones found either in the ducts or gall-bladder, nor any evidence of partial or complete destruction of the ducts by cicatricial contraction, consequent upon the previous passage of gall-stones along the ducts. These cases were not microscoped. Even if they be included as possible instances of primary carcinoma of the bile-ducts consequent upon irritation of stones which had ultimately been passed into the intestine, the percentage incidence in relation to gall-stones is only 0·95.

It will thus be seen (*vide* Table IV) that the percentage incidence of primary carcinoma of the gall-bladder and biliary passages in the insane, as afforded by the Claybury figures, is markedly less than the hospital percentage incidence. As the incidence of these diseases among a general population is based upon hospital statistics, such a wide variation between the Guy's Hospital figures and those of Claybury requires some comment, especially as gall-stones are considered to play so important a part in the production of primary carcinoma of the gall-bladder and biliary passages.

Opinions as to the Rôle played by Gall-stones in the Causation of Primary Malignant Disease of the Gall-bladder.—The opinion of those who have studied this subject is almost entirely in agreement—namely, that gall-stones are a cause of primary malignant disease of the gall-bladder and ducts, especially of the former.

Dr. Hale White, quoting from Ticehurst's figures, states that the latter found among the 11,031 autopsies at Guy's Hospital (in which there were 333 cases of gall-stones) 45 examples of primary carcinoma of the gall-bladder, and in 43 of these, or 95 per cent., gall-stones were present; and there were 15 cases of primary carcinoma of the bile-ducts, and gall-stones were present in 11, or 75 per cent. Hale White concludes that about 20 per cent. of those who have gall-stones subsequently suffer from malignant disease of the biliary passages. He further emphasizes that this is not as widely understood as it should be, the chief reason being that most cases of malignant disease of the biliary passages are called malignant disease of the liver, and hence the causative importance of the gall-stones is missed.

Table IV.—Showing a Comparison of the Incidence of Gall-stones and Primary Carcinoma of the Gall-bladder and Biliary Passages in Cases dying at Claybury Asylum and Guy's Hospital.

	Total No. of autopsies	Total No. with gall-stones	Percentage incidence of gall-stones	No. of autopsies over 20	Percentage incidence of gall-stones over 20	No. of cases of primary carcinoma of gall-bladder	Percentage	No. of cases of primary carcinoma of bile-ducts	Percentage	No. of cases of primary carcinoma of gall-bladder and bile-ducts	Percentage
Guy's Hospital	11,031	333	2·08	7,875	6·3	45	13·5	15	4·5	60	18·0
Claybury Asylum	2,228	315	14·13	2,212	14·24	2 1 ?	0·65 0·95 ?	0 3 ?	0 0·95 ?	2 4 ?	0·65 1·9 ?

Moynihan states that one of the most serious sequelæ of cholelithiasis is malignant disease of the gall-bladder or ducts, and that the close connexion between gall-stones and malignant disease has never lacked recognition.

Mayo Robson, on the other hand, states that cancer of the gall-bladder is by no means frequent, and as a primary affection is somewhat rare. It is usually due to the irritation of gall-stones or to extension from adjoining organs.

Rolleston writes that special interest is attached to the association of gall-stones and carcinoma of the gall-bladder—Musser, 69 per cent.; Courvoisier, 91 per cent.; Siegert, 95 per cent.; Fütterer, 70 per cent.; Winton, 81 per cent.; Zenker, 85 per cent. Conversely, it appears that primary carcinoma of the gall-bladder occurs in from 14 to 4 per cent. of all cases of cholelithiasis.

Schroeder estimated that 14 per cent. of persons with cholelithiasis eventually became the subjects of carcinoma of the gall-bladder.

In 149 cases of gall-stones, abstracted from the records of Guy's Hospital by Keay, there were 17 cases of carcinoma of the gall-bladder or cystic duct, equivalent to 11·4 per cent.

Riedel estimates the percentage of primary carcinoma in cholelithiasis to be from 7 to 8 per cent.

Among 242 cases of gall-stones at St. George's Hospital there were 10 cases of primary carcinoma of the gall-bladder, or 4·1 per cent.

Rolleston sums up by saying that "gall-stones are present in the great majority of cases of primary malignant disease of the gall-bladder, while carcinoma develops in 14 to 4 per cent. of cases of cholelithiasis."

The evidence, therefore, shows that gall-stones are present in practically 80 to 90 per cent. of all cases dying from primary carcinoma of the gall-bladder, while it has been remarked by Ticehurst and others that gall-stones are rarely found when the biliary passages are affected by growth secondary to that elsewhere. So far as the Claybury statistics show they confirm Ticehurst's statement. Both cases of primary carcinoma of the gall-bladder contained calculi, while cases in which the liver was affected with growth secondary to malignant disease commencing in some other organ showed little or no excess in the incidence of gall-stones.

The point, however, to which I desire to draw attention, is as to whether the association between gall-stones and primary malignant disease of the gall-bladder is so frequent as to justify the opinion that because calculi are found in 90 per cent. of cases of primary carcinoma of the gall-bladder they are to be looked upon as so potent a factor in the causation of this disease. On the one hand we have statements to show that no less than 20 to 18 per cent. (Hale White and Ticehurst) and 14 to 4 per cent. (Rolleston) of cases of cholelithiasis succumb to malignant disease of the gall-bladder and ducts. On the other hand, Mayo Robson states that cancer of the gall-bladder is by no means frequent, and as a primary affection is somewhat rare. These two statements are diametrically opposed. The estimates of Hale White, Ticehurst, and Rolleston are based on hospital statistics. They cannot, in my opinion, be taken to represent the incidence among the general population. They are based on selected cases among the general population, who, on account of symptoms arising from the onset of carcinoma, seek hospital advice. The inclusion of these cases will at once increase the incidence of carcinoma of the gall-bladder in relation to gall-stones and upset the balance of comparison with that of a general population. The incidence of carcinoma in a general hospital could not be taken as indicative of the prevalence of this disease in a general population, nor could the incidence of lung affections among the insane be used to indicate the frequency of these conditions among the sane.

If we take Hale White's estimate (viz., that about 20 per cent. of cases of gall-stones ultimately develop primary carcinoma of the gall-bladder and biliary passages) and apply it to the Claybury statistics, then out of the 315 cases there should have been as many as 60 cases of primary malignant disease of these regions, or assuming that 10 per cent. of a general population surviving the age of 20 suffer from gall-stones, then two out of every 100 persons should develop primary carcinoma—a supposition which I think is not justified. It may be suggested that the smaller incidence of primary carcinoma among the insane is due to the fact that their average age at death is lower than the average age at death of a general population surviving the age of 20. If all the cases of gall-stones dying before the age of 60 be eliminated from the Claybury statistics, there still remain 197 cases of gall-stones occurring from the age of 60 upwards. If Hale White's estimate be applied to these cases only, there should have been close on 40 cases of primary carcinoma of the gall-bladder in the Claybury statistics.

It is not my intention to discuss the various theories as to how gall-stones act in producing carcinoma of the gall-bladder, or as to whether gall-stones precede or are formed after the commencement of malignant disease of that organ; there is probably truth in both suppositions. If, as Mignot believes from experimental researches, a stratified well-formed biliary calculus can be formed in six months, it seems quite possible that a calculus could be formed as the result of changes set up in the mucous membrane by the onset of carcinoma before operation on, or death of, the patient.

From the evidence obtained from the post-mortem statistics at Claybury Asylum I am, however, inclined to the opinion that the incidence of primary carcinoma of the gall-bladder and biliary passages in its relation to gall-stones among the general population is by no means so great as hospital statistics would lead us to believe, and that consequently there is not sufficient justification for assigning to gall-stones so important a rôle in the production of primary malignant disease of the gall-bladder and ducts.

BIBLIOGRAPHY.

BEADLES. *Trans. Path. Soc. Lond.*, xlvii, p. 82.

HALE WHITE. *Brit. Med. Journ.*, 1908, ii, p. 1729, and "Tumours of the Liver," Allbutt and Rolleston's "System of Medicine," 1908, iv, part i, pp. 204-222.

MAYO ROBSON. "Diseases of the Gall-bladder and Bile-ducts," Allbutt and Rolleston's "System of Medicine," 1908, iv, part i, pp. 223-242.

MOYNIHAN. "Gall-stones and their Surgical Treatment," 2nd ed., 1905.

ROLLESTON, H. D. "Diseases of the Liver, Gall-bladder, and Bile-ducts," and *Med. Chron.*, Manchester, 1895-96, N.S. iv, p. 241.

SCHROEDER. Quoted by MAYO ROBSON and TICEHURST.

TICEHURST, N. F. Thesis for M.B. Degree, Cantab., 1903.

WARNOCK. *Med. Times and Hosp. Gazette*, 1894, xxii, p. 667.

A complete bibliography is given by these writers.

Variability in the Gas-forming Power of Intestinal Bacteria.

By W. J. Penfold.[1]

British Medical Association Scholar.

In the following paper I propose to bring forward evidence showing that it is possible, by means of a suitable chemical agent incorporated with the nutrient medium, to select out a strain of *Bacillus coli* which fails to produce gas from certain mono- di- and poly-saccharides. Fluctuations of the gas-producing power of *Bacillus coli* have been frequently observed in the course of laboratory culture, but at present there is no convincing proof that this important property can be specifically removed at will. At the same time, so far as I am aware, gas-producing strains of the typhoid-dysentery group have not occurred in Nature or under artificial laboratory culture, and all attempts to confer this property by processes of training or otherwise have failed. For these reasons bacteriologists have always regarded gas production from sugars as one of the most stable and fundamental differences separating the coli from the typhoid-dysentery groups.

During the past few years comparatively few observations have been recorded which deal with coliform organisms showing variability in gas production when tested shortly after their isolation from Nature, but it is of some interest that the source of these organisms has in several instances been the urine.

Mair (1906) isolated from the urine a bacillus which produced acid and gas in media containing mannite, but acid only in media containing glucose and lactose. The same strain was examined later by Wilson (1908), who confirmed Mair's observations. He noted in addition, however, that the organism produced acid and gas in sorbite media, but acid only in media containing lævulose, maltose, arabinose, salicin, rhamnose, and dextrin. I shall refer later to the resemblances presented by this natural variety to the artificial variety which forms the subject of this paper.

Another organism also isolated from urine has recently been described by Wilson (1910), who regards it as intermediate between the *Bacillus coli* and the *Bacillus typhosus*. This strain produced gas from mannite but not from glucose, and further it appeared that the yield of gas from

---

[1] From the Lister Institute, London.

mannite and maltose was much greater when growth took place at 22° C. than at 37° C.

Some observations by Herter (1909) are of interest in connexion both with these naturally occurring urinary strains and with the method of selection adopted by me in my work. He found that the presence of sodium benzoate or hippurate in dextrose-peptone-water cultures of *Bacillus coli* diminishes the gas production relatively more than the vegetative activity. In his experiments the actual inhibiting agent is always present, and there is no evidence that the effect continues after subculture into sugar media containing no benzoate. The fact, however, that benzoates have this effect may afford some explanation of the presence of atypical coliform strains in the urine as this drug is not infrequently employed therapeutically in cystitis cases.

Recently, Jacobsen (1910) has described a typhoid bacillus which, when grown on repeatedly autoclaved agar, showed two distinct types of colony, viz.: (1) a very tiny colony, and (2) a large colony. Subculture of the large colony on the same agar gave rise to a pure race showing large colonies only. Secondary colonies or papillæ were not observed by Jacobsen in the case of the *Bacillus typhosus* he experimented with.

It was a very similar phenomenon observed by me when examining the influence on intestinal organisms of the constituents entering into the production of glycocoll (Penfold, 1910) that led me to investigate further the properties of the strains selected out.

AUTHOR'S EXPERIMENTS.

If the *Bacillus coli* (Escherich) be grown on agar containing a suitable concentration of monochloracetic acid (sodium salt) the colonies show a marked diversity of size, some being very large and others very minute.

Preparation of Medium.

Tubes of 2 per cent. peptone-agar (without broth) are put up, each containing 15 c.c. A known weight of monochloracetic acid is dissolved in a small amount of water and the solution is made just faintly alkaline to neutral litmus paper by means of sodium carbonate in saturated solution. The solution is then made up to a known volume and filtered through a Berkefeld filter. It must not be boiled, otherwise the monochloracetic acid is decomposed. Any required quantity of this solution is then added to a series of plates, after which the melted peptone-agar is poured in and thoroughly mixed. Throughout this paper the strength of medium is always expressed in terms of the original acid used.

Appearance of Chloracetic Acid Agar Plates of Bacillus coli (Escherich).

Concentration, 0·01 per cent.: After twenty-four hours' incubation at 37° C., a luxuriant growth occurred, the colonies being all of uniform size. About the fourth day all the colonies at the margin, which are usually somewhat larger owing to less crowding together, showed papillary projections, and two days later the number of papillæ had in some cases increased to six. These papillæ were situated irregularly on any part of the colony. Even a concentration of 0·001 per cent. was sufficient to produce these papillæ, but they were invariably of much smaller size.

Concentration, 0·05 per cent.: With this concentration the plates showed a mixture of very tiny colonies and very large ones when examined on the second or third day. By the sixth day the large colonies all showed papillæ. There was no tendency for the large colonies to appear in the marginal non-crowded portions of the plate. In fact they were more numerous in the densely crowded central portions.

Concentration, 0·1 per cent. to 1·5 per cent.: With these high concentrations no small colonies appeared, and it was not till the second or third day that any colonies appeared at all. These were very few, but attained large dimensions.

Examination of other Organisms on the Monochloracetic Acid Medium.

Bacillus enteritidis (Gaertner): This organism showed exactly similar appearances to *Bacillus coli* on plates containing a suitable concentration of the acid, and, as with *Bacillus coli*, a high concentration of the acid was required to inhibit entirely the growth of the small type of colony.

Coliform Organisms freshly isolated from Human Fæces and Urine.—Several lactose-fermenting organisms from these sources were plated out on chloracetic acid agar, when it was found that in each case large colonies and very small colonies developed, similar in appearance to those of *Bacillus coli*. These two types resulted even with concentrations of 0·3 per cent. and 0·5 per cent. of the acid, and apparently the fresh strains were more resistant to the acid than the stock strain of *Bacillus coli* (Escherich). Papillæ-formation also appeared in five of the six strains at the above concentrations, the papillæ attaining a considerable size by the seventh day.

Special attention must be drawn to the fact that in concentrations preventing the growth of all small colonies, one or two colonies quite commonly appear late and can be easily proved not to be contaminations; e.g., in the strain of *Bacillus coli* (Escherich) which I employed no small colonies appeared in a series of plates where the concentration exceeded 0·08 per cent. of chloracetic acid, but one large colony appeared on a 0·5 per cent. plate and one on a 1·5 per cent. plate. These late colonies were carefully examined and found to be colonies of *Bacillus coli* (Escherich). Similar differences in resistance on the part of different colonies of *Bacillus enteritidis* (Gaertner) were also noted, but they were not so marked.

Bacillus typhosus: Nine strains of this organism were plated out on the same medium, but only a moderate amount of variability occurred in respect of the size of the colonies. No papillæ-formation was observed.

Further Selection of Bacillus coli (Escherich) on the Monochloracetic Acid Medium.

After one week's growth on a 0·5 per cent. plate a *large* colony of *Bacillus coli* was picked off, subcultured into broth, and replated on chloracetic agar. The concentrations of acid in these new plates varied from 0·3 per cent. to 2·0 per cent. All the plates showed an excellent growth after twenty-four hours, and no colonies of the *small* type developed. Papillæ appeared on the colonies by the ninth day, and were best developed on plates with a concentration of 1·3 per cent. A subculture on ordinary agar was made from one of the colonies on a 2 per cent. plate, and from this agar culture, peptone-water Durham tubes containing various carbohydrates were inoculated. Each tube contained about 6 c.c. of the peptone solution containing the carbohydrates. The internal gas tubes were 2 in. long, with a diameter of approximately a $\frac{1}{4}$ in.

All the experiments on gas production recorded in this paper were performed in this manner, and the results are therefore subject to the degree of accuracy of the Durham tube method.

In Table I are recorded the fermentation reactions of the chloracetic acid variety with those of the normal *Bacillus coli*, from which it was selected. The observations were made on the third day. Two marked differences will be noted in the above table—viz., the new strain developed no gas in glucose and practically none in lactose. Both tubes, however, showed full acidity after twenty-four hours' growth. A further change, not shown on the table, was that the normal strain clotted milk in two days, while the new variety required three days to do so.

TABLE I.

Normal *Bacillus coli* (Escherich)	Glucose	Mannite	Dulcite	Lactose	Saccharose	Sorbite	Gelatine	Litmus milk	Broth	Morphology motility
Tubes examined on third day after seeding	A F G $\frac{1}{2}$	A F G $\frac{3}{4}$	A F G $\frac{1}{2}$	A F G $\frac{1}{2}$	—	A F G $\frac{2}{3}$	No liquef. observed, 14 days	Acid and clot	Cloud and slight film	Motile rod
Bacillus coli (Escherich) from the second chloracetic agar plate										
Tubes examined on third day after seeding	A F No G	A F G	A F G $\frac{1}{2}$	A F G trace	—	A F G $\frac{3}{4}$	No liquef. observed, 14 days	Acid and clot	Cloud and slight film	Motile rod

A F = full acidity. The fractions show the amount of the gas tube occupied by gas. G = gas.

The selective process was continued, the new variant being plated out again on chloracetic agar. Good growth took place in concentrations as high as 2 per cent. and slightly at 3 per cent. At higher concentrations the plates were sterile. After three days' growth, two colonies

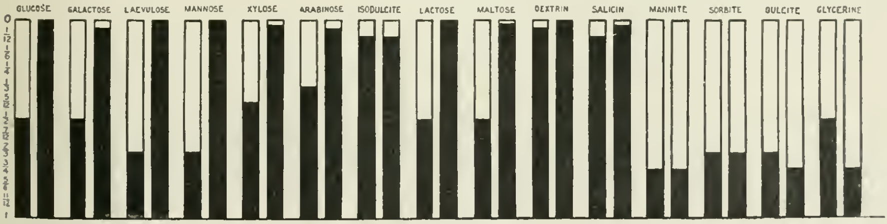


CHART I.

Bacillus coli communis (Escherich). Left-hand member of couple = normal strain; right-hand member of couple = variant strain. In this chart the black columns represent gas tubes containing no gas, while in the other columns the white spaces represent the quantities of gas formed. All the tubes were incubated for five days at 37° C., and the amount of gas indicated in the chart is the *greatest* amount found in the individual tubes during the whole period. The tubes are arranged in couples, the left-hand member representing the normal *Bacillus coli*.

from a 2 per cent. plate and two from a 3 per cent. plate were inoculated into lactose-peptone-water and incubated for six days at 37° C. Full acidity developed overnight in all cases, and three of the tubes did not show the slightest sign of gas formation during the whole period, although they were observed daily. The fourth tube developed a bubble of gas of the size of a pin's head. From a 3 per cent. plate of this third selection a colony was taken and plated out on McConkey's medium. Four separate colonies from the latter were inoculated into glucose-peptone-water and incubated for eleven days at 37° C. No gas formation occurred. A fourth selection was made on 3 per cent. chloracetic agar when papillæ again developed. These were small by the third day, but quite prominent by the fifth day. A colony from the 3 per cent. plate was then inoculated into the various carbohydrate media, with results shown in Chart I.

ANALYSIS OF RESULTS.

Gas production is depressed in glucose, lævulose, mannose, lactose, and dextrin completely, in galactose, maltose, xylose, arabinose and salicin almost completely, while isodulcite is not affected. Also in mannite, dulcite, glycerine, and sorbite the gas production by the new strain shows no diminution from the normal. Apparently, therefore, the gas production from sugars and bodies yielding sugars on decomposition (eleven in all) is entirely, or almost entirely abolished, with the exception of isodulcite, whereas the gas production from alcohols is undiminished. Probably the presence of a methyl group in isodulcite, which is a methyl pentose, may account for its difference of behaviour from the other sugars.

Identification of the New Variety.

In all questions of variation the careful examination of the identity of the varied organism has to be attended to. The following evidence is adduced in support of identity :—

(1) The production of acid occurred in the same carbohydrates exactly as in the case of the normal strain.

(2) The normal and variant agreed in general morphology, staining reactions, motility and indol production.

(3) The variant did not clot milk, but this point is of little importance, as the partially selected variety took, as previously stated, a longer time to clot milk than the normal strain.

(4) Neither form liquefied gelatine.

(5) A rabbit was immunized with the variant strain, and its serum agglutinated the normal *Bacillus coli* in all dilutions from 1 in 10 to 1 in 1,600. Before immunization the serum of the rabbit did not agglutinate the normal *Bacillus coli* in dilutions of 1 in 10.

(6) Complement-fixation experiments were kindly performed for me by Dr. H. R. Dean, with extracts of the normal and variant strains and the immune serum from the rabbit. His results are recorded in Table II, and afford further evidence of the identity of the two strains. We are therefore justified in believing that the new variety is a genuine descendant of the *Bacillus coli* (Escherich), from which we set out.

Testing the New Variant derived from Bacillus enteritidis (Gaertner).

The *Bacillus enteritidis* (Gaertner) was subjected to the same treatment as *Bacillus coli*, and after four platings on the special medium, yielded a strain whose fermentation reactions are recorded in Chart II, as compared with the normal organism. It will be observed that the depression of the gas-producing power again affects all the sugars except isodulcite. A slight depression in the amount of gas formed from alcohols is also noticeable, but a sharp line of demarcation can still be drawn between sugars and alcohols in respect of gas production by the varied strain. We have seen that the variant of *Bacillus coli* fermented three hexoses without gas formation, while from the pentoses a slight amount of gas was still developed. With the Gaertner variant, on the other hand, it is the pentoses (arabinose and xylose) which are fermented without gas formation, while the hexoses still yield a small quantity of gas.

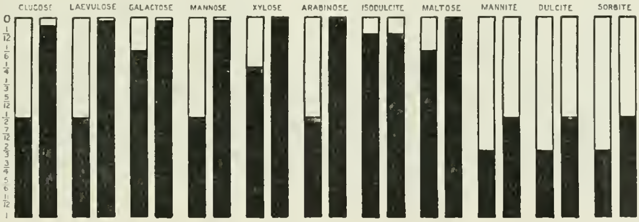


Chart II.

Bacillus enteritidis (Gaertner). Left-hand member of couple = normal strain; right-hand member of couple = variant strain.

Table II.—To show Scheme of Complement-fixation Experiment with Extracts of Normal and Variant Bacillus Coli and Antiserum, obtained by Immunization of the Rabbit with the Variant Strain (Dr. H. R. Dean).

To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. After one hour in the incubator at 37° C. there were added 0·5 c.c. of a 1 in 100 dilution of heated hæmolytic serum (rabbit *v.* sheep) and 0·5 c.c. of a 1 in 20 suspension of sheep's corpuscles. The tubes were then incubated for forty-five minutes and the results read and recorded.

Tube	Extract of normal *Bacillus coli;* 0·5 c.c. of dilution	+ 0·5 c.c. normal saline	+ 0·5 c.c. of 1 in 10 dilution of normal serum	+ 0·5 c.c. of 1 in 10 dilution of antiserum	+ 0·5 c.c. of 1 in 100 dilution of antiserum
1	1 in 10	Complete hæmolysis	Almost complete hæmolysis	No hæmolysis	Slight hæmolysis
2	,, 20	,,	Complete hæmolysis	,,	,,
3	,, 40	,,	,,	,,	Marked hæmolysis
4	,, 80	,,	,,	Trace of hæmolysis	Almost complete hæmolysis
5	,, 160	,,	,,	,,	,,
6	,, 320	,,	,,	Almost complete hæmolysis	Complete hæmolysis
7	,, 640	,,	,,	,,	,,
8	,, 1,280	,,	,,	Complete hæmolysis	,,
	Extract of variant *Bacillus coli;* 0·5 c.c. in dilution				
1	1 in 10	Complete hæmolysis	Marked hæmolysis	No hæmolysis	Slight hæmolysis
2	,, 20	,,	Almost complete hæmolysis	,,	,,
3	,, 40	,,	,,	,,	,,
4	,, 80	,,	Complete hæmolysis	,,	Marked hæmolysis
5	,, 160	,,	,,	Slight hæmolysis	,,
6	,, 320	,,	,,	Marked hæmolysis	Complete hæmolysis
7	,, 640	,,	,,	Almost complete hæmolysis	,,
8	,, 1,280	,,	,,	Complete hæmolysis	,,

The above table shows that no difference can be detected by this method between the extracts prepared from the normal and from the varied strain of *Bacillus coli.*

Identification of the Variant Form of the Bacillus enteritidis (Gaertner).

The sugar reactions of the new strain again agreed with the normal in respect of acid formation. Both strains showed similar morphology and staining reactions, they were equally motile, and produced no liquefaction of gelatine. The variant strain was also agglutinated by a normal Gaertner serum in a dilution of 1 in 30,000. The acidity in milk is more transient in the case of the varied than in the case of the normal strain.

Bacillus Grünthal.

One of the coliform organisms freshly isolated from fæces was found to give reactions practically identical with those of the Bacillus of Grünthal.

By similar methods of selection a variant of this organism was produced which yielded the fermentation reactions recorded in Chart III.

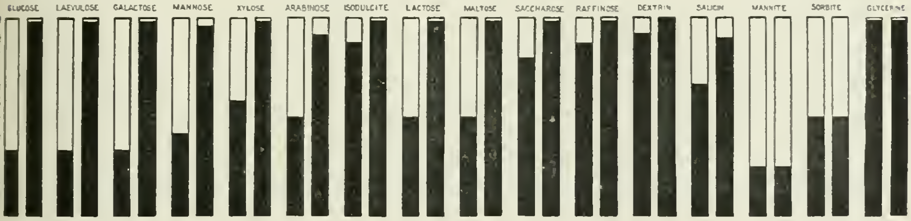


CHART III.

Bacillus Grünthal. Left-hand member of couple = normal strain; right-hand member of couple = variant strain.

The same differentiation as between sugars and alcohols is apparent, and in this case isodulcite falls into line with the other sugars. Some experiments have also been made with the *Bacillus lactis aerogenes*, but so far the fermentation reactions of the variant are not sensibly altered from the normal. The selective process will require to be continued.

Durability of the New Character.

After three platings on chloracetic acid agar, the variant *Bacillus coli* was tested on glucose-peptone-water in Durham's tubes, and gave no gas. The strain was then plated out on McConkey's medium, and four separate colonies inoculated into glucose-peptone-water. These were subcultured in the same medium every day for six days, as four separate series, and each separate tube was incubated for five days at 37° C. Full acidity appeared overnight in each case, but in no tube out of the twenty-four did a single gas bubble appear. Daily examinations were made. Two series of subcultures of the same strain on peptone water, extending over five days, showed similar complete retention of the new character. Also from above-mentioned sixth generation in glucose-peptone-water a subculture was made into peptone water containing sodium acetate. On this new medium it was grown for ten days, and then re-tested on glucose, when it was found to yield a tiny bubble of gas of the size of a pin's head, after forty-eight hours' growth. This amount did not increase.

It may therefore be concluded that the variant is in a fairly stable, though not entirely stable, position with regard to its action on glucose. I have shown in the case of *Bacillus typhosus* and dulcite (Penfold, 1910) that a new unstable character may be rendered stable by further selection, and it is only reasonable to suppose that further growth on chloracetic acid will give the desired permanency.

Discussion on the Fermentation Properties of the Variant Bacillus coli.

Harden (1901) has shown that *Bacillus coli* gives only a small yield of formic acid from glucose at ordinary atmospheric pressure. If the pressure be increased, the yield of gas diminishes, but the yield of formic acid increases. He thinks, therefore, that it is not unlikely that the gas produced by *Bacillus coli* arises from the decomposition of formic acid.

I have tested the action of the normal *Bacillus coli*, and the variant form on peptone water containing sodium formate, and find that a good yield of gas occurs in both cases. It may be suggested, therefore, that if Harden's view prove to be correct, the selective process has caused the removal of the formic-acid-forming ferment, but apparently has not interfered with the formic-acid-splitting ferment.

It is known that the *Bacillus typhosus* produces formic acid from glucose, but no gas. Neither can it produce gas in peptone water containing sodium formate. When, however, our new variant of *Bacillus coli* is grown in symbiosis with the *Bacillus typhosus* in a glucose medium, gas is produced. This is almost certainly due to the fact that the formic acid produced by the *Bacillus typhosus* is split up by the variant *Bacillus coli*. In the peptone water containing sugar, the acid production of the varied strain is apparently not diminished. This suggests the presence of another ferment attacking sugar without the formation of formic acid.

When grown under anaerobic conditions in a Buchner's tube, or in the anaerobic gas tube described by the writer (Penfold, 1910), the variant *Bacillus coli* still refuses to produce gas from glucose, except to the extent of a pin's-head bubble, although the full acidity develops overnight. This marked acid reaction occurring overnight in the anaerobic state suggests that the yield of acid is not the result of oxidation of the glucose by atmospheric oxygen. The facts so far ascertained with regard to the fermentative activity of the variant *Bacillus coli* suggests that the gas derived from glucose by the normal *Bacillus coli* is the result of at least two ferments acting in sequence, the first forming formic acid, and the second splitting up the acid so formed. It seems probable that a common ferment acts in some stage of the production of gas from all or the majority of the sugars. This is the most easy explanation of the simultaneous modification of this one function in the case of so many sugars.

The ferment which is removed by the selective process is evidently not concerned in the production of gas from alcohols. The essential difference in the structure of the two classes of substances—alcohols and sugars—is the presence of the aldehyde group in the latter, so that probably the missing ferment has some relationship to this group. Dr. Harden is at the present time making an investigation into the chemical activity of the variant *Bacillus coli*, which will doubtless throw much light on its peculiarities.

Wilson's very remarkable organism referred to in an earlier portion of this paper showed greater similarity in its action on maltose and mannite than on maltose and glucose—a fact which at least suggests that it is in no way related to the *Bacillus coli communis*. Mair's organism, on the other hand, is almost certainly a natural variant of the *Bacillus coli* group, and but for the fact that it does not ferment raffinose, it may very well have been derived from the *Bacillus acidi lactici*.

Significance of the Papillæ.

In order to throw some light on this question, the following experiment was performed: A "small" colony from the first chloracetic acid plate of *Bacillus Grünthal* was re-plated on chloracetic acid agar. From the latter three subcultures into glucose-peptone-water were made: (1) From a "small" colony; (2) from the flat portion of a "large" colony; and (3) from the papilla of a large colony. The yield of gas after twenty-four hours was as follows:—

(1) "Small" colony tube	One-third of a Durham tube.
(2) "Large" colony tube	One-twelfth of a Durham tube.
(3) Papilla tube	Pin's-head bubble.

It is evident, therefore, that the papillæ, which are composed of the most resistant bacteria on the plates, are at the same time the poorest gas producers.

How does the Monochloracetic Acid Act?

Does the acid act directly, or does the organism attack it first, and produce from it a toxic compound which inhibits its growth? If the latter explanation held, it is easy to see that the power to attack chloracetic acid would result in the inhibition of growth of those individuals using it, while individuals using it less would soon outgrow their fellows.

The following substances have been experimented with in the hope of getting more information on this subject: Trichloracetic acid and dichloracetic acid have been neutralized and added to agar in the same way as the monochloracetic acid. Colonies of *Bacillus coli* on the dichloracetic acid medium show no great variability in size, and the normal organisms are not prevented from growing by so large an amount as 2 per cent. of the neutralized acid. This is a very different result from that obtained with the monochloracetic acid which frequently, in a concentration of ·1 per cent., prevented the growth of small colonies. No papillæ developed on the colonies in the dichloracetic acid plates. The neutralized trichloracetic acid prevented the growth of *Bacillus coli* on agar at a strength of 2 per cent., but not at 1 per cent., and no marked variability in the size of the colonies was observed. No papillæ developed.

With cyano-acetic acid no variability in size resulted and no papillæ appeared. Normal strains of *Bacillus coli* and of *Bacillus Gaertner* have also been grown on peptone water containing sodium acetate. No gas was formed and no papillæ developed on colonies of *Bacillus coli* grown on agar containing the salt, concentrations varying from 0·001 per cent. to 1 per cent. being tried. Hitherto, therefore, I have got no evidence as to why the monochloracetic acid acts in the selective fashion described in this paper.

SUMMARY AND CONCLUSIONS.

(1) The gas-producing power of *Bacillus coli* on glucose and lactose media can be removed by growth on monochloracetic acid agar.

(2) This depression of the gas-producing power is valid for all sugars fermented by the organism (except isodulcite), while it is not valid for the alcohols fermented.

(3) *Bacillus enteritidis* (Gaertner) and *Bacillus Grünthal* are affected in a similar way by growth on monochloracetic acid agar. The depression in the case of *Bacillus Grünthal* is, however, general for all the sugars tested.

(4) The power of producing gas from formates is retained by the new varieties.

(5) Strains of intestinal organisms highly resistant to sodium chloracetate are readily produced from many natural varieties.

(6) When *Bacillus coli* (Escherich), *Bacillus enteritidis* (Gaertner), *Bacillus Grünthal,* and many other intestinal organisms are grown on monochloracetic acid agar, a marked variability in the size of the colonies occurs, and secondary colonies or papillæ develop on the surface of the large forms.

(7) The investigation of the chemical activity of the new varieties is likely to throw some light on the mechanism of the fermentation processes of bacteria.

In conclusion, I desire to thank Dr. Ledingham, Dr. Harden, and Dr. Maclean, of the Lister Institute, for much help in many points of difficulty; and also Dr. H. R. Dean for kindly applying the complement-fixation test in the identification of the variant *Bacillus coli.*

BIBLIOGRAPHY.

HARDEN. "The Chemical Action of *Bacillus coli communis* on Carbohydrates and Allied Compounds," *Journ. Chem. Soc. Trans.*, 1901, p. 610.

HERTER. "Notes on the Action of Sodium Benzoate on the Multiplication and Gas Production of various Bacteria," *Journ. of Biol. Chem.*, Baltimore, 1909, vii, p. 59.

JACOBSEN. "Mitteilungen über einen variablen Typhusstamm," *Centralbl. f. Bakt.*, Jena, Abt. I, Orig., 1910, lvi, p. 208.

MAIR. "Notes on a Paracolon Bacillus found in Urine," *Brit. Med. Journ.*, 1906, i, p. 438.

PENFOLD. "Bacterial Variation," *Journ. of Path. and Bact.*, 1910, xiv, p. 406.

Idem. "Variations of the Fermentation Properties of the *B. typhosus*," *Brit. Med. Journ.*, 1910, ii, p. 1672.

Idem. "Studies in the Anaërobic Culture of the Intestinal Micro-organisms," *Brit. Med. Journ.*, 1910, ii, p. 1513.

WILSON. "Colon Bacilli in the Urine," *Journ. of Hyg.*, Camb., 1908, viii, p. 543.

Idem. "Variability of Bacteria," *Brit. Med. Journ.*, 1910, ii, p. 1909.

On the Microscopic Structure of Uric Acid Calculi.

By S. G. SHATTOCK.

De structurâ calculorum penitiori ex acido urico constantium.

SUMMARIUM.

AD structuram suam in genera duo describi possunt calculi ex acido urico constantes: alii cancellati, alii compacti.

Haec differentia, ut sectionibus microscopicis monstratur, a crystallizandi modo dependet.

In calculis cancellatis nucleus de crystallis cubicis construitur, in trabeculis, sine ordine, conjunctis. Super nucleum imponuntur porticuum series ex crystallis cubicis quoque constructa: ita constituitur calculi corpus.

De varietate compactâ, construitur calculus de crystallorum longorum conis radialiter e mediâ concretione evenientibus.

Striis tenuissimis transverse imprimuntur coni, atque zonulis substantiae minute divisae quae inter crystallos productos includitur, et ex acido urico constat.

Crystallorum productio supersaturatae urinae conditioni ascribi debet.

Crystallos quum in liquore supersaturato oriuntur longitudinem insignem acquirere bene cognoscitur.

Hanc productionem Miers ita explicare conatur[1]: aliquantulum desaturatur liquor a lateribus cujusque crystalli, ipso facto crystallizandi; apud extremitates ejus, autem, crystallus in portionem supersaturatam liquoris sine impedimento crescere potest.

Liquore supersaturato, insuper, agitato crystalli minuti abundanter pariuntur: in liquore minus saturato, crystallis jam praesentibus, idem evenit.

In hoc modo, forsitan, explicanda est depositio crystallorum minutorum acidi urici, in calculis et compactis et cancellatis.

De striis tenuibus, jam descriptis, in portionibus quibusdam calculorum compactorum, explicandae sunt et acidum uricum de urinâ abstrahendo, et caloris incremento cum crystallorum formatione sociato. His causis conjunctim desaturatur liquor qui calculum proxime circumdat; et depositio ad tempus differtur.

---

[1] *Mineralogical Magazine*, 1908, xv, No. 68, pp. 39-41.

Structura laminata ubicunque reperitur, concretionem passubus crevisse indicat: etsi credere licet nucleum ipsum citius ex liquore majus saturato ortum esse.

In the forty-sixth volume of the *Pathological Society's Transactions*[1] I have described, in conjunction with the late Dr. William M. Ord, the microscopic structure of urinary calculi composed of calcium oxalate. Although Dr. Vandyke Carter had previously published a small work on the microscopic structure of urinary calculi, his examination was very defective, for the reason that the only method practised by him consisted in the study of the minute particles obtained by crushing, or similar means of disintegration. In the communication referred to, I adopted the method of preparing microscopic sections of entire calculi after the manner employed by petrologists, and which I had already utilized for the examination of certain iridescent prostatic calculi, described in the thirty-seventh volume of the same *Transactions*.[2]

In the selection of material for microscopic study, calculi of small or moderate size are preferable, and those, moreover, from groups, in order that more than a single example may be examined, and a full chemical analysis carried out.

TECHNIQUE.

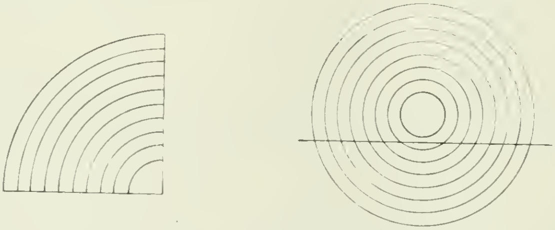
The method consists in rubbing away half of the calculus on a file or on glass-paper, the final part of the grinding being carried out on a wet hone. In some calculi the nucleus is so differentiated as to readily allow of identification. This is, however, not always the case; one has to guard against the fallacy of mistaking the section of a zone around the proper nucleus for the nucleus itself, since both will present an equally circular figure. This difficulty is usually surmountable by examining the wet surface of the grinding at intervals with a hand lens. When doubt still exists the only sure method is to grind the calculus in two planes, at right angles to each other, so as to obtain a sector consisting of a fourth of the whole; the centre can then be determined by its exposure in both planes.

After the process is completed, the ground surface is carefully washed and cleared of débris by allowing water to drop from a height through a cone of filter paper; the half of the calculus so prepared is excluded from dust and allowed to dry. It is in the next place cemented to a slide with solid Canada balsam, a fragment of which is heated on the slide over a spirit lamp until it melts; and in order to diminish the brittleness of the balsam, a small quantity of the ordinary mounting solution in xylol is first placed on the slide, the materials being mixed after heating by rocking the latter to and fro. The half of the calculus is now placed on the slide, the central portion of which is already covered with the melted balsam. A copious effervescence takes place from the under side of the calculus, owing to the expansion of the air in its interstices. The slide must now be turned over so that the ground surface can be viewed, the calculus being moved about and gently pressed until no trace of air remains between the specimen and the glass. As soon as the balsam has set the material is ground away from the convex side until the chief bulk has been removed. The last and most delicate part of the process is the grinding of the section to such a degree of thinness as to allow the light to pass through it. This is done first on fine glass-paper, but completed on the hone with water. When the requisite thinness is reached, the surface is washed by allowing water to drop on to it through a cone of filter paper; lastly, when the section is dry, a xylol solution of balsam is placed on it and the cover-glass applied.

---

[1] *Trans. Path. Soc. Lond.*, 1895, xlvi, p. 91.

[2] *Ibid.*, 1886, xxxvii, p. 325.



A Fig. 1. *B*

A.—Method of grinding in two planes at right angles in order to ensure the identification of the centre.

B.—Showing the risk of mistaking a section of a zone near the nucleus for the nucleus itself, either by not reaching the true centre or by grinding past it.

I may now proceed to describe in detail a selection of typical specimens, and afterwards make some general observations from the data they furnish.

One of the most obvious macroscopic differences that arrests attention on inspecting the divided surfaces of uric acid calculi is the variation in their density. Some are so compact throughout that no interspaces whatever appear; others are markedly porous. The two varieties may be termed compact and cancellous. Of these, the cancellous depart the least in their microscopic characters from uric acid sediment or gravel; the compact are more highly differentiated.

Nearly all the specimens which I have studied are from the large collection in the Museum of the Royal College of Surgeons, the chief part of which were analysed some years ago by Mr. Taylor, but in many cases Mr. H. R. Le Sueur has made a further chemical examination of those selected.

THE CANCELLOUS VARIETY.

To take the cancellous variety first, I may describe examples from four different groups. The structure of calculi composed of urate is altogether different, and is reserved for a future communication.

Specimen I (A 19), Museum of the Royal College of Surgeons.—Uric Acid Series.

Of this group I have examined three. The special analysis by Mr. Le Sueur showed an absence of sodium and ammonium. A second critical examination for ammonium was equally quite negative; a considerable bulk of material was used. The macroscopic characters presented by the divided surface of a fourth, oval, specimen 2·5 cm. by 1·5 cm. in chief diameters, are as follow: The exterior is quite smooth. There is a nucleus 1·5 cm. long, of coarsely porous structure, the trabeculæ of which are devoid of any disposition, either concentric or radial. Beyond the nucleus the "body" is constructed of thin concentric laminæ, slightly undulatory in course, and separated by narrow intervals which are partly filled with deposit. Near the exterior the thickness of the concentric laminæ increases and the intervening spaces become narrower; the laminæ themselves, moreover, are more compact and well defined. The cancellous character is due to the incomplete filling of the interlamellar spaces. The calculus is of a pale brown colour.

Microscopic structure of a calculus about 1 cm. in diameter: The nucleus consists of large, pale yellow, cuboidal crystals, cohering in irregular trabeculæ so as to produce a coarsely cancellous structure. Intimately intermingled with the large crystals composing the trabeculæ there are dense clusters of very fine crystals, which might be described as bacilliform, the presence of which produces a marked diminution in the transparency of the section. These minute rod-like crystals are not set in spherular aggregations. The "body" starts by the aggregation of crystals similar to those of the nucleus, but smaller in size, to form a continuous concentric zone which is not geometrically circular, but undulatory, like those that follow it. The body beyond this zone, in short, consists of a series of concentrically disposed undulating laminæ interconnected at intervals so as to produce a regular series of shallow arcades. The intervals between the arcades are incompletely filled with cuboidal crystals like those of the nucleus, but somewhat smaller in size. The crystals forming these arcades are, again, smaller than those between them, and they are amassed without any trace of order. In the mid-substance of each arcade there is an ill-defined line of less transparency, due to the intercalation of minute bacilliform crystals like those already referred to in the trabeculæ of the nucleus. Towards the exterior of the calculus the narrow arcades are replaced by a broader zone of more compact structure, made up of radially set, much elongated, yet comparatively slender crystals, between which the less transparent bacilliform material is deposited, so as to produce a series of ill-defined and incomplete concentric lines. This more compact and more "organized" zone is succeeded by laminæ of the same kind as the arcades on its inner aspect. And the actual surface of the calculus is constituted by a compact lamina like that just described, the elongated crystals of which are wanting in exact radiality, and obliquely intersect. At the growing surface itself there appear the free ends of these long crystals, and between them the finely divided material, more than once referred to; but even under $\frac{1}{6}$ obj. the surface is almost smooth, or regularly curvilinear.

The microscopic sections of a second and of a third calculus from the same group exhibit closely similar appearances, except that the periphery in each is represented by a thicker zone of the long radiating crystals crossed by less transparent, ill-defined concentric lines of the finely divided crystalline material. The crystals vary in length; some of them pass through several such concentric zones, others only through a few. The free surface is quite smooth, or very slightly jagged, and formed by the growing ends of the radiating elements. Between this outermost zone and the arcades near the nucleus there occur arcades of a transitional character, which are constructed in part of elongated crystals, more or less radially disposed, and amongst which there is the usual less transparent material, the spaces between the arcades being loosely filled with coarse cuboidal crystals.

Specimen II (A 1), Museum of the Royal College of Surgeons.

As very similar to the foregoing, I may describe what happens to be the very first example of the uric acid series of calculi in the College collection. The calculi compose a small group, of which I examined three. Mr. Le Sueur was unable to find any sodium, calcium, or ammonium ; the last was particularly looked for on two occasions, a considerable bulk of material being used. The *nucleus* is built up of pale yellow cuboidal crystals of large size, clustered without any order, and so loosely as to leave lacunæ of considerable extent, the whole presenting an irregularly cancellated structure. The form of the individual crystals composing the trabeculæ is difficult to trace, owing to their closeness of apposition ; but when this can be observed it is that of rectangular plaques or prisms. The larger crystalline blocks in the trabeculæ are mostly of irregular polyhedral form, from modifications due to the completion of their growth whilst in imperfect apposition, and, like the other crystals, they present markings arising from secondary cleavage, the cleavage, in some instances, having taken place in two directions at right angles to each other. Certain of the trabeculæ, as in the preceding specimen, present opaque centres arising from the intermixture of finely crystalline material like that occurring in the concentric zones of the body. That this material is not the result of the secondary cleavage of larger crystals appears from the absence of parallelism in it. The massive elements of the nucleus are succeeded by a continuous zone of much smaller crystals, many of which are of elongated form, though quite slender. In this zone there is a considerable amount of the finely divided bacilliform material which renders it to a large degree opaque. The body so commencing is thereafter more or less regularly constructed of slightly wavy concentric zones of slender, elongated and other crystals without any orderly disposition, the zones presenting one or more ill-defined, less transparent lines of more finely divided crystalline material. In the irregular mass of crystals composing such a zone well-formed rosettes may be embedded. Between the wavy laminæ there is a series of spaces loosely and incompletely occupied by coarser cuboidal crystals. Here and there a certain portion of the circuit of a lamella is of more definite and compact structure, and consists of elongated crystals radially set with respect to the centre of the calculus, and marked with one or more ill-defined lines of finely crystalline and less transparent material. The porosity of the body is due to the incomplete way in which the interlamellar spaces are filled in with the coarser cuboidal crystals.

Specimen III.—Uric Acid with intercalated zones of Calcium Oxalate.

The following specimen I may adduce as a further example of the cancellous variety, though associated with the presence of narrow zones of calcium oxalate. Mr. Le Sueur was unable to discover any sodium or

EXPLANATION OF PLATE I.

Fig. 1 (A 19, Museum of the Royal College of Surgeons).—Cancellous variety of uric acid calculus (2 in. obj.), showing a small nucleus of (cuboidal) crystals, followed by a series of undulating, interconnected laminæ or arcades. The structure is particularly open in the neighbourhood of the nucleus.

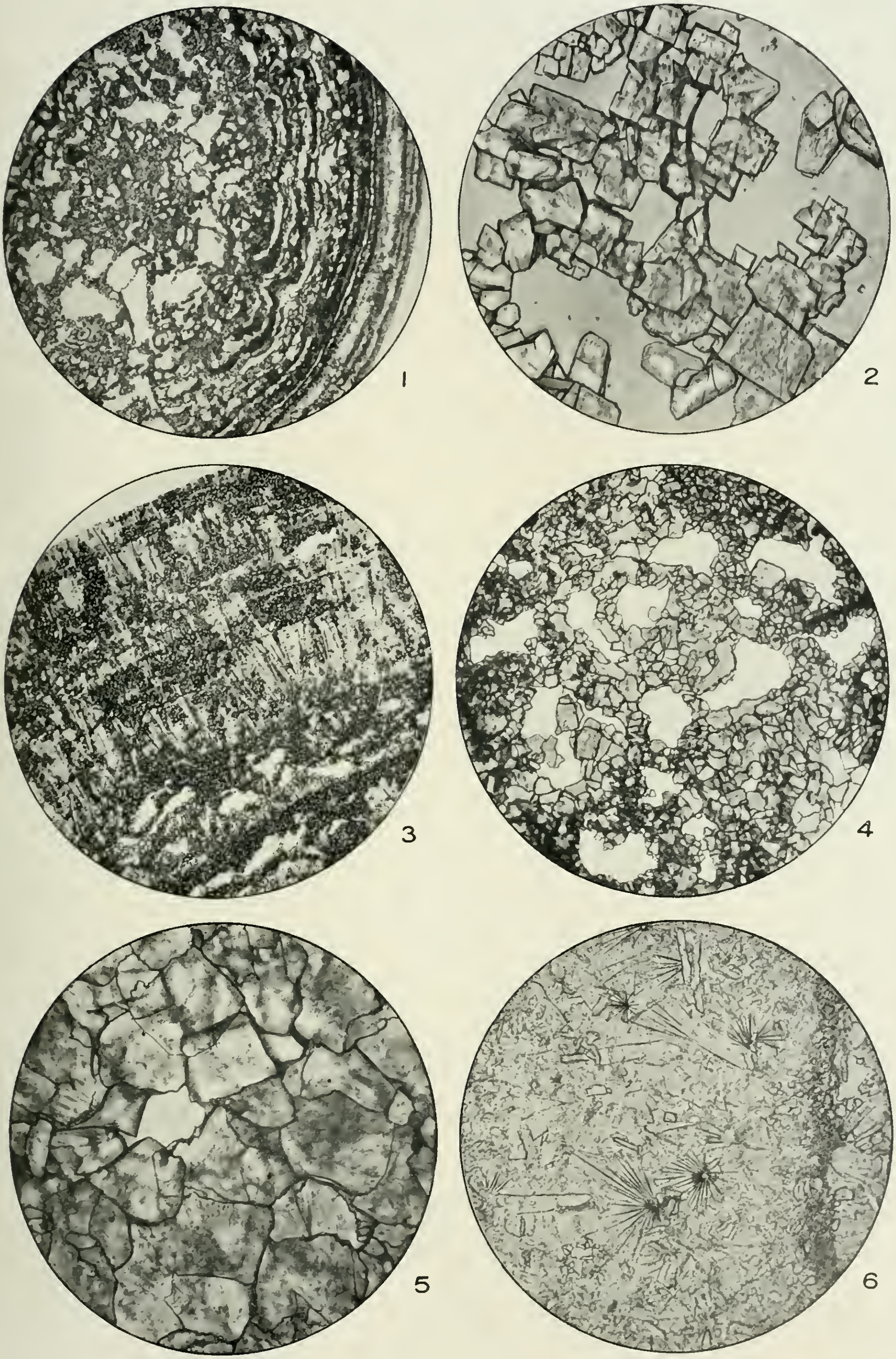
Fig. 2 (A 19, Museum of the Royal College of Surgeons).—Portion of the nucleus of the preceding calculus ($\frac{1}{6}$ obj.), showing the large cuboidal crystals of which it consists, cohering to form irregular trabeculæ with interspaces.

Fig. 3 (A 19, Museum of the Royal College of Surgeons).—The outermost portion of another calculus of the same group ($\frac{2}{3}$ obj.), showing a compact crust of elongated, somewhat radially disposed crystals between which there is much finely divided uric acid. Below the compact crust is seen the outer part of the open system of arcades which construct the chief bulk of the body of the calculus; these arcades consist of crystals without elongation and with much of the material just referred to intermingled. The change in construction is attributable to the presence of urine more highly saturated with uric acid. The elongation of the crystals, with their interspaces, corresponds with the appearances arising in supersaturated solutions of various salts when crystallization is started in such. The shower of minute crystals, such as fill the interspaces, is also imitable in supersaturated or "labile" solutions, when subjected to movement; and the same will happen in less saturated solutions under similar circumstances, provided crystals are already present.

Fig. 4 (A 1, Museum of the Royal College of Surgeons).—Cancellous variety of uric acid calculus ($\frac{2}{3}$ obj.), showing the nucleus, which consists of cuboidal crystals aggregated to form irregular trabeculæ, with intervening lacunæ.

Fig. 5 (A 1, Museum of the Royal College of Surgeons).—Portion of the same nucleus ($\frac{1}{6}$ obj.), showing the cuboidal crystals composing it.

Fig. 6 (A 1, Museum of the Royal College of Surgeons).—Showing the structure of one of the zones ($\frac{1}{6}$ obj.) entering into the construction of the body, the zones themselves being separated by a series of intervals which are loosely and incompletely occupied by coarser cuboidal crystals. The zone consists of slender, elongated, and other crystals, without any orderly disposition; rosettes occur embedded in the general mass. The elongation and loose arrangement of the crystals indicate that the zone is due to the presence of urine more highly saturated with uric acid than was the case with those constructed of cuboidal crystals.



SHATTOCK: Microscopic Structure of Uric Acid Calculi. Plate I.

ammonium; some calcium was present (combined with oxalic acid). The calculus is oval, and measures 7 mm. in the longer diameter. The nucleus, which is quite small, and does not fill the field under a $\frac{2}{3}$ obj., is a well-defined agglomerate of cuboidal crystals of medium size, closely compacted and without any order. To this succeeds the body, which is constructed of concentric laminæ interconnected by well-pronounced radial columns. The crystals in both positions are of coarse cuboidal character; though in the case of some of the columns there are massive spear-like forms set radially with respect to the centre of the calculus. The cuboidal elements are very closely fitted, without interspaces, and construct concentric zones and radial columns; they are individually of no mathematically regular form, and seem to have grown whilst in imperfect apposition until the intervals between them became completely filled.

The first deposit of calcium oxalate appears as a narrow, beautifully transparent, and colourless zone arising directly on the disorderly mass of uric acid crystals which form the roof of one of the arcades. The oxalate, as in all cases where it forms the body of a calculus (as distinguished from the nucleus) assumes the form of transparent, colourless cones lying in lateral apposition, and doubly striated—i.e., presenting a fine crystalline, divergent marking, and a cross striation almost as regular and delicate as that of a muscle fibre; the latter is quite independent of any interposed material.

Certain of the cuboidal uric acid crystals in this calculus, as in those previously described, exhibit a fine secondary cleavage. This zone of oxalate completely encompasses the concretion, although too fine for recognition by the naked eye. The outer border of the zone is undulatory, in correspondence with the bases of the cones constructing it; the undulations present, in addition, a secondary dentate or finely jagged edge resulting from the slightly irregular projection of the free ends of the tuft of crystals composing the cones. The calcium oxalate is followed by a zone of massive pale yellow uric acid crystals without any order, and like those composing the nucleus; in this zone interspaces are present, but there is scarcely any indication of a columnar disposition. This is followed by a second zone of calcium oxalate narrower than the first; then a zone of disorderly cuboidal uric acid crystals; then a third line of oxalate narrower than either of the others; and in places interrupted in its circuit by uric acid crystals. The body beyond this is built up in the same manner—viz., by alternating zones of calcium oxalate and broader zones of massive uric acid crystals presenting very little columnar disposition, and amongst which irregular lacunæ occur. Only here and there do the crystals produce an arcade, of which the pillars are made up of somewhat elongated massive forms set with an attempt at radial disposition.

In this calculus the scantiness of elongated uric acid crystals, either coarse or fine, is noteworthy.

Specimen IV (A 45), Museum of the Royal College of Surgeons.—Uric Acid, with Urate of Ammonium, and a trace of Urate of Sodium.

Of this group I examined four. The collection comprises several small pisiform concretions removed from a patient 65 years of age. They consist of uric acid, but though classed in this series in the College collection, they contain also urate of ammonium and a trace of urate of sodium. The uratic portions are readily differentiated microscopically, and constitute only a small proportion of each calculus.

Example A : Nucleus.—This consists of a circular compact group of cuboidal crystals of medium size, and others somewhat elongated, but devoid of any order; intercalated between them there are a certain number of more massive forms which exhibit the usual secondary cleavage. Irregular, less transparent patches occur in the general conglomerate, some of which are due to the presence of finely divided rod-shaped crystalline material, though others are due to a minute parallel cleavage of certain of the other crystals. The massive blocks above referred to are of very irregular shape with jagged borders; they are not sections of a series of crystalline columns radiating from the centre of the nucleus, for they are fewest in the centre of the section, where they would form a close mosaic were they cross-sections of an orderly set of columns arising from a central point which had not been accurately struck in the section. Moreover, they are comparatively few in the general mass of crystalline "rubble," and even at the periphery of the nucleus, where a few longer forms occur, these have no strict radial direction. The nucleus is somewhat sharply bounded by a narrow, opaque line of fine, rod-shaped crystalline material, which is intermingled with the coarser crystals without producing a complete interruption of structure. Beyond this the body consists, in the first place, of a zone of medium-sized cuboidal crystals, like those on the inner or nuclear aspect, and equally devoid of any arrangement. This zone supports an arcade of similar crystals, the component elements of its columns being either of the same character, or here and there elongated, coarse, and radially set, though with very little geometrical arrangement. A second arcade follows. In these wavy concentric collections of crystals, ill-defined, less transparent lines occur, which follow the general course of the zones, and are due to the engagement of the fine crystalline material so frequently referred to. At its periphery the structure of the calculus abruptly changes and becomes compact. This compact crust, which arises directly on the roof of an arcade, consists of long transparent, pale yellow crystals closely arranged, with a general, yet imperfect, radial direction, for they obliquely intersect. Bacilliform crystalline material lies between the elongated elements in very incomplete radial lines, and also as a concentric deposit, the opaque circular lines so resulting varying in their thickness, and the distances at which they lie from one another. It may be observed that these concentric lines or zones do not really interrupt the main crystalline structure, but that the finer deposit has been periodically laid down between the elongated elements, which thus perforate it, though the material is in addition more sparsely included within the other crystals. Immediately upon the cancellous portion of the body the radially disposed crystals of the compact crust are finer and shorter than those beyond, and the concentric lines of the still finer material are closer and narrower. The long uric acid crystals present none of the delicate parallel cross striæ which those of calcium oxalate so regularly do in microscopic sections of calculi of the last-named substance, though they exhibit a fairly fine secondary longitudinal cleavage. It is of interest to notice that this calculus shows the occurrence of multiple spontaneous fracture, followed by partial repair. The fractures involve the compact crust, which they traverse at right angles to the surface, and have been followed by a partial dislocation outwards of the more exterior portion of the crust by reason of the fracture having involved the periphery itself in the concentric direction. The resulting fissures and spaces have in different degrees become filled in with a deposit of spherular urate. The fractures have, moreover, allowed of the access of urine to the first of the subjacent interlamellar spaces, with the result that this space in the neighbourhood of the fracture is likewise lined with spherular urate. These spherules, which are perfectly characteristic, lie in lateral apposition, it may be in a single row, to line the cavity or faces of the gaping fissure, and in such circumstances they are not completely spherical, but fixed on the attached side by a broad base, being really hemispheres or larger segments of complete spheres. These spherules individually present a finely radially crystalline structure, the striæ reaching the true centre, although there is a more transparent central spot of more pronounced yellow colour. The light spot is not abruptly demarcated from the substances beyond, and appears to be due to the closer arrangement of the component crystals in this position, whilst further outwards a diminution in transparency is brought about by the interference to the passage of light occasioned by the oblique way in which the crystals intersect.

Example B: A Second Calculus, 7 mm. in diameter, from the same group—viz., A 45, Museum of the Royal College of Surgeons.—In this the nucleus is a sharply defined, compact sphere of long massive crystals which radiate from a small centre of closely set cuboidal crystals, and present no interspaces; in size it somewhat exceeds the field of a $\frac{2}{3}$ obj. It is crossed by four or five concentric lines of fine rod-like crystalline material, which produce a corresponding loss of transparency. The massive crystals present a secondary longitudinal cleavage, and some are fissured transversely (it may be with slight obliquity to the long axis) so as to resemble a series of superposed blocks or tablets. The lateral limits of these long crystals are seldom straight, but sinuous or with angularities. The nucleus is succeeded by a zone of cuboidal crystals, the deepest portion of it being rendered opaque by the presence of finely crystalline material; this zone supports a massive arcade with long stout columns; this arcade is followed by another of less dimensions. The crystals composing all these structures are mostly short or cuboidal, though here and there a long radially set element enters into the construction of the columns. The calculus is completed by a dense zone of compact substance built up of radially crystalline uric acid, with which the usual opaque bacilliform material is intermixed so as to produce concentric lines, the whole in every detail resembling the crust in the foregoing specimen. The exterior of the concretion is regularly invested with a thin zone of uratic spherules in single series, laterally apposed, and fixed by their bases to the surface below, the spherules being incomplete, or flattened, in this position. As in the preceding specimen, the compact crust presents a certain number of vertical fractures of which the faces are coated with spherules of urate, and the adjacent portion of the space beneath, so opened up, has become partly lined with similar spherules, or partly filled with pale brown, finely crystalline or amorphous urate, as a result of the access of urine which at this date was depositing such material.

A third and a fourth specimen of the same group show a construction corresponding with the other two.

Specimen V (A 63), Museum of the Royal College of Surgeons.—Uric Acid.

I selected this calculus in order to study the structure of one of the coarsest or most cancellated varieties. The specimen is of flattened spherical, or discoidal form, 3·5 cm. in chief diameter, and reddish-brown in colour. To the naked eye the divided surface presents a nucleus, not sharply defined, yet of somewhat different construction to the body, for it has a finely cancellous texture like closely cancellated bone, the trabeculæ being, however, without any definite disposition. The body, on the contrary, consists of extremely coarse radiating columns with wide intervening spaces, the columns themselves being irregularly interconnected, yet without sufficient regularity to produce a proper concentrically laminated architecture. The nucleus merges into the body, without there being any sharp line of demarcation between the two. The microscopic sections were made to include the nucleus and the whole of the body.

The nucleus, which is about 7 mm. in diameter, consists of pale yellow cuboidal crystals agglomerated, without any order, into trabeculæ with intervening spaces. The trabeculæ have no definite arrangement, though the spaces bounded by them tend to a polyhedral shape, the whole being much like a section of cancellous bone.

The coarse radial columns, which constitute the salient feature of the body, are irregularly connected at the sides, but not in any strictly concentric manner. In the vicinity of the nucleus the columns consist of crystals like the trabeculæ of the latter, the one structure passing directly into the other. More outwardly the trabeculæ attain what is, relatively speaking, a gigantic size, and may be designated compound in structure. They present a central core of disorderly cuboidal crystals, but upon the sides of this there arise compactly arranged crystalline cones which are set at right angles to the long axis of the core, and in all ways resemble those which ordinarily constitute the body of the compact variety of calculus. These compactly arranged columns are secondarily subdivided in their long axis, and some show a fine cross striation independently of the presence of included finer material. In the larger columns, central lacunæ occur in the core of crystalline "rubble" as they do in the proper nucleus. The periphery of the calculus is formed like the compact variety in general. This results from a change in the direction of the crystalline columns, which are set not only on the sides of the central core, but, where the latter terminates, arise in a fan-like way

EXPLANATION OF PLATE II.

FIG. 7 (A 1, Museum of the Royal College of Surgeons).—A more highly magnified portion of the preceding zone, showing the elongated crystals and the intermingled finely crystalline uric acid. Repeated critical chemical examination proved that the material in question is uric acid, as no base was detectable. The finer crystals are, moreover, traceable by gradations into the larger.

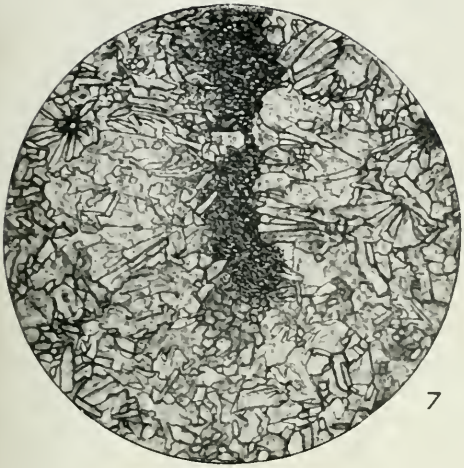
FIG. 8.—Cancellous variety of uric acid calculus (2 in. obj.), showing the small oval nucleus (about 1 cm. in the longer axis in the photograph) composed of closely agglomerated cuboidal crystals, succeeded by a body constructed of concentric laminæ interconnected with radial columns.

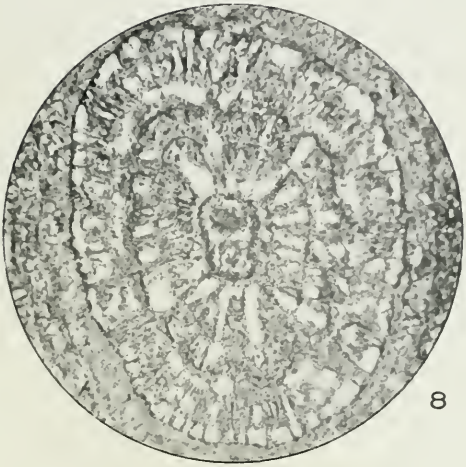
FIG. 9.—Portion of the body of the same calculus ($\frac{2}{3}$ obj.), showing the concentric laminæ interconnected with radial columns. The crystals in both positions are of coarse cuboidal character, though in the case of some of the columns there occur a few massive spear-like forms, set radially with respect to the centre of the calculus.

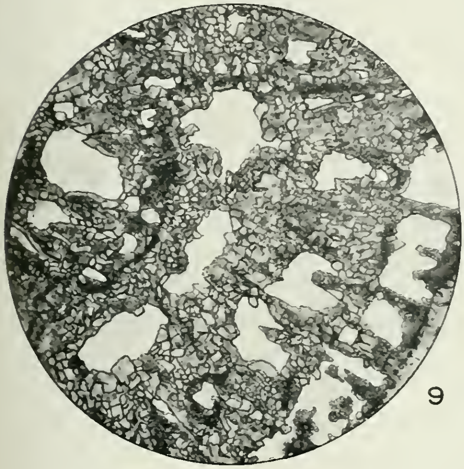
FIG. 10 (A 45, Museum of the Royal College of Surgeons).—Cancellous variety of uric acid calculus ($\frac{2}{3}$ obj.), showing, on the left, a portion of the spherical nucleus consisting of cuboidal crystals. This is bounded by a wavy line of fine bacilliform crystals which render the structure opaque. Beyond this the body consists of arcades of crystals similar to those of the nucleus. Here and there a huge spear-shaped crystal aids in the construction of the columns of the arcades. One such crystal (of less transparency than the cuboidal) is shown in the middle column close beyond the nucleus.

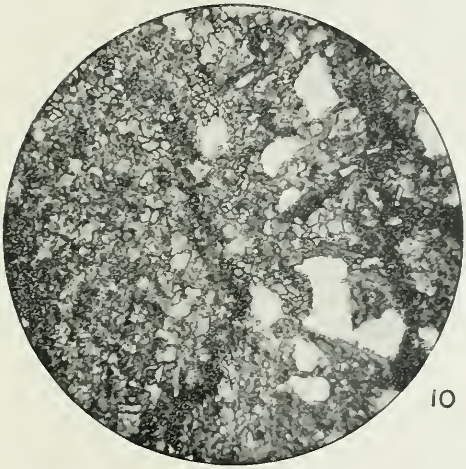
FIG. 11 (A 45, Museum of the Royal College of Surgeons).—Showing the particular column ($\frac{1}{6}$ obj.) referred to in the preceding description. It consists of cuboidal crystals, but on the upper border it is constructed of a huge spear-shaped element set radially with respect to the centre of the calculus.

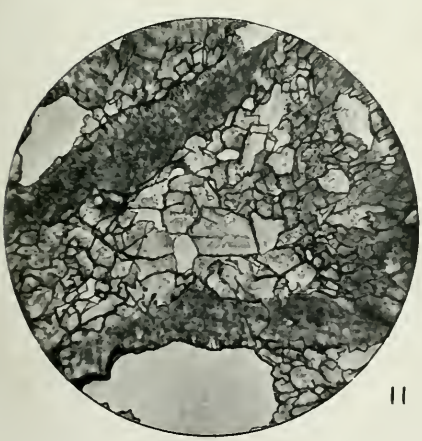
FIG. 12 (A 63, Museum of the Royal College of Surgeons).—Cancellous variety of uric acid calculus (2 in. obj.), part of the body of an extremely coarsely cancellous uric acid calculus. (The nucleus consisted of irregular trabeculæ of cuboidal crystals with intervening spaces.) The body is constructed of coarse, radially set columns, which are connected laterally, but not in very strictly concentric manner. The columns consist of a central core of irregular or cuboidal crystals (with interspaces), upon the sides of which there are compactly arranged, coarse crystalline cones set at right angles to the long axis of the column. The dark areas are due to the presence of more finely divided crystalline material.

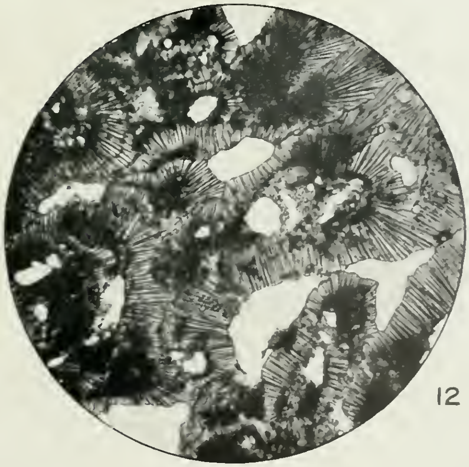

7


8


9


10


11


12

SHATTOCK: Microscopic Structure of Uric Acid Calculi Plate II

upon its end. These fan-like masses lie in lateral contiguity, but without necessarily coalescing, for the line of their apposition remains plainly discernible.

A certain amount of finely divided material is irregularly distributed through the calculus. This is especially abundant where the long columns arise on the sides and summits of the central core of crystals.

THE COMPACT VARIETY.

Let me next pass on to the compact variety of uric acid calculi. In these no spaces whatever are visible, either to the naked eye or in microscopic sections, but the calculus is built up throughout of radiating crystalline cones starting from a centre, in some cases without a differentiated nucleus, the whole being simply a gigantic crystalline sphere. Even large concretions may retain this structure throughout their formation.

Specimen VI.—Uric Acid.

As examples of small size I may take some from a collection of about the dimension and form of lentils, of which I examined five, and of which the description of one may suffice.

Specimen A.—Under a low power the section shows throughout a compact structure of massive radiating columns, without interspaces, there being no differentiated nucleus. As studied under a higher power ($\frac{1}{6}$ obj.) these gigantic columns are regularly and closely fissured in the transverse direction. The fissures of adjacent columns do not exactly correspond, for as they are directed across the long axes of the latter, and as the columns are not geometrically radial in arrangement, the cross fissures do not produce a continuous concentric striation. The cross cleavage differs from the cross striation of calcium oxalate in that there are distinct interspaces, or true clefts, in the structure. The clefts are not geometrically parallel but are slightly wavy, and moreover they may not traverse the entire breadth of the column. The thin plates may be themselves subdivided or broken up in the radial or longitudinal direction, though without any regularity, the substance of the column consisting then of superimposed series of minute flattened, or less regular elements, like the bricks in a Roman wall. In the midst of the compact substance there appear at intervals the pointed outer ends of radially set columns, the further growth of which has, for some reason, ceased; these are regularly fissured in the transverse or concentric direction, but they present, in addition, a much coarser lamination parallel with the pointed end, somewhat like the concentric markings in the enamel of a tooth. The markings described indicate that the growth of the crystal has occurred by additions to its pointed end, which has become ensheathed in a succession of superimposed deposits, and that the end of the crystal has freely projected from the growing surface. The uniformity of the construction is only disturbed by the interposition of a few narrow, wavy, concentric zones of finely crystalline material. These zones occur at comparatively wide distances, and are mostly incomplete or fragmentary, appearing only for short stretches of the entire circumference, or even only on the rounded or flattened summits of certain single gigantic radial columns. The finely crystalline deposit is nearly always immediately followed by a single series of fan-like sprays of coarser crystals, of singular transparency, and devoid of the pale yellow colour of the surrounding material; some of these crystals are arranged in rosettes. These crystals can be exactly matched in calculi of calcium oxalate, and may be taken as consisting of this substance. On the exterior of the longer stretches of this composite kind the columnar structure of the calculus takes a new departure, as shown by the pointed inner or centralward ends of the radial crystals which succeed. But where the stretches are short, and occur on the summit of a massive cone, the flat, growing adjacent columns simply continue to grow up and coalesce over it. Patches of the finely divided material occur fairly frequently, embedded or included in the substance of individual columns, and this associated or not with the larger uncoloured fan-like groups already described. The want of interference in the construction of a column by these lesser inclusions is remarkable, for on either side of it the transverse fissuration of the huge crystal retains its parallelism, the ends of the thin plates simply abutting on the included material. Some of the radial columns are less transparent than others, although of the same form, and regularly intercalated amongst them. This is due not to the inclusion of finer material but to the obliquity of their section, the cross fissures being viewed sideways instead of directly. This difference in transparency can be recognized in the section even by the naked eye. In the immediate vicinity of the geometrical centre the picture is to a slight extent confused by the fact that here and there the massive radially set cones happen to be divided obliquely or transversely. The surface of such a cone is divided into a series of irregular polyhedral segments corresponding with those seen in the longitudinal views.

Specimen VII (A 196), Museum of the Royal College of Surgeons.—Uric Acid.

As a second example of the compact variety I may give the results of the microscopic examination of three of the uric acid group A 196, Museum of the Royal College of Surgeons. Mr. Le Sueur was unable to discover, on careful analysis, any ammonium, sodium, or calcium.

The entire structure of these small pisiform concretions is remarkably regular and compact. There is a certain nuclear differentiation brought about by a change in the size of the elements, rather than in their character. The central part, or nucleus, consists of coarse crystalline wedges of pale yellow colour, delicately cross fissured, and including in their substance a certain amount of less transparent finely crystalline material. The latter is irregularly distributed throughout the substance, but is more particularly aggregated in ill-defined concentric lines or narrow zones. The columns are not actually continuous, for the jagged lines of their lateral apposition are easily to be recognized. The individual columns are, moreover, compound, or cleft in the radial direction, but this in a coarse manner as compared with the transverse ; the lateral limits of the subdivisions are lowly undulatory, the general appearance being that of a closely pressed bundle of twigs, none of the individual elements of which are geometrically straight, though they are so closely apposed that there are no intervals between them. This method of construction is brought to a close by the deposition of a more abundant shower of the bacilliform crystals. Upon this the structure takes a new departure, in the form of a second series of massive cones, though these are not individually so gigantic as those which construct the nucleus. The finer opaque material extends for a short distance outwards between the pointed inner end of these cones. The general structure is itself like that already detailed, the cones being subdivided radially, and in a finer, more minute and regular way transversely. Opaque material is included in the whole of the structure, more particularly in narrow and not very sharply defined concentric lines. This construction obtains almost to the surface, its compactness being maintained by the intercalation of new cones between the the diverging, more outward parts of the deeper. Near the exterior a diversion of the general structure is brought about by a somewhat abundant finer deposition. This is followed by a concluding crust of the general construction, the uric acid taking a new departure here, in the form of a further series of cones, between the pointed ends of which, extensions of the opaque

EXPLANATION OF PLATE III.

FIG. 13.—Compact variety of uric acid calculus ($\frac{1}{6}$ obj.), showing the structure of the body of a compact uric acid calculus. It consists of long, radially disposed, crystalline cones, without interspaces, the lateral limits of which are readily recognizable. The cones are regularly fissured at right angles to their long axis. The actual fissures are of secondary and artificial formation, though they indicate that growth has taken place by periodic addition.

FIG. 14.—Portion of the body of the same calculus, less highly magnified ($\frac{2}{3}$ obj.), showing the long, crystalline columns composing it. The darkness of some is due to the obliquity of their section, the cross-fissures, in oblique view, interrupting the passage of light. In the midst of the compact substance there appear, at intervals, the pointed outer ends of certain radially set columns the further growth of which has ceased. The columns in question are regularly fissured in the transverse direction, but they present, in addition, a coarse lamination parallel with the angular end, indicating that the growth of the crystals has occurred by additions to the apex, which has become ensheathed in a succession of superimposed deposits.

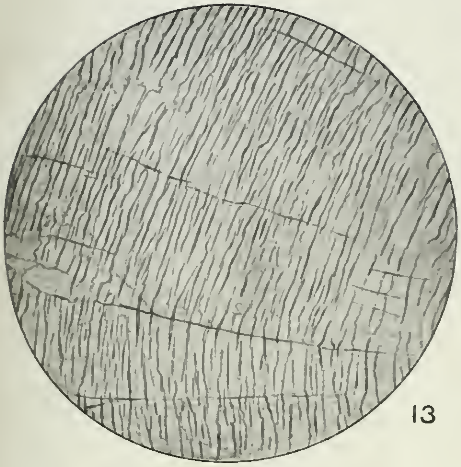
FIG. 15.—Portion of the body of the same calculus ($\frac{1}{6}$ obj.), showing the interposition, in the midst of the body, of a vertical and fan-shaped series of transparent crystals of calcium oxalate, set upon a subjacent deposit of finely crystalline material. Both formations have occurred on the growing surface of a transversely fissured column, which radiates from the more central portion of the calculus. These deposits of oxalate are incomplete, or cover only parts of the circuit of the calculus.

FIG. 16 (A 196, Museum of the Royal College of Surgeons).—Compact variety of uric acid calculus (2 in. obj.), showing the entire concretion. Although the composition is throughout of uric acid, a small nucleus is differentiated from the rest; this is composed of huge crystalline cross-striated wedges radiating from a central point, and intermingled with finely crystalline opaque material. The nucleus is terminated by a zone of similar material. Upon this the body takes its departure in the form of a second series of smaller fibrous cones. The compact mass is finely cross-striated, and in addition rendered opaque by coarser striæ of finely crystalline material, deposited at intervals on the surface; a broad zone near the exterior is particularly dark from this cause.

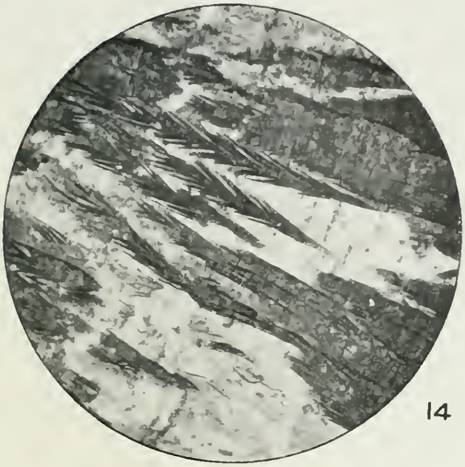
FIG. 17 (A 196, Museum of the Royal College of Surgeons).—Showing the deepest part of the preceding calculus as far as its centre ($\frac{2}{3}$ obj.). Although the composition is throughout of uric acid a small nucleus is differentiated from the rest; this is composed of huge crystalline, cross-striated wedges, radiating from a central point, and is intermingled with finely crystalline material. The nucleus is circumscribed by a zone of similar material. Upon this the body takes its departure in the form of a second series of smaller cones. The compact mass is finely cross-striated; and is, in addition, rendered opaque by coarser striæ of finely crystalline material deposited at intervals on the growing surface.

FIG. 18 (A 196, Museum of the Royal College of Surgeons).—The outermost portion of the body of the same calculus ($\frac{2}{3}$ obj.), including its free surface. The general structure is interrupted near the surface by a belt of finely crystalline material, in connection with which there has been an abortive attempt to reproduce the original construction, as appears by the presence of the pointed inner ends of stunted massive, transversely fissured cones, like those forming the nucleus. Upon this belt the final crust arises as the body itself does upon the nucleus.

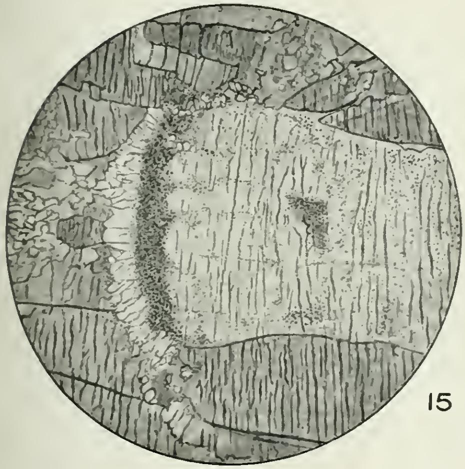
PROC. ROY. SOC. MED.
Vol. IV. No. 4.
Pathological Section.



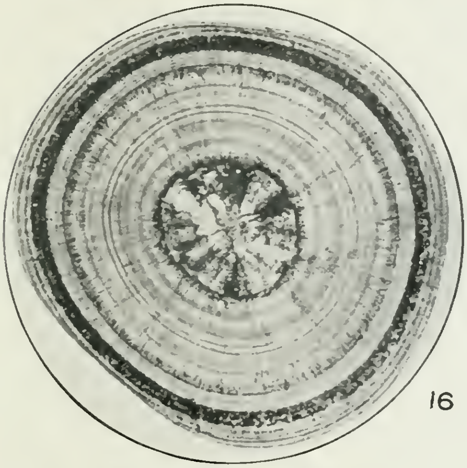
13



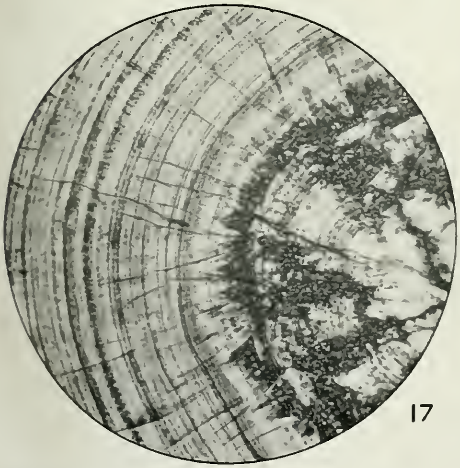
14



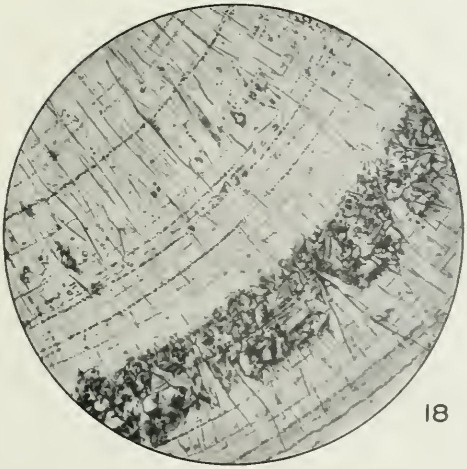
15



16



17



18

SHATTOCK: *Microscopic Structure of Uric Acid Calculi.* Plate III.

material lie. Immediately beyond the finer deposit there has been an attempt to reproduce the original construction, as seen by the presence of the pointed inner ends of stunted massive, transversely fissured cones like those forming the nucleus of the calculus. These, however, are short and fragmentary, largely intermixed with the finer substance, and at last covered in by it. Upon this the final formation of properly developed and more slender cones arises. The growing surface itself is quite smooth, the free ends of the growing radial crystals producing scarcely a recognizable undulation.

A second and a third specimen from the same group show precisely the same structure, the several differences in each being accurately represented in the rest, proving that the calculi have grown synchronously.

Specimen VIII (A 107), Museum of the Royal College of Surgeons.—Compact Variety of Uric Acid Calculus.

As an example of a compact calculus of more considerable dimensions, I may select one of a small group, A 107, in the Museum of the Royal College of Surgeons. The calculi are of regular pyramidal form, and measure about 1·5 cm. in chief diameter. On allowing the half of the calculus to dry, the body spontaneously split into three wedge-shaped pieces, and a minute oval nucleus, about 2 mm. in its longer diameter, was detached. In the centre of this nucleus there is a fine, slit-like space, arising doubtless from the loss of the most central crystals during the preparation and washing of the section.

The nucleus consists of long, radially set columns, subdivided in the radial direction, but without cross-markings, save here and there in the most central parts. In the substance of the closely compacted component crystals are a certain number of concentrically arranged, ill-defined zones of finely divided material, and similar material occurs, also, radially disposed between the crystals, though without any regularity or uniformity; and, more sparsely, it is distributed throughout their substance. None of these columns reach the true centre. They arise in a regular way upon an oval collection of pale yellow cuboidal crystals, the chance aggregation of which has formed the beginning of the calculus, and in this sense constitutes its real nucleus. In the centre of this collection there is a narrow, slit-like space which has doubtless arisen from the loss of the loose central elements in the process of grinding and washing the surface of the section before it was cemented to the slide. The detachment of the central sphere of the calculus

EXPLANATION OF PLATE IV.

FIG. 19 (A 107, Museum of the Royal College of Surgeons).—Compact variety of uric acid calculus ($\frac{2}{3}$ obj.), showing the central part of one of the group. The calculus has the usual structure of closely compacted cones or brushes of crystals without interspaces, the whole substance being striated both radially and concentrically. The cones arise upon an oval group of cuboidal crystals, which serve as a nucleus, the most central portion of which has been lost in the preparation of the specimen. The central part shown was spontaneously detached during the course of preparation; this is due to the presence of a zone of cuboidal crystals, portions of which are shown in the figure. The calculus beyond was a reproduction of the main bulk of that represented.

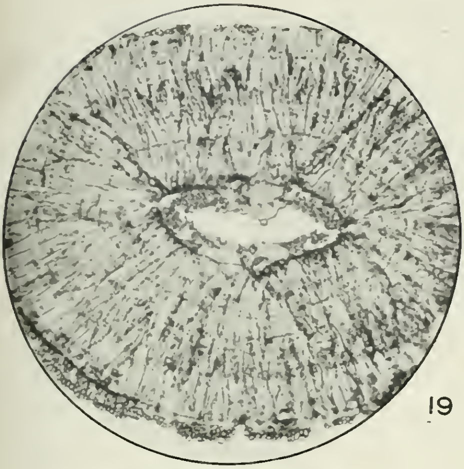
FIG. 20 (A 107, Museum of the Royal College of Surgeons).—Compact variety of uric acid calculus ($\frac{1}{6}$ obj.). A typical portion of the body of another calculus of the same group, showing the close, long crystals set in the radial direction, and a marked amount of the finely crystalline and less transparent material deposited at intervals upon the surface during its growth.

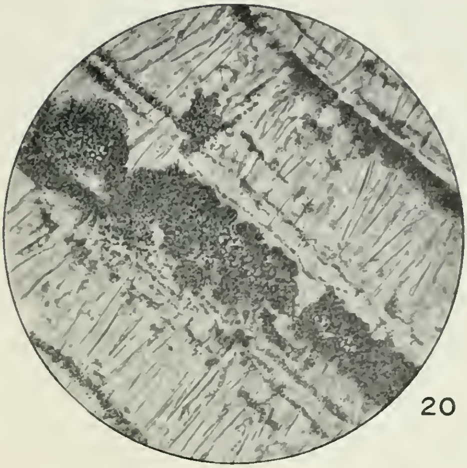
FIG. 21 (A 107, Museum of the Royal College of Surgeons).—Compact variety of uric acid calculus ($\frac{1}{6}$ obj.), showing the growing surface of a third calculus of the same group as the preceding. The long, radial crystals reach the exterior; these are crossed at irregular intervals by opaque, ill-defined lines of finely crystalline material, which lies partly in the interstices between them, so that the radial elements may be traced, in places, through the opaque concentric zones.

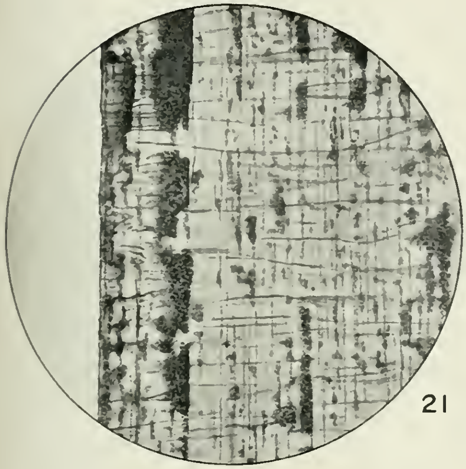
FIG. 22.—Compact variety of uric acid calculus ($\frac{2}{3}$ obj.), showing a centre of huge cross-striated cones, succeeded by closely packed crystals of smaller, shorter forms without radial disposition.

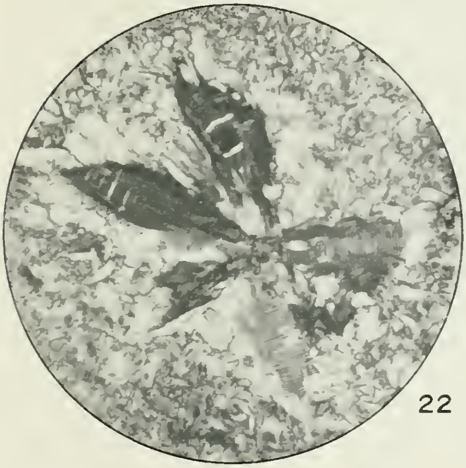
FIG. 23.—Compact variety of uric acid calculus ($\frac{1}{6}$ obj.), showing the actual centre of the preceding section. The cross-striated cones arise upon a small collection of shorter forms which serve as a nucleus.

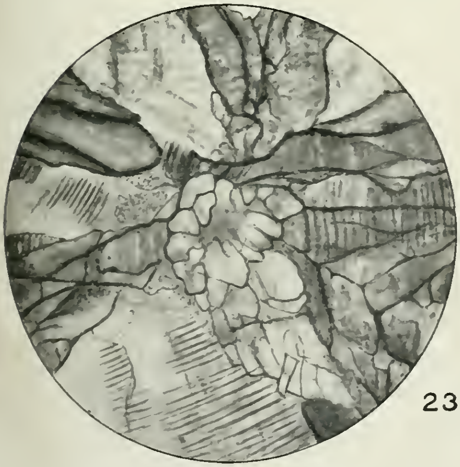
FIG. 24.—Showing the periphery of the preceding ($\frac{1}{6}$ obj.), which is composed of an aggregation of shorter crystalline forms; many present the elongated, whetstone shape.

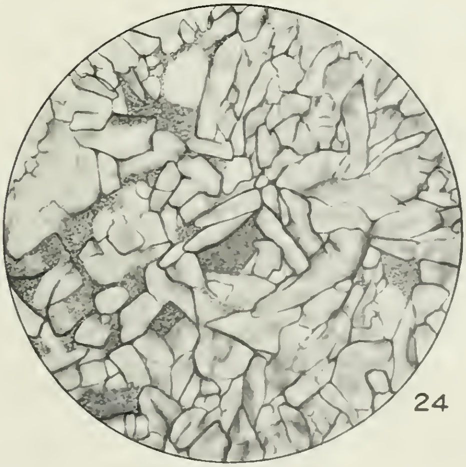

19


20


21


22


23


24

SHATTOCK: Microscopic Structure of Uric Acid Calculi. Plate IV.

during the drying of the hemisection is readily explained by the presence of a zone of similar cuboidal crystals on its exterior. I may notice, in passing, that in the course of preparation of a second specimen of the same group a minute central sphere was similarly detached, and for a like reason.

The section of the more peripheral part of the calculus, made so as to include the growing surface, consists throughout of radiating crystals of considerable length, becoming finer as the exterior is approached; these are crossed by concentric lines or zones of less transparent, included, material, these lines becoming more closely set towards the free surface so as to produce a comparatively fine lamelliform structure; there is, in addition, a finer cross striation due to cleavage, apart from the presence of the other material. The actual growing surface is constituted by the ends of what are here the finer crystalline columns, and is minutely irregular in correspondence, although to the naked eye and touch it would be called quite smooth.

Specimen IX.

I may next describe a minute calculus (from a private source) of compact, but somewhat irregular, construction as compared with the foregoing. The nucleus consists of a stellate group of coarse crystalline and transversely fissured cones, as in certain of the foregoing specimens. Some of the cones present a certain degree of opacity arising from the minute subdivision of the thin superposed horizontal or transverse plates of which they are made up. Beyond this, the calculus is made up of a disorderly collection of rhombic or whetstone crystals, with a certain amount of less transparent, finely divided material. Here and there a fan-shaped, radially striated element of calcium oxalate is to be encountered; some of these are striated crosswise as well as radially. The periphery of the calculus is formed by the reappearance of cross-striated cones like those of the proper nucleus. Examination with a higher power shows that the nuclear cones radiate from a small cluster of cuboidal or rhombohedral crystals like those which form the chief part of the body.

Specimen X.

In this case the compact calculi, for which I am indebted to Dr. L. S. Dudgeon, are remarkable for their smallness. I have examined four. They are all spherical, and considerably smaller than pins' heads.

In minuteness they can be matched by the smallest among the "miliary" calculi of calcium oxalate. Of the latter there is an excellent collection in the College Museum; the calculi, many thousands in number, were taken from the pelvis of a hydronephrotic kidney; they are quite spherical, the smallest being almost microscopic.

In both these cases the structure of the calculi is a miniature of the larger. The most minute of the oxalate consist of a transparent, regularly concentrically striated body, deposited upon a nucleus of irregular form, which may consist of one or more gigantic, ill-shapen octahedra, or of a cohering group of oxalate spherules. Both forms of nuclei are to be encountered in the larger calculi of calcium oxalate.[1]

The miliary uric acid calculi under consideration are too small to allow of sections being made, and their construction can be discerned without, after the material has been treated with absolute alcohol, xylol, and mounted in balsam; yet I examined some by grinding them away from one side only. They consist of long, coarse, pale yellow crystals, radially set so as to produce a compact sphere. The growing surface of the sphere, however, is not smooth, as it is in the case of calcium oxalate, but regularly jagged from the projection of the free ends of the crystals.

SUMMARY AND GENERAL CONSIDERATIONS.

The study of these calculi by means of microscopic sections confirms of course, in the first place, the macroscopic observation that some have a porous or cancellated structure; others a compact, without interstices. This difference depends upon a difference in the method of crystallization. In the cancellous the deposition, in general, departs but little from certain of the forms of sediment.

The Cancellous Variety.

To summarize this variety first: The calculi present a *nucleus* of crystals which may be designated as cuboidal—i.e., they are not of elongated form, or tabular. These crystals are almost invariably aggregated so as to leave lacunæ, but neither the trabeculæ nor the interspaces have any definite or regular form. The nucleus may aptly be described as crystalline "rubble" upon which the body of the calculus is built. Embedded in it there are sometimes more massive crystalline blocks which present parallel striæ arising from periodic growth or from secondary cleavage. Crystalline material which, from the minute size and rod-like character of its elements, may be called "bacilliform," is intermingled in varying amount with the crystals forming the trabeculæ; the minute elements composing the material have no kind of orderly disposition, but are simply felted together, as it were, in ill-defined patches and lines. The presence of this finely crystalline substance markedly reduces the transparency of the section by reason of the manner in which it interferes with the passage of light; except in particularly thin sections, it is almost opaque.

---

[1] W. M. Ord and S. G. Shattock, *Trans. Path. Soc. Lond.*, 1895, xlvi, p. 91.

The *body*, in the simplest forms, is constructed of concentric laminæ, of similar, though smaller, crystals, and, like those of the nucleus, they are intermingled with the more finely crystalline material. These laminæ do not lie in apposition, but are interconnected by columns of different height (or length) in different cases, so as to construct a series of arcades. Sometimes the laminæ are regularly undulatory, the depressions between the undulations corresponding with the sites of the interconnecting or radially set columns. The intervals between are incompletely filled with the coarser cuboidal kind of crystals.

In regard to the columns of the arcades, these vary considerably as to radial height and thickness in different calculi. They may consist simply of cuboidal crystals like those of the concentric laminæ or the roofing of the arcades; or coarse, spear-like forms of crystal, may enter into their construction, these being radially set with respect to the centre of the calculus as a whole. During the progress of growth, it will be clear that the columns, until the roofing-in of an arcade, must have grown up with very little support; and, remembering their delicacy and want of fibrous structure, it is not easy to explain why they have not been fractured and detached in the building. One must assume, I think, that the mucin in the urine has formed a supporting investment to the growing surface. In the most coarsely cancellous kinds, the interconnecting radial columns are particularly massive, and present a structure which may be described as compound. Such columns consist of a core of cuboidal crystals, upon the sides of which core long coarse crystals are compactly set at right angles to its axis; and where the core terminates, these are arranged in a compact, fan-like manner upon its summit.

The periphery of the cancellous variety has usually the structure of the compact kinds, and consists of long, radially set crystals closely arranged, and with varying amounts of finely crystalline material which has been periodically deposited in ill-defined concentric zones during the growth of the longer crystals. The growth of the latter is not necessarily interrupted by this deposition, which lies chiefly in the lateral interstices between them, so that the elongated elements may perforate or traverse several such sedimentary layers. This material may also be deposited between the crystals, in the radial direction, at least for short distances; and lastly, in less amount, it is included in the crystals themselves. Zones of this compact structure may again be followed by the open system of arcades; and these, again, by others like the first. Laminæ or zones of a transitional or intermediate character may be encountered. These are constructed of elongated crystals of different lengths, but without any proper radial disposition; coarse rosettes may occur in the midst of such crystalline collections.[1] And it may even happen that a zone in part of its circuit is of well-formed, compact structure—i.e., of long, close-set radial crystals, whilst elsewhere it is composed of similar crystals without the radial order.

The actual surface of the final, compact, encrusting zone is formed by the free, growing ends of the elongated elements, but even under $\frac{1}{6}$ obj. it is almost smooth, or curvilinear, though it may be slightly jagged from the projection of the ends of the individual crystals. I have not yet examined any cancellated calculus in a stage of growth at which it had not acquired a compact crust.

The Compact Variety.

In this form the construction is of a more regular and more highly differentiated kind. In place of an aggregate of cuboidal crystals with interspaces the structure consists of long radially disposed crystals or conoidal columns, the more central of which may be of gigantic size. In the more peripheral parts of the calculus the radial crystals, though compactly arranged, are as a rule more slender. The compactness of the growing concretion is maintained by the intercalation of further crystals between the diverging outer ends of those earlier formed. The more massive cones are finely fissured at right angles to their long axis, so that when the crystals are not mathematically radial the fissures of the apposed columns do not correspond in such a way as to be continuous. This cross cleavage is quite independent of any periodical sedimentary inclusion. Nor is there a true continuity of the radiating columns themselves, as is evident in the presence of a dark, often zigzag, border which marks their lateral apposition. This cross cleavage is absent in the zones or parts constructed of the more slender elongated crystals. The thin resulting plates may be subdivided or broken up in the radial or longitudinal direction, though without any regularity, the substance of the column consisting, in this case, of a superimposed series of flattened elements like the bricks in a Roman wall. Both directions of cleavage are at times observable together in the larger crystals of uric acid sediments. The phenomenon is well represented in Rieder and Delépine's Atlas (Pl. xi, fig. 1). In sedimentary crystals of uric acid the cleavage is stated to be especially noticeable when ammoniacal fermentation has taken place. As the calculi in question, however, exhibit no phosphatic admixture, this association is obviously not an invariable or necessary one. The fissures themselves may be attributed to artificial causes, but their direction is determined by the intimate constitution of the crystalline substance. The differences in size of the radiating crystals indicate differences in rate of growth, the more slowly growing forms being the coarser. Equally striking differences in size may be observed in individual sedimentary crystals. I have gigantic macroscopic crystals of ammonio-magnesium phosphate which were allowed to grow for a prolonged period in ammoniacal urine.

---

[1] Such rosettes, as met with in uric acid sediments, are well figured in Rieder and Delépine's "Atlas of Urinary Sediments," 1899, pl. vi, fig. 3.

As to the nucleus, in some instances the crystalline columns or cones take their point of departure upon a minute—i.e., microscopic—nucleus of cuboidal crystals; in others, they are in central apposition.

As in the cancellous varieties, finely crystalline material is invariably intermixed, chiefly in narrow concentric zones—i.e., periodically deposited on the surface during the course of growth. It occurs also, less regularly disposed, in the radial direction, in interstices which have existed between the growing ends of the radiating crystals. The concentric zones of this material by no means necessarily interrupt the growth of the longer crystals, which often pass through many such zones. In the more slowly growing gigantic columns the material may be deposited as transverse inclusions which do not destroy their continuity, and more sparsely it is often scattered throughout the whole of the crystalline substance.

A Comparison of the Crystals forming Calculi, with Uric Acid Sediments.

Most of the elements in the cancellous variety may be matched by those occurring in urinary sediments. The large cuboidal forms or thick, four-sided plates are well illustrated in the atlas of Rieder and Delépine (Pl. xxii, fig. 6), and in Beale's "Kidney Diseases and Urinary

Deposits" (3rd ed., 1869, Pl. xxvi, fig. 146). Elongated crystals such as form the compact zones of the cancellous variety are, however, very seldom encountered in uric acid sediments, the spear-shaped forms resulting from the elongation of the acute angles of the uric acid rhomb (the fundamental type of the deposit) being very rare. A highly pronounced example of elongated uric acid in my collection (from the sugary and albuminous urine of a case under the care of the late Dr. W. M. Ord) shows that some forms result as above stated, but others are due to secondary spinous growth occurring on the pointed ends of rhombs, the limits of which can be recognized in the crystalline substance: sometimes the elongation affects only one end of such a rhomb. Examples of the last will be found in Pl. xxix, fig. 1, Rieder and Delépine, from a urine (strongly acid) in a case of calculous pyelitis.

It is noteworthy that spear forms occur in strongly acid urines, whilst the whetstone and barrel forms are more frequently met with in urines of less acidity. The elongated forms entirely constitute the compact, as distinguished from the cancellous, variety of uric acid calculi. It would seem, therefore, that the differences of structure in the two depends upon the degree of urinary acidity. This I will again refer to under the cause of crystalline elongation.

The study of uric acid calculi has shown that none are composed solely of the commoner microscopic elements of uric acid sediments, even when the spear form is included. An admixture of finely crystalline material is always present, both in the cancellous and the compact varieties of the calculi. This material, as already described, is deposited between the elongated crystals, as well as between the cuboidal crystals of the nucleus, and of such concentric laminæ as consist of them; and by its interference with the passage of light it produces a marked opacity in those parts of the section in which it is distributed. At the outset I assumed that it consisted of urate, either of ammonium or sodium, until the contrary belief was forced upon me: urate of calcium is to be met with in calculi, as it is in urinary sediments, but this salt is so distinctive in character that it may be excluded on microscopical grounds, as well as chemical.

The substance under consideration is not granular, but consists of fine, short, rod-shaped crystals, which might be named bacilliform; and they are invariably coloured. Beale (loc. cit.) states that ammonium urate crystallizes in delicate needles only if artificially prepared, since the slightest trace (Bence Jones) of sodium chloride causes the salt to assume the amorphous character, and increases the solubility of the urate by one-half. He figures (Pl. xviii, figs. 98 and 100, loc. cit.), such crystals of ammonium and of sodium urate, so prepared, in order to contrast them with the natural spherular and amorphous forms. This assertion, however, is too exclusive. Short and finely crystalline acid ammonium urate is figured by Rieder and Delépine, arranged in irregular dumb-bells and stellate groups (Pl. vii, fig. 5, loc cit.), and as a profuse deposit in stellar and less regular groups, from a specimen of ammoniacal urine (Pl. xxii, fig. 2). Acid sodium urate in rare cases may take a like form. Sheaves and Maltese crosses of short fine crystals of this salt are represented in the more recent work of Daiber, "Mikroskopie der Harnsedimente," 1906 (Tab. v, fig. 9); and by Ultzmann and Hoffmann, "Atlas der Harnsedimente" (Pl. ix).

This finely crystalline material is present, and in conspicuous amount, in all the sections of uric acid calculi which I have studied. That it is really uric acid appears (1) from the complete absence of ammonium as shown by the special analyses made by Mr. Le Sueur, of calculi of which the microscopic sections showed its presence in noteworthy proportion; (2) from the observations that in some zones of these particular calculi, all transitions are traceable between the longer uric acid crystals and the finely crystalline intermingled material; (3) from the circumstance that fine crystalline sediments of uric acid are not altogether unknown in the urine. Forms as fine as those in question are figured, in pale yellow sheaves and Maltese crosses, by Rieder and Delépine (Pl. ii, fig. 6), and by Daiber, in similar, orange yellow clusters (loc. cit., Tab. iv, fig. 7).

Beale (loc. cit., p. 195) avers that uric acid may be so abundant in solution, in urine, that a drop of nitric acid will cause a dense amorphous precipitate like albumin; the amorphous particles, after a time, "increase in size and become characteristic." A similar observation is made by Rieder and Delépine (p. 32), but a different and doubtless more correct interpretation is placed upon the precipitation—viz., that the precipitate, which re-dissolves on heating, and reappears on cooling, is of urate, and not uric acid. Beale himself, indeed, states (p. 195) that if the ordinary amorphous deposit of urate is merely dissolved by warming, the urate often becomes decomposed, and as the urine cools, crystals of uric acid are deposited. This is what appears to have happened in the case of the amorphous precipitate which he took to be one of finely divided uric acid. The possible explanation of this marked contrast in the size of the uric acid crystals I will refer to later on.

The presence of vertical fractures in three of the calculi of one of the groups (A 45, Museum of the College of Surgeons) is worth notice in passing. Examples of spontaneous fracture have been recorded by Dr. W. M. Ord[1] and others, but these are nearly all of them fractures attended with an actual detachment of fragments, by which, indeed, the occurrence was in most cases recognized. The incomplete fractures or fissures in the calculi under notice would hardly be discoverable, except in microscopic sections. The explanation of spontaneous fracture advanced by Dr. Ord was that it is due to the swelling of mucoid material entering into the composition of the calculus, the swelling being brought about by reduced acidity, or by alkalinity of the urine. Spontaneous fracture has not been observed macroscopically in calculi of calcium oxalate; nor have I ever seen it in any of the many microscopic sections of such calculi which I have prepared. It is noteworthy that the fissures, which involve only the compact crust, are filled with spherular urate, and that this has been deposited at the same time as a narrow zone on the exterior of the calculus. For it indicates that the deposition of urate coincided with an alteration in the reaction of the urine of the kind supposed by Dr. Ord, and that this alteration led to the fractures, the faces of which became thereupon coated with spherular urate which was then being deposited; the deposition was, of course, in no sense a reparative process induced by a prior occurrence of the fracture. In these calculi there is a system of open arcades beneath the compact crust, the swelling of any organic material in which might easily lead to the fracture of the superjacent investment.

The Nature of the Nucleus of Uric Acid Calculi.

In the genesis of urinary calculi the nature of the nucleus has always been a question of particular interest. It has commonly been supposed that some adventitious substance is necessary to serve as a starting point, whether shed epithelium, mucus, blood-clot, bacterial colonies, or foreign bodies strictly so called—i.e., bodies introduced from without. It is, of course, beyond dispute that a foreign body may form the nucleus of a concretion—i.e., may serve as a centre on which crystallization may proceed, although its presence cannot determine crystallization, except in a supersaturated solution. When uric acid or urate is concerned, the presence of such a nucleus is altogether exceptional. There is only one example of it in the great collection of the Royal College of Surgeons. The calculus in question consists of such impure uric acid, and its microscopic structure, even immediately upon the foreign body, is so typically uratic, and not uric, that I may defer a description of it until dealing with the subject of uratic calculi.

---

[1] "Influence of Colloids upon Crystalline Form" (1879, p. 93), where references are given to cases previously observed.

The study of the centre of uric acid calculi by means of microscopic sections proves that the starting point of the concretion may be an irregular aggregate of what in a discrete form constitutes a urinary sediment, or, as it might be termed, "crystalline rubble"; or, that the calculus originates in the manner of a crystalline sphere, or stellar aggregate. This, it may be observed in passing, is equally true of calculi of calcium oxalate, for the nucleus of these is sometimes a disorderly aggregate of massive octohedra; and at other times the calculus is, from the outset, a radially and concentrically striated crystalline sphere, and continues to grow as such throughout its course.[1] In what I have designated the cancellous form of uric acid calculi the nucleus consists of an aggregation of cuboidal crystals, entirely wanting the elongation of those which construct the compact variety. Such cuboidal crystals may be encountered singly or even in groups in uric acid sediments (Rieder and Delépine's Atlas, Pl. vi, fig. 4; Pl. xxii, fig. 6).

The term "nucleus" is commonly used to imply that the central portion of a calculus has a different chemical composition from the rest, so that a calculus of uniform composition is not spoken of as having a nucleus at all. The study of microscopic sections shows that structurally such a definition is inadequate. In the cancellous variety the nucleus, as already mentioned, is an aggregate of loosely arranged crystals, which is followed by a "body" of wavy interconnected zones, or arcades, of somewhat similar crystals, devoid of radial elongation; and in the compact variety, the dense mass of radiating columns in some cases takes its point of departure upon a minute collection of cuboidal crystals. In urinary sediments of uric acid one may recognize in miniature these different methods of construction.

It is one of the established facts in regard to crystallization that the genesis of crystals, as distinguished from their growth, takes place only in a supersaturated or "labile" solution. In the production of such no foreign material is necessary. In the case of less highly saturated or "metastable" solutions, crystallization is started only by the presence of crystals introduced or previously formed during a labile condition.

---

[1] *Trans. Path. Soc. Lond.*, xlvi, p. 91, W. M. Ord and S. G. Shattock, "On the Microscopic Structure of Urinary Calculi of Oxalate of Lime."

It will be evident from this that in the formation of uric acid sediments as well as of calculi, the secretion of a urine supersaturated in uric acid is necessary before crystallization can occur; the presence of shed epithelium, &c., is not *necessary* to start it in the supersaturated fluid, and it would be quite unable to do so in a urine that was simply saturated.

The Elongation of the Crystals.

Although the radial distribution of the elongated crystals might be attributed to a central attraction, experiments have hitherto failed to show that the direction of crystallization can be influenced by magnetic causes. In certain investigations by Mr. T. C. Porter,[1] it seemed at first as though an orientation of crystals growing in a magnetic field (as watched under the microscope) was attributable to the last-named cause, but the orientation admitted of being explained by the direction in which the surface of the glass side had been rubbed before cleaning it for experiment. If there be any such effects they require, remarks the author, in order to show them indisputably, more powerful fields than those of the very powerful electro-magnets used by him. The radial disposition is one thing; the length of the crystals in the compact variety, or in the compact zones of the cancellous variety, is another.

One of the most striking differences between the crystalline forms met with in urinary sediments of uric acid, and in calculi, is the extraordinary length assumed by the crystals in the compact varieties. The hypothesis best fitted to explain primary crystalline elongation is that put forward by Miers, and is that it arises in connexion with supersaturation of the fluid from which crystallization takes place. One of the most striking illustrations of the phenomenon is the well-known form of crystallization which occurs in a supersaturated solution of sodium hyposulphite or thiosulphate. If a crystal of the salt be dropped into a beaker of the supersaturated fluid fine crystals start out radially from the body introduced, extending through the whole volume of the fluid, which in a short space of time becomes completely solid with the production of a sensible degree of heat: the insertion of the crystal is not essential, for movement alone of the flask or beaker will bring about a general, though less regular, crystallization.

The explanation of crystalline elongation put forward by Miers[2] is that, given an initial aggregate of crystals spontaneously formed in a supersaturated or "labile" solution, the fluid immediately around the sides of each will be reduced in its degree of saturation *ipso facto* by the abstraction from the fluid of the material which has resulted in the crystallization; the process of accretion will, therefore, be retarded or temporarily suspended in this situation, whereas, on the contrary, the end of the crystal is free to advance unchecked, or checked in a less degree, into the still supersaturated fluid beyond. It might be thought that it would follow as a corollary that an isolated crystal, of which the normal figure was of the shorter type, would not elongate in the same way, since the desaturation or reduction of the "labile" state of the circumjacent fluid to the "metastable," would not be greater at one spot than at another; and that the elongation would require the presence of other crystals in its immediate vicinity, the continued growth of which would reduce the saturation between the adjacent portions of the elements of the group. It has been shown, however, that a series of currents are set up on the sides of the growing crystal which result in a flow of the fluid towards and beyond its point; this may explain the undoubted fact that a crystal may elongate to a marked degree when far removed from others in its neighbourhood. The elongation of one or both ends of a rhomb (already referred to under uric acid sediments) is explicable on the same principle—viz., the advent of a supersaturated urine upon crystals which have already grown under other circumstances.

---

[1] *Proc. Roy. Soc. Lond.*, 1904, lxxiii, p. 5.

[2] H. A. Miers, F.R.S., "Some Recent Research upon the Birth and Affinities of Crystals," *Science Progress*, No. 5, July, 1907; "Crystallization of Potassium Bichromate," *Mineralogical Magazine*, April, 1908, xv, No. 68, pp. 39-41; H. A. Miers and I. Chevalier, *ibid.*, xiv, No. 65, pp. 123-133.

Differences in the degree of saturation of the urine may, on this hypothesis, determine the initial difference in structure between the cancellous and the compact varieties of uric acid calculi, the latter of which is particularly characterized by the length of the crystals. The occurrence of compact zones in the cancellous variety, composed of long slender radially set crystals, may be attributed to the temporary excretion of a more highly saturated urine; and the occurrence of cancellous zones in the compact variety, to the opposite condition. In the case of compact calculi without a differentiated nucleus, the crystallization would start in a supersaturated urine as a microscopic radial aggregate. Once started, however, the subsequent history may be one of slow or of more rapid growth, according to the condition of the urine; and in the same calculus there may be evidence that the rate has varied at different periods. The huge transversely striated, closely compacted cones that construct the centre of the compact variety of calculus have grown slowly in urine, not saturated to the labile degree, any interstices originally present in the initial stage (which may have been rapid in its course) having slowly become filled, and the solid sphere so resulting having continued to increase by the periodic addition of layers to the bases of the individual columns or cones forming its surface.

The zones constructed of more slender, long, radially set crystals with intermingled fine, rod-shaped crystalline material are of more rapid production. As already noticed, this fine material consists, like the rest, of uric acid. At first a certain difficulty presents itself in the fact that the same chemical substance should assume two so different forms. But such fine crystallization can be brought about in both labile and metastable solutions by shaking. Miers has noticed that the passage of a solution to a supersaturated or labile condition is recognizable by a sudden change in the refractive index, and by the showers of crystals produced if the liquid is agitated. If there are crystals already present, clouds may also appear in the metastable solution under similar conditions.

In the case of calculus formation it would appear, then, that the movement of a labile or even of a metastable urine will account for the constancy and abundance of the finely divided crystalline material which is showered upon the surface and between the crystals of the longer kinds produced in the supersaturated solution, as well as about the shorter cuboidal forms growing in a metastable fluid.

That no material centre is needed as a nucleus in the genesis of rosettes or of radial aggregations is shown by Miers's observation, that in a supersaturated solution in a tube, crystallization may start at several independent centres, possibly since the degree of saturation in such dense fluids is not absolutely uniform throughout, the process commencing at foci which are the most highly supersaturated; his experience is that the presence of impurities, or of insoluble or other material, makes no difference to the spontaneous crystallization. Hence, although in the case of the urine, the presence of shed epithelium or of mucin would invariably be available to start crystallization in a supersaturated urine, this is not theoretically necessary; it is an accident, and nothing more, in the process. As before observed, showers of minute crystals may be originated in simply saturated or "metastable" solutions by agitation, provided, in this case, that crystals are already present. A number of minute crystals, as Miers observes, grow on the surface of the other crystal or crystals, and many of these may get loose and become new centres of growth under the above conditions. In the cancellous variety of calculus finely crystalline uric acid is always found entangled between the groups of cuboidal crystals that construct the nucleus and open laminæ. And their presence is explained by these observations. In the production and growth of calculi some importance may be attributed to the movement of the urine. Besides the currents set up by the passage of the urine from the ureters, it must be remembered that the unceasing rythmical contraction of the bladder will materially add to the movement of the contained fluid. In the case of renal or ureteral calculi there would be the movement arising from the constant flow of the secretion.

As to the origin of the cuboidal crystals that start the cancellous variety of calculus, seeing that crystallization can commence only in labile solutions, and seeing that the form of these crystals is not of the elongated kind characterizing growth in a labile fluid, it must be supposed that the spontaneous crystallization has occurred in the finer form, and that thus, once started, the crystals have continued to grow without elongation, in consequence of the secretion having later on been of a less highly saturated kind, their primitive form and mode of origin having in consequence become obscured.

Miers's views offer an explanation, also, of the fine regular cross striation of the larger conoidal masses which construct the central parts, or the whole, of the compact calculus. These cross-markings, as already noticed, are in no way due to the periodic inclusion of the finer crystalline material. Their remarkable regularity and equable distance prove, moreover, that they are not to be accounted for by a temporary cessation in growth produced by changes in the general composition of the urine, or by its periodical evacuation. On the growing surface of a compact collection of radially set ones there will be periodic growth, for the reason that after a given degree of accretion in a metastable solution, the fluid in immediate contact with the surface will be reduced in its degree of saturation, and this will result in a temporary cessation of growth; when, by diffusion, the more distant fluid again reaches the surface, deposition will be renewed. The automatic recurrence of this variation in the saturation of the circumjacent fluid would thus bring about the regular striation present. The actual clefts present in some of the sections are artificial in origin, and are due to the heating of the ground surface of the calculus during the process of cementing it to the slide, and to its grinding. Such results are well recognized in the preparation of petrological sections. The concentric striation is particularly delicate and regular in calculi of calcium oxalate, and is unattended in this case with fissuration. With this, another factor mentioned by Miers (and to which Sir James Dewar was inclined, in conversation with me, to attach the higher importance) must be associated—viz., the thermal increase arising as a result of the crystallization. The crystallization on the surface would be attended with the evolution of heat; this would raise the temperature of the fluid in the immediate vicinity and increase the solubility of the substance in solution; when, by convection, this fluid is replaced by that beyond, deposition will again commence, and so on interminably.

The Distribution of Radial Form.

By reason of its simplicity and wide occurrence, the radial form may be regarded as one of the fundamental or elementary constructive types in Nature. To this type many of the illustrations adduced by Sir Thomas Browne in his "Garden of Cyrus, or the Quincuncial Lozenge,"[1] may, with a greater degree of truth, if less of fancy, be referred. The quincunx is the device represented by a central and four equidistant points set at the angles of an imaginary square. And amongst the multifarious illustrations which Browne, in his pursuit of the quincunx, contrives to bring together, are all the cruciform dispositions obtaining in plants and animals. It will be obvious, however, that the cross as distinguished from the five imaginary points which may be taken to underlie it, is really a simple form of radial construction, the radii of which happen to be four instead of three, or five, or any other number, occurring amongst natural objects.

The crystalline rosettes and spheres of urinary sediments and calculi are of very diverse magnitude. They range from microscopic objects to the largest forms of compact calculi. The spherules produced in crystallizable solutions, by the presence of colloids,[2] are not strictly abrogations of crystalline form, but modifications. For a close examination shows that the spheres, however regular their exterior may be, are constructed of minute radially set crystals. The spheres and dumb-bells of urinary sediments, whether of sodium or ammonium urate, or of calcium oxalate, are equally crystalline in their minute constitution. Between these spherules or microscopic, and macroscopic calculi there are all gradations. The smooth spherical variety of oxalate calculus is constructed in precisely the same way as the microscopic spherule, and so likewise is the compact variety of uric acid. The gigantic enteroliths which not rarely form in the stomach and intestinal canal of the horse exhibit a similar construction. There is a unique collection of such calculi in the Museum of the Royal College of Surgeons, the largest being no less than 2 ft. 3 in. in circumference. Chemically they consist of ammonio-magnesium phosphate, and in the centre of not a few there is displayed a nail, or a piece of flint, or a pebble, which has served as a nucleus.

---

[1] Printed 1658.

[2] G. Rainey: "Mode of Formation of Shells of Animals, &c.," 1858; W. M. Ord: "Influence of Colloids upon Crystalline Form," 1879.

Of the different theories which have been advanced regarding the construction of the Earth, the one which has been longest held is that its centre consists of molten metallic and mineral substance. This theory is based chiefly on the fact that the temperature rises with the distance from the surface, and upon the evidence furnished by the exit of molten material in volcanic eruption. Against this view it has been pointed out by Lord Kelvin that the Earth's crust, were the globe for any great extent centrally fluid, is not of a sufficiently coherent nature to enable it to withstand the stress to which it is subjected by the attraction of the sun and moon, the production of the tides being a ready proof of its general rigidity. Hence the belief now current that the centre is solid. Whatever its chemical composition, I venture here to suggest that the centre is not merely solid, but that it is radially crystalline.

Fancy might picture the peaks, say, of the Himalayas as the free ends of primitive gigantic radial columns of the original sphere, but the fact that these, like the Alps, consist extensively of organically formed rock—of limestone, composed largely of the exoskeletons of marine life—proves that they have once formed the beds of oceans from which the sea has been displaced by their upheaval. The Earth's crust, with which alone geology is concerned, has an ever-changing history; the central core is stable. Some form of nebular hypothesis is that still in favour as best explaining the origin of the solar system. Whether the revolving nebula, conterminous with what is now the farthest planet of the system, were originally gaseous, or were a cloud of discrete solid masses, would not effect the final result. For if gaseous, the production of fluid would intervene before that of solid material, and if a solid sphere arose as a result of the attraction and fusion of lesser discrete masses, a fluid state would have at some time to be postulated from the heat generated by the impact. In discussing the cause of the Earth's internal heat, George Romanes[1] argues that there would be few sufficiently violent collisions to liquefy such masses until the conglomerate into which they might collect became so large as to cause very high velocities of approach by reason of their gravitating influence: not until a central mass approximated the size of the moon would it have power to produce such velocities of impact as would liquefy the body striking and the part struck, and such heating would always be superficial. He holds, after Helmholtz, that the internal heat is due to contraction. The view that the primal nebula was gaseous, is supported by the fact that existing nebulæ give spectra such as are furnished only by material in a gaseous state.

---

[1] *Proc. Roy. Soc. Edin.*, xxiv, 1903.

The genesis of the moon is best explained, moreover, by its having been centrifugally detached from the Earth whilst the latter was still in the fluid state. Now, given a sphere of molten material revolving free in space and unacted upon by environment, on solidifying it would, I venture to submit, crystallize in the radial direction, though not from its centre, but from without inwards. The solidification would commence at the surface as a result of the radiation of heat, and it would proceed thence inwards. *Pari passu* with the contraction of the solidifying exterior an increasing pressure would be exerted upon the fluid within; the latter would acquire, as a result, the solid condition even though its temperature exceed that of the surface, which still holds true. The two things would proceed concurrently; the increasing pressure from without, and the progressive solidification within, until the whole of the remaining fluid in the centre were eventually rendered solid. The process of solidification would probably take a main course of the kind to be observed in a spherical flask of supersaturated solution of sodium hyposulphite when crystallization is started at its surface.

If such a flask is cooled on the exterior by pumping ether over it, crystallization starts at a certain number of points on the inner surface of the glass, and rapidly extends radially from each as a centre. The result is the production of a series of huge compact cones, the lateral extension of each of which is limited by its meeting with those which are simultaneously growing from neighbouring points.

The lines of apposition are quite obvious. The method, I have indicated in the accompanying diagram, although the natural result is, of course, wanting in the mathematical regularity shown (fig. 2).

That no such radial construction is at present evident in the crust is, of course, only what may be expected, by reason of the long series of upheavals of which it has been the seat. It is commonly held to be the general contraction which has brought about the irregular elevations of the exterior, and that the splitting of the crust accompanying volcanic eruption has the same source. It is assumed that collections of molten material are still actually present in the deeper parts, but it seems to me that such a supposition is not strictly necessary, and that, on the local diminution of the superjacent pressure which would result from fission, the deeper material might again become fluid, and appear as the molten substance ejected in volcanic eruption.

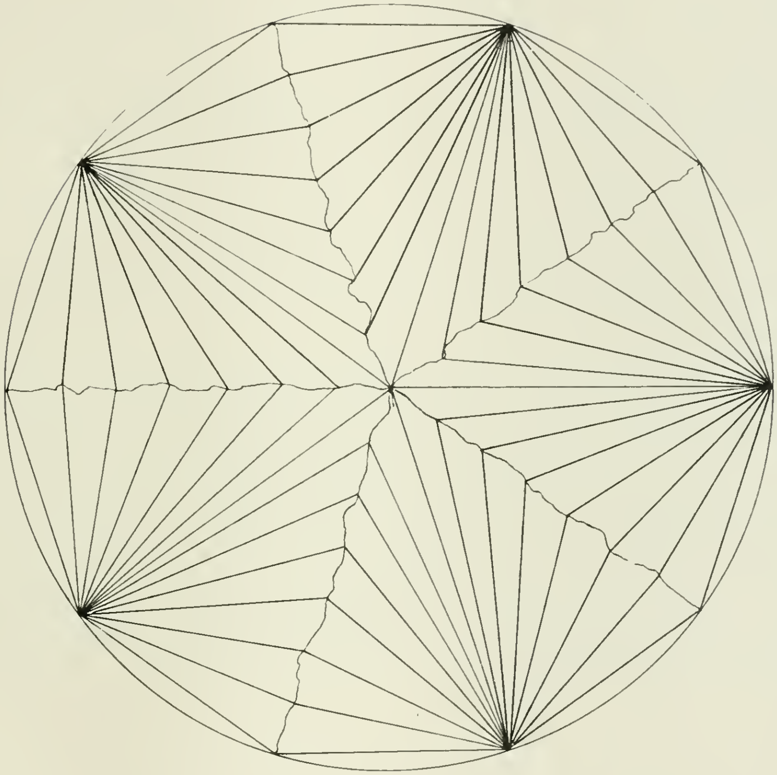


FIG. 2.

Showing the method of crystallization of a supersaturated solution of sodium hyposulphite as observed in a spherical flask, when started by cooling the surface with ether. The course of the crystallization occurring under these conditions possibly reproduces that by which the fluid mass originally composing the Earth, became solid, though the production of secondary centres of crystallization in the deeper fluid during the solidification would disturb the regularity of the final result. The crystals formed in the experiment are acicular; their direction only is indicated in the diagram.

I have systematically inspected the large collection of meteorites in the British Museum, and those in the Museum of Geology, with the object of seeing whether they throw any light upon such a view of the radial construction of the Earth's core. But they do not, and cannot, for the reason that, contrary to other belief, meteorites, whatever their composition,[1] reach the earth in the same condition in which they enter its atmosphere. The circulating interplanetary solid is not rendered gaseous and then reconstructed during its passage through the Earth's atmosphere; the surface of the meteorite becomes incandescent, and nothing more. Not a single one is spherical, or shows any segment of a sphere, as some assuredly would upon fracture, were they once spheroidal. The fact that such bodies, moreover, in a few instances have been found almost cold when examined very soon after their fall, proves that they cannot have lost and regained their original solid condition; the incandescent surface, in fact, becomes cooled during the transit of the body through the air. The action of acid upon the polished surface of siderites brings out lines of crystallization, known, after their discoverer, as Widmanstatten figures (1808), but these observe no radial disposition.

Mr. Prior was good enough to examine, microscopically, with me some of a small sample of dust obtained from a Greenland glacier, and preserved in the mineralogical collection at the British Museum, for it seemed to me that, if meteoric, it might possibly consist of spherules reconstructed from solid which had passed through a gaseous or a molten state. We found, however, that the dust consisted solely of irregular particles, which Mr. Prior pronounced to be aggregated crystals of quartz. The dust in question is almost certainly not of meteoric but of terrestrial—i.e., volcanic—origin, and has been carried by winds to the region whence it was obtained.

---

[1] (1) Meteoric iron, "Siderites"; (2) Nickeliferous iron in sponge-like masses, of which the interstices are filled with silicates, "Siderolites"; (3) Silicates, &c., with smaller amounts of nickeliferous iron, "Aerolites."

Pathological Section.

February 7, 1911.[1]

Seven Cases of Amaurotic Idiocy (Tay-Sachs Disease).

By Hildred B. Carlyll and F. W. Mott, F.R.S.

PART I.—By Dr. Carlyll.

Amaurotic family idiocy is a name which has been given to a rare disease of the nervous system occurring in Hebrew children. For these reasons a practical knowledge of the disease, as regards clinical symptoms and diagnosis, can be possessed only by a few; and, further, the objection raised by the Jewish race to autopsies increases the difficulty of obtaining material for systematic pathological research.

The clinical symptoms of the disease are so characteristic that, if borne in mind, no difficulty in diagnosis should occur; indeed, the pathology is so distinct as to indicate a correct diagnosis.

In recent years, as knowledge of the disease has increased, many accounts of cases, partial or complete, have been published; so that a correct diagnosis is now arrived at more frequently. As yet, however, the majority of text-books contain no adequate account of this interesting disease. It is therefore with no apology that I put on record the notes of six cases which have come under my personal observation during twelve months' residence at the East London Children's Hospital, together with those of a case recently under Dr. Mott's care. That the details of some of these cases are incomplete will be evident to the reader, but I trust that the shortcoming may be condoned when it is remembered that in these alien families the details of the medical history had frequently to be obtained through an interpreter.

---

[1] Laboratory meeting held at the National Hospital, Queen Square.

My six patients are not akin to one another, but an elder brother of the first child whom I saw suffering from the disease had died three years before of the same complaint. His case, with another under Dr. Mott's care, was published in detail by Dr. Mott in the third volume of the *Archives of Neurology*, and I shall draw from the notes of that case for purposes of comparison with those of his sister (Case I). Case VII is a patient that recently came under the notice of Dr. Mott at Charing Cross Hospital, and, with his permission, I have included the history with my series of cases.

One of my patients (Case IV) came under observation in an interesting way. The mother of the two children mentioned above, a woman of unusual intelligence, noticed this child in his mother's arms in a tram-car. A rapid inspection convinced her that the child could not see, and she quickly came to the conclusion that his state was identical with that of her own children. The child's mother was so impressed with the statement that her baby could not see, which fact she had not discovered for herself, that she readily consented to bring him to the hospital.

The seven patients are all children of Jewish patients, coming from Russia or Poland. Some of the children were born abroad, others in England. Five cases are of girls, two of boys. Another member of the family of my first patient (Case I) died of the disease; and in Case III there are reasons to suppose that the only other child of that family may have died of the same affection. The youngest child in the family of which the patient (Case II) was a member presents certain very suspicious symptoms, and will be kept under observation.

All the children have now succumbed and their ages at death were as follows:—

Case I, 2 years 3 months.
,, Ia, 12 months.
,, II, 2 years 4 months.
,, III, 1 year 8 months.
Case IV, 2 years 3 months.
,, V,[1] 13 months.
,, VI,[1] 18 months.
,, VII, 17 months.

Dr. Mott's other case, reported in the third volume of the *Archives of Neurology*,[2] died at the age of 2 years.

In the light of these figures, and of those of other published cases, it is justifiable to tell the parents that children with this affection will not reach the age of 3 years. In none of the families from which these cases are taken is it recorded that the firstborn children were afflicted with the disease (except in Case III).

---

[1] These cases have died since this paper was written.

[2] *Arch. of Neurol.*, 1907, iii, p. 218.

In Case I and in Case 1A the fourth and fifth children of the family were affected.

In Case II the patient was the fourth child of the family.

In Case III the second child was affected (the first child is now known to have died from the disease).

In Case IV the patient was the second child of the family.

In Case V the fourth child was affected.

In Case VI the third child was affected.

In Case VII the seventh child was affected.

In every case (except Case III) the patient had healthy brothers or sisters.

In the case of Jenny M. (Case I) the mother sought advice as to whether she would be wise in having any more children or not. She was told there was no reason to believe that her next child would be afflicted.

CASE I.—JENNY M.

Under the care of the late Mr. Hancock at the East London Children's Hospital, 1909.

Family history: Parents alive and well. They are Jews, the father coming from Russia and the mother from Poland. They reside in Spitalfields. No consanguinity. When married, father was 18 and mother 17 years old. The patient is the fifth child of the marriage. The eldest child died at 8 months, of diarrhœa. The second child is now 8 years old and is well. His eyes have been examined and nothing abnormal was detected. The third child, a girl, died of pneumonia when a baby. The fourth child, a boy, afflicted with amaurotic idiocy, died at Shadwell at the age of 12 months (Case IA). The fifth child is the patient. No miscarriages. The parents have decided not to have any more children.

Mother's family: The mother's parents are alive and well. No consanguinity. She is one of seven children, of whom three are dead, the cause being unknown. One of her brothers is married, but has no children; her other brother has had four children, who are all healthy.

Father's family: There is no information about the father's grandparents; his parents are alive and well. No consanguinity. He has two brothers and seven sisters; of the latter, one died at the age of 3 years from croup, and one after living a few weeks. The remaining five are between 12 and 22 years of age; they are healthy; none are married. Of the two brothers, one is married and has two children who are well, the other is 6 years old.

History: Full-time child, no instruments; breast-fed up to eleven months. The mother, a very intelligent woman, brought her to see Mr. Hancock when she was 4 weeks old because she thought the child was "going like Jack." When the child was 3 months old the mother brought her again, as she noticed that she was partially blind. (Mr. Hancock found the typical ophthalmoscopic signs.) She noticed also that the limbs were stiff because she had difficulty in powdering the groins. The child cried less than other children and did not take any notice of things. From birth she never recognized the difference between the mother who nursed her and other people who lifted her up; nor did she ever play with toys. The general health for twelve months was fairly good, but gradually she got thinner and took her food badly. She always lay quietly where she was put, and the power in her limbs rapidly decreased.

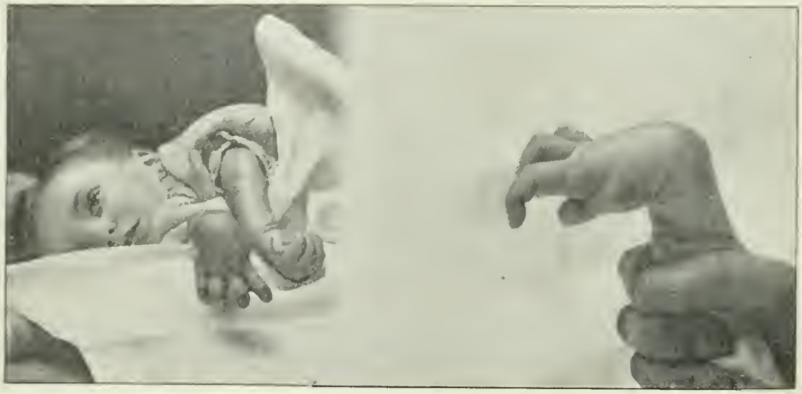


Fig. 1. Case I.—Jenny M. Fig. 2.

July 9, 1909. July 9, 1909. Showing the early "main en griffe."

On admission, January 9, 1909, aged 1 year and 4 months (the following meagre notes are all that are obtainable): Well nourished and plump. Takes very little notice of people or surroundings, and appears only to eat and sleep. Heart and lungs normal. No convulsions. No strabismus nor nystagmus. Limbs spastic. Weight, 19 lb. 11 oz.

Progress: There are, unfortunately, no notes about her progress; but I am told that the child became progressively thinner, and that nasal feeding became necessary about May. In July, her weight was 14 lb., and the photographs taken then show the hyper-pronation of the left forearm. On the right side, the hand has assumed a position of "main en griffe" (figs. 1 and 2).

November 9: Always lies in a semi-comatose condition; eyes continually open; pupils dilated, reacting slightly to a strong light. The eyelids sometimes blink, and the eyes are at times withdrawn from a bright light. Optic atrophy marked. Usually there is a slight internal strabismus of recent date. The mouth is kept shut, but can be moved well. There is a slight sucking reflex. The child seems insensitive to sound; head retracted; back considerably bent with scoliosis and kyphosis. Right side: Arm kept slightly from the body and extended at elbow; forearm fully pronated; wrist acutely flexed, and has sore places on dorsum. Fingers hyper-extended at metacarpo-phalangeal joints, and flexed elsewhere; thumbs fully adducted and slightly opposed; slightly flexed at metacarpo-phalangeal joint and extended at the inter-phalangeal joint. Leg flexed at hip and knee; sores present over knee; foot and toes plantar flexed to the fullest extent. Left side: Arm slightly flexed at elbow; forearm hyper-pronated; leg straight at hip and knee; position of foot as on right side. These contractures are well shown in figs. 3 and 4. Abdomen very lax; no enlargement of liver or spleen. The plantar reflex is extensor on each side, as exemplified by a slight straightening of the great toe. Deep

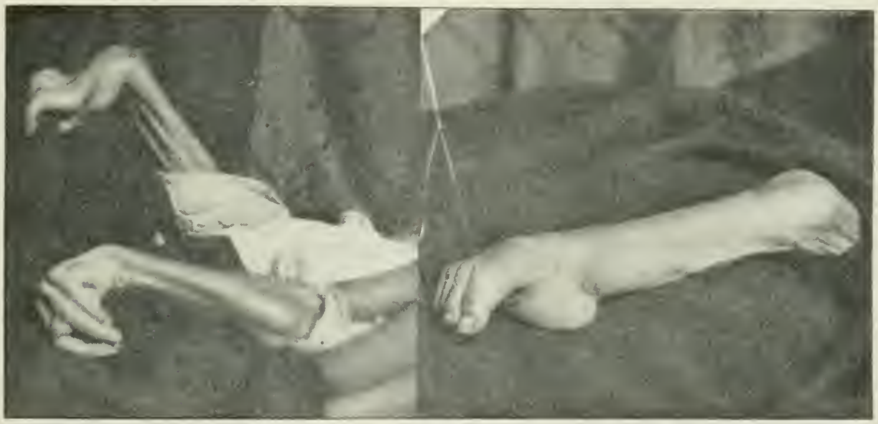


Fig. 3. Case I.—Jenny M. Fig. 4.

November 12, 1909 (a few hours before death). Note the difference in the deformity of the two limbs.

November 12, 1909.

reflexes not obtained. There is great muscular wasting. The position of the joints can be altered by force, but they tend to return to the original position, except in the case of the right knee, the muscles of which are very weak and lax, so that the joint can be placed in any normal position. There are numerous pressure sores all over the body. Urine and fæces are passed in the bed. Nasal feeding has been employed for five or six months. There have been several slight attacks of bronchitis. The temperature has been normal throughout, except for some terminal pyrexia.

November 12, 1909: The child died, aged about 2¼ years.

Immediately after death had occurred the eyes were removed and placed in fixing solutions. The brain was removed later and hardened in Müller's fluid, and subsequently examined by Dr. Mott. No further examination was permitted.

CASE IA.—JACK M.

This child was under the care of Dr. Eustace Smith, Dr. Coutts, and Mr. Hancock at the East London Children's Hospital, in 1906. Dr. Mott performed the autopsy, and has published the result of his chemical and histological examination in the *Archives of Neurology*, iii, 1907. With his permission, and as the second case in the family (Case I) has recently proved fatal, I give his notes here for comparison :—

Family history: *See* Case I.

History: Quite healthy at birth. Normal confinement. No urine passed for twenty-four hours. When six months old the child developed a rash which lasted four or five days. Soon afterwards he developed pneumonia, being dangerously ill for a week. Since this illness he has not noticed things, and his mother thinks this may be due to deafness. His attention cannot be attracted. The eyes wander aimlessly. The mother thinks he should hold his head up better for his age. He has had an aural discharge for five months. No squint. Breast-fed always; no other food; bowels regular. Has never walked nor crawled, but at 6 months old could stand firmly on his legs when allowed to do so. No definite paralysis. The back muscles appear to be quite strong. At six months old the child was taken to the Moorfields Eye Hospital. He was admitted, but having contracted measles, was discharged shortly afterwards.

On admission, January 13, 1906, aged 8 months: Weight, 20 lb. 10 oz. Very well nourished, rather pale. Head of good shape; fontanelle widely open; no cranio-tabes; hair normal. Eyes wander aimlessly. Attention appears to be attracted, however, by the ticking of a watch, and he looks for it in the right direction. When placed in a sitting position his head does not fall about. Muscular development appears to be very good. Skin normal. Sleeps and eats well. A very amiable child. Gastro-intestinal system: Two lower incisors only; tends to protrude tongue like a Mongol. Abdomen large; spleen and liver not felt. Bowels usually regular; stools normal. Thorax: Well covered, good shape; nothing abnormal in heart or lungs. Nervous system: Cranial nerves intact; pupils equal in diameter; normal reactions direct and consensual; eye movements good in all directions; no nystagmus nor strabismus; no paralysis nor muscular wasting. Reflexes normal for child's age. Fundi present characteristics of the disease.

Discharged January 25, condition "in statu quo."

Re-admitted February 7, 1906, suffering from pneumonia, following what was said to have been measles, but which was probably scarlatina. Very weak. Still well nourished. Neck muscles now obviously weak, causing head to fall back; no rigidity; no obvious muscular wasting; reflexes are still brisk; plantar reflex extensor; eyes are unchanged; well-marked signs of broncho-pneumonia.

February 18: Has been in a critical condition, but signs in lungs are now clearing up. Reflexes brisk. Head muscles weaker, but can move head a little from side to side.

February 24: Still some bronchitis. Right arm now appears to be rigid. Desquamating freely.

February 26 to March 10: Temperature is very irregular, reaching 103° F. at times. Losing weight (14 lb. 14 oz., March 8). Well-marked œdema of hands and feet; semi-conscious. Rigidity of limbs variable. Resents ophthalmoscopic examination.

March 31: Weakness increasing. Temperature subnormal. Profuse hæmaturia. No casts in urine.

April 10: Marked anæmia; skin very dry. Much œdema; abdomen very sunken; skin loose and inelastic; has fits of crying and irritability. Does not know anyone; no hæmaturia; rarely moves limbs, and then only the upper; no vomiting. Eyes nearly always open; they wander aimlessly. Limbs alternately rigid and flaccid. Lower limbs powerless; knee-jerks brisk; Babinski's sign variable.

April 12: Child died suddenly, aged 12 months.

CASE II.—MILLY T.

Under the care of Dr. Eustace Smith in the East London Children's Hospital in 1909. Died at St. George's Infirmary, E., 1910. No autopsy.

Family history: The parents are Russian Jews, and have been in England seven years. They are both strong and well. They have had five children, three older and one younger than the patient. The older children are boys, aged 12, 10, and 5 years respectively (1910). The youngest of the family is a girl aged 8 weeks. I have recently seen this child, and she presents a suspicious similarity to early sufferers from amaurotic idiocy. I was not given permission to examine her eyes. There is no consanguinity between the parents. When they married the mother was aged 20 and the father 24. They do not know of any similar cases of illness in their respective families.

The patient was born at full term without the assistance of instruments, and was brought up on the breast and the bottle.

History: The child was quite well until a year old, when she seemed not to see, and was taken to a hospital. She seemed to notice only bright lights, and could not support her head. At 3 months old she had fallen on her head. Is usually constipated, and has been wasting latterly. Has had no infectious fevers.

Admitted July 8, 1909, aged 16 months. Well developed and well nourished. Skin pale. Weight, 16 lb. General muscular rigidity, especially in arms and legs, which are slightly extended. This stiffness is easily overcome. No head retraction or spinal rigidity. Child lies in a restless condition. Eyes rather fixed, and kept open. Sight seems impaired, but she is not blind, for she turns eyes towards objects presented. (There is no note of an ophthalmoscopic examination.) Cranium well shaped; anterior fontanelle almost closed; no cranio-tabes. Nostrils triangular shaped. Mouth kept open; tongue protruded and often sucked. Abdomen protuberant and rather flaccid. Liver and spleen not palpable. Knee-jerks decidedly exaggerated. No ankle-clonus. Plantar reflex extensor. Lungs and heart normal. Child swallows badly.

Discharged July 14, 1909. Whilst under observation, the temperature ranged continuously from 100° to 102° F. Lumbar puncture revealed a sterile fluid containing a few lymphocytes.

Admitted to infirmary, October 25, 1909, aged 19 months. Weight, 18 lb. 10 oz. The pupils were small and reacted. There was apparently no sight. The face twitched symmetrically. Kernig's sign was marked. There was much salivation.

November 13: Weight, 18 lb. By the kindness of Dr. Bowlan I was allowed to watch the child's progress.

December 8: Weight, 17 lb. The child lay where placed, but started at sudden noises. Pale and flabby, features becoming pinched. Much crying. Swallowed fairly well, and did not require nasal feeds. Head fell back when child lifted. Limbs slightly rigid. Knee-jerks brisk. Plantar reflex extensor. Thumbs markedly adducted. Examination of the fundi revealed the characteristic appearances. Lumbar puncture gave a small quantity of fluid under very low pressure. Shortly after this child became worse, limbs being much contracted. Nasal feeds became necessary for a time.

December 26: Weight, 16 lb. 8 oz.

January 3, 1910: Weight, 16 lb.

January 12 (*see* fig. 5): Swallowing well. Pupils react to light. Limbs not markedly contracted, but both hands clenched over adducted thumbs. Plantar reflex indefinite but not extensor. A quantity of fluid was obtained by lumbar puncture.

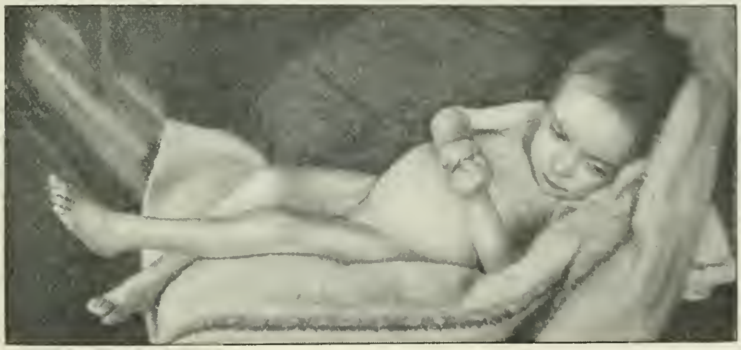


Fig. 5.
January, 1910. Case II.—Milly T.

The child stayed in much the same condition for several months, becoming more wasted and rigid.

The cerebrospinal fluid (first sample) was alkaline and contained some sugar. Noguchi's test showed an absence of globulin. There were neither cells nor organisms present. Second sample: Protein content normal. Choline test negative. No lipoids beyond a trace of cholesterol present.

July 18, 1910: Death occurred from pneumonia, following an attack of measles, at the age of 2 years 4 months. No autopsy was allowed, in spite of the most urgent appeals for permission.

Case III.—Fanny M.

Under the care of Mr. Hancock at the East London Children's Hospital for a short time. Transferred, on account of an epidemic of measles, to the Evelina Hospital in 1909, where she was under the care of Dr. Briscoe. (Owing to Mr. Hancock's death, the child's condition while at Shadwell is not known.)

Family history (1910) : The mother was married once previously in Poland when she was 16 years old. Of this marriage she had one miscarriage. She was aged 24 when she married, in 1904, her present husband. He was 30 years old. No consanguinity. They are Polish Jews, and have lived in England for five years. The mother's parents died when she was a baby. She has one brother, aged 35, who has six healthy children. The father has one sister and several brothers, who have healthy children. The parents have never seen any children with amaurotic idiocy in Poland. They have had two children in all ; the first, a boy (J. M.), was born in Poland, and died in 1907 at the German Hospital, aged 2¼ years. The case-notes are not obtainable. He was weakly from birth, and said to be rickety. Breast-fed for twelve months. Never used his legs. Became very thin before death. I have seen two photographs of this child, taken at 3 and 12 months respectively. The first photograph shows a fat, intelligent-looking baby with widely open eyes. In the second he is also fat, but presents a rather idiotic look. The limbs appear normal. The mother cannot say anything definite as to his powers of vision, but I think it probable that this child was an amaurotic idiot. The mother agrees that this is probable. (A recent examination of the sections which were prepared when this boy died, leave no doubt that he died of this affection.)

History: Born in England at full term ; large child at birth ; no instruments. Breast-fed to 8 months. Well up to 6 months, when she became weaker and could not hold up her head. When about 8 months old it was noticed that she did not observe things like other children. Sudden noises would start " convulsions."

General condition : The child was sent to the Evelina Hospital on October 7, 1909. She was then 18 months old. Mr. Hancock was anxious to know how she progressed, and I was very kindly allowed to examine her there on November 23, 1909, when her condition was as follows : The face presented a look of dementia, and the child lay where placed. There were no voluntary movements, even when lumbar puncture was performed; nor did the child cry. No obvious muscular wasting and no contractures were observed. The arms and legs were extended and slightly rigid, but flexion could be performed. Thumbs markedly adducted. Some foot-drop, but this was not well marked. Extensor plantar reflex well marked on each side. Knee-jerks obtained, but not exaggerated. Eyelids sometimes open; no movements of eyes; complete blindness; well-marked optic atrophy. At the macula was a circular area rather larger than the disk, of the colour of dirty cotton-wool, with an ill-defined edge. In the centre of this area was a well-marked liver-coloured spot. The child on admission had taken food fairly well, but for some weeks past had been fed by a nasal tube. Lumbar puncture was readily performed. There was no vertebral rigidity. Two large test-tubes were rapidly filled with clear fluid. Shortly afterwards the child developed chicken-pox, and on December 20, 1909, she died, aged 1 year and 8 months. I was told that no improvement had followed the lumbar puncture (fig. 6). The specimen of cerebrospinal fluid was free of blood, and was not abnormal in cell-content. Reaction alkaline. Noguchi's test showed that it contained no globulin. With Fehling's solution a rapid reduction took place, as is usual with normal cerebrospinal fluids. There was but a small amount of protein. Choline and cholesterol were absent.

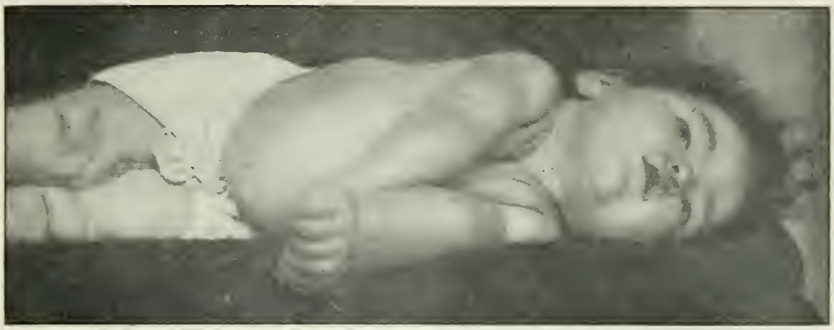


Fig. 6.

Case III.—Fanny M.

Post mortem (December 22, 1909): Well-nourished child. Fontanelle closed. Brain very hard; weight, 33 oz. Ventricles not distended, but a quantity of fluid escaped on opening the skull cavity. Some pus was found in the right pleural cavity.

Through the kindness of Dr. Briscoe, I was enabled to be present at the autopsy, and to obtain portions of the nervous system and other tissues for pathological research. This was carried out under Dr. Mott's direction, and a portion of the histological and chemical investigations are based upon researches made upon this material (*vide* Part II).

CASE IV.—ABRAHAM C.

Under the care of Dr. Eustace Smith in the East London Children's Hospital, Shadwell, 1908 and 1909. Death occurred in the St. George's Infirmary, E., 1909. No autopsy.

Family history, 1910: Two other children, one aged 6, healthy and at school; the other died of "cough" at 2 weeks old. No miscarriages. The parents come from Russia and have been ten years in England. When they married the mother was aged 20 and the father 22. The mother's parents are alive and well. She has three brothers, aged 12, 10, and 6 respectively, and four sisters. Of these two are married and two are still young. Of the former one has two girls and one boy; the other has one girl. The father's parents are alive; he has brothers and sisters, but nothing is known of them.

History: Full-term child; breast-fed; no instruments. At 3 months old suffered from cramps and constipation; occasional fits. Irritable for two months and suffered from screaming attacks. For two months previous to admission mother had doubted whether he could see properly. A diagnosis of rickets was made at several hospitals.

Admitted to the East London Hospital, October 30, 1908, for bronchitis. Aged 1 year. Weight, 22 lb.; very rickety and unable to stand. Well covered with flesh. Irritable and cries a great deal. Occasional nystagmus; unable to fix objects with eyes. Characteristic appearances seen in fundi. Limbs flaccid. Both knee-jerks brisk. Sometimes a slight ankle clonus occurs. Plantar reflex extensor. Fontanelle normal for age. Liver and spleen not palpable. Four central incisors, two upper and two lower appearing.

Discharged November 9, 1908.

Re-admitted January 30, 1909, for bronchitis following a recent attack of measles. Weight, 15 lb. Child apparently quite unconscious. The only movement is a slight slow rolling of the head from side to side. Slight general rigidity. Eyes half-closed, exhibiting slow lateral conjugate movements; pupils small, equal, and circular; no nystagmus; optic atrophy. Abdomen flaccid and retracted. No œdema.

February 3: Lumbar puncture was followed by a slight improvement. The fluid was sterile and showed a few small lymphocytes, but not in pathological numbers.

February 9: Sometimes swallows naturally but generally requires nasal feeds.

February 19: Gaining weight; total weight now 18 lb. Nasal feeds not required.

Discharged March 8. Some improvement.

Re-admitted October 1, 1909, for the third time. Weight, 20 lb. Has taken food well since last admission. Very constipated. Feet and abdomen said to have recently swelled. Fairly well nourished. Lies on side with head retracted. Eyes open and staring. When disturbed eyes move ceaselessly from one side to the other side with rapid, jerky movements. Pupils do not react to light. Legs extended at knee and ankle, but slightly flexed at hip-joints. Plantar reflex flexor; the stimulus usually gives rise to slight clonic spasm of arms and legs, as does any movement of the legs. Knee-jerks increased. No genuine patella or ankle clonus. Arms slightly flexed at elbows; wrists flexed; claw hands. Joints are resistant to passive movement, but on continuing it they become relaxed, and complete flexion and extension can be obtained. The usual position of the joints is, however, quickly resumed (fig. 7).

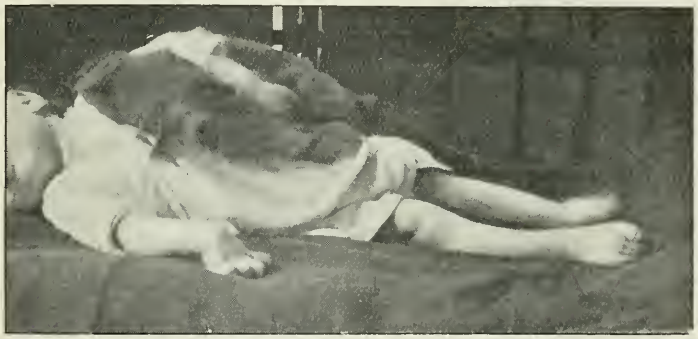


FIG. 7.

October, 1909. Note the claw hand. Case IV.—Abraham C.

Discharged October 17, 1909.

Admitted November 2, 1909, to the St. George's Infirmary. By the courtesy of Dr. Bowlan I was permitted to examine the child on

December 8. Shortly after admission he had convulsions associated with œdema of the legs. He swallowed well and cried but rarely. It was thought that at times he recognized his mother. The head and arms could be moved by the child. The legs were kept extended and rigid. Plantar reflex flexor. Kernig's sign not marked. Well-marked "claw" position of right hand only. Thumbs on each side much adducted and opposed. Eyes showed bilateral movements. Lumbar puncture was performed, but the fluid was not found to be under any pressure. It contained some blood, was alkaline, and contained sugar. Choline and cells absent.

Death, Christmas, 1909. Age about 2¼ years.

CASE V.—HARRY K.

Under the care of Dr. Coutts at the East London Children's Hospital, 1910.

Family history: The parents are Russian Jews and are alive and well. They were not related before marriage. They came to England in 1903. There are three other children, a boy and two girls, aged 8, 5 and 2½ years respectively; all alive and well. No miscarriages. The father is one of seven children; his parents are alive. The mother is one of four children; her parents are dead. I can find no evidence pointing to other cases of the disease in this family.

History: The child was born at full term and without difficulty. He was brought to Hospital because his back was weak, and because his mother thought he was "not like other children."

On admission, July 12, 1910, aged 9 months: Weight, 17 lb. Well nourished; pale; does not look intelligent. Fontanelle not closed. Plenty of hair. Circumference of head 18½ in. Has difficulty in sitting up. Is at times sensitive to sudden noises. Takes food well. Nothing abnormal in chest. Abdomen full and lax; nothing abnormal felt. No teeth. Long eyelashes. Cranial nerves intact. Eyes of oval shape, usually open and staring; no lateral movements; no strabismus. The fundi present a typical picture. The pale area at the macula is about one and a half times the size of the disk. Optic disks show early atrophy (Mr. F. Juler). The arteries are small. The pupils react strongly to light. The child sees objects and follows them with his eyes, but has no sense of their position when trying to grasp them. There is no head retraction, but the neck muscles are very weak. The legs are extended and are spastic, but not markedly so. Knee-jerks very brisk. Kernig's sign indefinite. The feet go into clonus on stimulating the soles, but the legs are only sluggishly moved. The arms move freely at will, the elbows are rigid and flexed. A Pirquet's reaction was positive.

July 20: A test-tubeful of fluid, flowing under considerable pressure, was removed by lumbar puncture. A specimen of blood was removed from the brachial vein.

July 25: There was no pyrexia after the lumbar puncture, but the rigidity was for a time less marked. The operation wound on the arm healed by first intention. The cerebrospinal fluid showed an absence of choline and cells. There was no excess of protein. The amount of carbonates and carbon dioxide in solution was not altered. Cholesterol was absent. There was no excess of lipoids or fats.

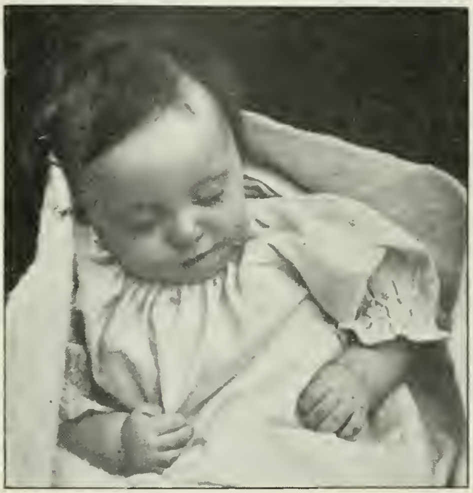


Fig. 8.

July 19, 1910. Case V.—Harry K.

August 26: Weight, 16 lb. Temperature normal. The child lies precisely where it is put, however uncomfortable the position. He is almost ridiculously good for his age, rarely crying, and then only on the greatest provocation. He usually looks about him with widely open, staring eyes, which produce an absurdly vacant expression. He grunts with pleasure when he is spoken to, and frequently bursts into laughter. It is evident that he does not recognize his mother. Perception of light and of bright objects is fairly keen. No squint nor nystagmus; pupils react strongly to light. Mouth almost always open; tongue is protruded sometimes. On being sat up, the weakness of the back muscles is evident; but if the head is pushed backwards, as it is very readily, the child can bring it forward to a flexed position. The arms are flexed at the elbows, and are constantly moving. The forearms are strongly pronated. The thumbs are adducted and opposed, and the fingers are kept flexed. Legs somewhat spastic; knees kept extended; thighs are adducted and forcible abduction causes pain and reveals the muscular rigidity. The plantar reflex is flexor; knee-jerks easily obtained, but not very exaggerated. The legs are not moved away from painful stimuli; they fall helplessly on being lifted. The child takes food well, but will not be bothered with a bottle. The bowels are regular; stools and urine normal. (*See* figs. 8, 9, and 10).

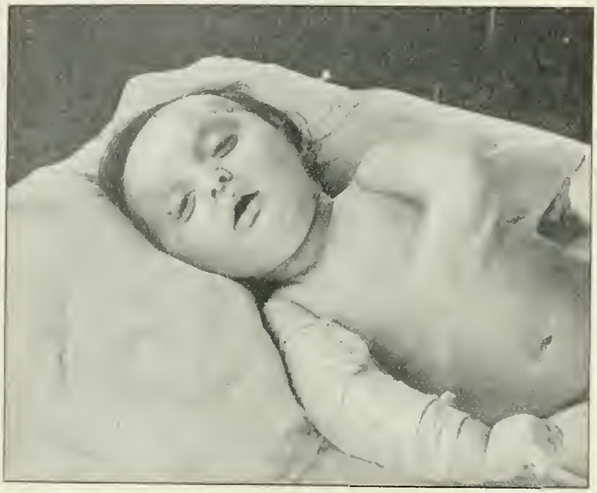


Fig. 9.

July 28, 1910. Case V.—Harry K.

October 1: Weight, 14 lb. 12 oz. Hands pronated and thumbs tucked in. Fingers move readily at will. Knees extended; no equinus; plantar reflex flexor; jerks readily obtained. There is some difficulty in swallowing at times, but nasal feeds are only occasionally needed. The child has no teeth. The general weakness has increased. On placing salt upon the tongue the child does not cry, but makes a slight grimace.

At times there are outbursts of vacant laughter. Pin-pricks do not readily cause crying. The pupils react slightly to light.

The child was removed by the parents from the hospital on October 31, 1910. Weight, 14 lb. The child died suddenly on November 4, 1910, possibly from some accident while being fed. No autopsy was permitted.

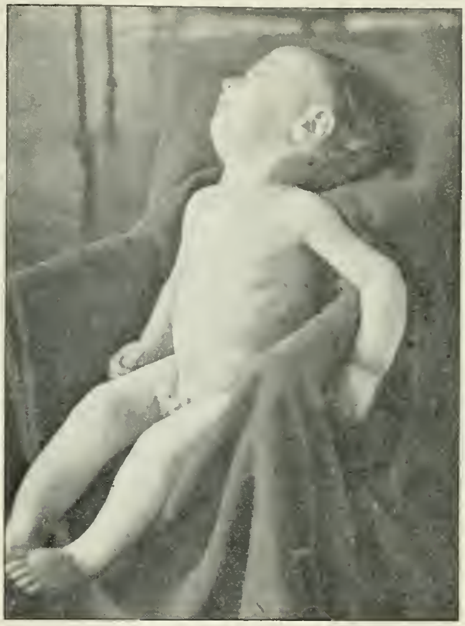


Fig. 10.

August 24, 1910. Note the results of weakness of the neck muscles.
Case V.—Harry K.

Case VI.—Sarah G.

In the Queen's Hospital for Children, 1910, under the care of Dr. Bellingham Smith, to whose courtesy in allowing me to admit the child for a time in the East London Children's Hospital, and to photograph her, I am much indebted. (This child was shown at the Royal Society of Medicine by Dr. Bellingham Smith in May, 1910.)

Family history: Patient is the third child of Polish Hebrews. The parents are alive and well. They were married in 1902, and have lived in England for five years. No miscarriages. According to the history, the two elder children, a boy and girl, aged 7 and 4½ years respectively, are both healthy, and this is corroborated by a photograph of them which I was shown. The father is one of seven children, two of whom died in early childhood. One of his brothers is consumptive. He has a step-brother and four step-sisters. The mother is one of eight, of whom one died at the age of 3 years. Two of her brothers have two children each, but she is unable to say anything as to the state of their health.

History: The child was brought to hospital for blindness and inability to sit up. She could not hold up her head. She has always been breast-fed. The symptoms had only been noticed for a month prior to admission, namely, when the child was 10 months old.

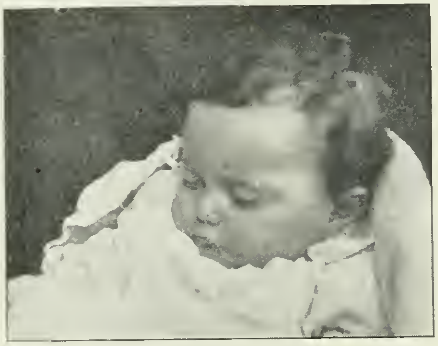


Fig. 11.

July, 1910. Case VI.—Sarah G.

On admission, April 27, 1910, aged 11 months: The child is exceedingly fat and well nourished; very apathetic; unable to sit up without assistance, but on doing so, is unable to support her head unaided. No movements of head or body are attempted, and those of the limbs are limited in character. The limbs are rigid to a variable degree, spasticity being more marked in the legs than in the arms. Plantar reflex flexor. The tongue is protruded. The appearance of the eyes is typical on ophthalmoscopic examination. Optic atrophy is fairly advanced, and the pale area at the macula is rather larger than the disk. There is no nystagmus, but the eyes exhibit aimless random movements. There is distinct appreciation of a strong light, but no notice is taken of surrounding objects. The Wassermann reaction and the Pirquet cutaneous reaction were negative.

July 18, 1910 (*see* figs. 11 and 12): Age, 14 months old; weight, 17 lb.; very plump; has two lower incisors; limbs very spastic; legs and arms being about equal in this respect; unable to sit up; plantar reflex definitely flexor; pupils do not react to light. After a lumbar puncture, at which some cerebrospinal fluid under pressure was removed, there was a pyrexia. The rigidity did not in any way decrease. Perception of light is slight. The pale area at the macula is smaller, and the liver-coloured spot larger than in Case V.

The child died in the autumn of 1910.

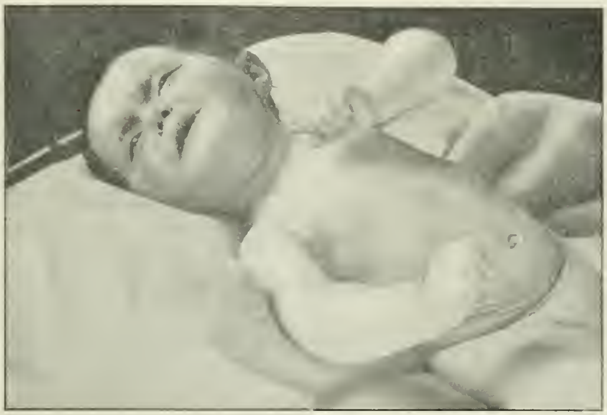


FIG. 12.

Case VI.—Sarah G.

CASE VII.—P. P.

Female, aged 1¼ years. Admitted to Charing Cross Hospital under the care of Dr. Hunter for broncho-pneumonia and rickets, May 25, 1910. It was thought the child did not see properly, and amaurotic idiocy was suspected. Mr. Treacher Collins made an examination of the eyes, with the following result: "Around each macula there is a circular area of white opacity on the retina. The macula itself appears as a dark red spot. The margins of the optic disks are well defined, but they are pale, especially on outer side. The case is one of so-called amblyopic idiocy met with in Jewish children" (E. T. C.)

The case was then transferred to the care of Dr. Mott. The mother, a Polish Jewess, who could speak only broken English, gave the following history: The child has been ailing for about four months with frequent colds and a cough. A week ago the cough became worse. During the last two days the child has been drowsy and partly unconscious at intervals; she has also had twitchings in the arms, and at times gives sudden jumps. There has been no diarrhœa or vomiting, and food has been taken well.

Family history: The following history and pedigree were kindly obtained by Dr. Rees Thomas. Patient is the youngest child of a family of seven. There is no family history of asthma, consumption, epilepsy, insanity, or rheumatism. No case of blindness occurred in any of the children that died young, but the first two children (girls) both died at the age of 1 year from pneumonia, and it is possible that they were afflicted with amaurotic idiocy which, as in this case, was not detected. The third child, a boy, died at the age of 2 years from measles. The other three children, one boy aged 6, and two girls aged 4 and 2½ years respectively, are alive and healthy.

On admission: The child lay in a semi-comatose state, slightly cyanosed, and took no interest while it was being examined; it neither cried nor struggled. Conjunctival reflex was very dull. Its hands and feet were cold. Respirations 70, pulse 170, temperature 103·4 F. Breath-sounds very harsh, and in patches bronchial in type. Moist râles, bronchi and crepitations could be heard. Abdomen lax. Head not retracted.

The physical condition of the patient improved under treatment. Its appetite never seemed to be satisfied; it was always crying, but upon giving it milk the crying would cease. It certainly heard loud noises, but exhibited no signs of disgust when medicine was given. Death occurred on July 17, 1910, and the autopsy was made by Dr. Mott.

Post-mortem notes: Physiognomy indicates no degeneration, like that of an idiot or imbecile. Palate shows no abnormality, broad and flat. Two central incisors, upper and lower, cut. Lower incisors notched, but certainly not indicative of congenital syphilis. No bruises or rashes. A little lividity of dependent parts. On opening chest and abdomen there were no adhesions or fluid. No fluid in pericardium. Slight beading of ribs. No indication of rickets in legs. Fontanelles still open. Tongue, fauces, tonsils, and larynx normal. Muscles good colour. Lungs: Some congestion of both bases. Heart apparently normal. Liver pale and mottled, suggestive of fatty change. Spleen normal. Adrenals small and pale. Kidneys rather paler than normal. Mesenteric glands: A few slightly enlarged; large gland near cæcum. Brain weighs 970 grm.: right hemisphere, 435 grm.; left hemisphere, 435 grm.;

pons, &c., 100 grm. No excess of fluid. No flattening of convolutions; decidedly firmer to touch than normal, has a leathery feel. Meninges not thickened; convolutional pattern complex. Sylvian fissure horizontal. Portions of the organs and the brain were removed for microscopical and chemical examination (*vide* pp. 188).

HISTORICAL SUMMARY.

It was in 1881 that Waren Tay, of the London Hospital, discovered this disease, and his description of the ophthalmoscopic appearances, which has now become historic, may be read in vols. i and iv of the *Transactions of the Ophthalmological Society*.[1]

Under the heading, "Symmetrical changes in the region of the yellow spot in each eye of an infant," he says that in a child aged 12 months he found "the optic disks apparently quite healthy; but in the region of the yellow spot in each eye there was a conspicuous, tolerably defined, large white patch, more or less circular in outline, and showing in its centre a brownish-red, fairly circular spot, contrasting strongly with the white patch surrounding it. This central spot did not look at all like a hæmorrhage, nor as if due to pigmentation, but appeared to be a gap in the white patch through which one saw healthy structure. In fact, the appearances may most suitably be compared with those we are familiar with in cases of embolism of the central artery of the retina." In a note made five months later he adds that "the disks are undoubtedly becoming atrophic."

The disease was named "amaurotic family idiocy" by Sachs in 1887 [11]. Twenty-eight of his cases occurred in fifteen families, and extensive degeneration of the cortical pyramidal cells was described. A few years after Sachs' publication, Hirsch found the same changes, not only in the cortical grey matter, but also in the grey matter of the entire central nervous system, including the spinal ganglia; and these observations were confirmed by Sachs in 1903 [12].

Further corroboration was brought by Schaffer in a recent study of eight cases [14]. The name Tay-Sachs disease, by which it is sometimes known, was proposed by Higier, and in 1908 Apert collected 106 cases, of which seventy-three occurred in twenty-five families [2]. The observation that the disease is confined to Hebrews was made by Carter [3]. There is abundant evidence to prove that the retinal changes are not present at birth.

---

[1] *Trans. Ophthalm. Soc., U.K.*, 1881, i, p. 55; 1884, iv, p. 158.

In a case reported by Koller [7], a child aged 2 months showed signs of muscular weakness; but no unusual appearance was seen except an indistinct brownish patch at the foveal spot in one eye.

In 1894, Kingdon published a case of a child aged 9 months, who showed the typical ophthalmoscopic appearances [5]. He had examined this child when it was 3 months old, and when muscular weakness was commencing. The fundus was then normal. At a second examination, when the child was 5 months old, there was a suspicious haze at each macula.

Children affected with this disease as a rule succumb at about 2 years old, but cases have been recorded of children who lived for some years longer. Sachs is surely unduly careful when he says that the disease is "generally fatal" [13]. One of his cases was 5½ years old, and Koller [7] has recorded a case of a patient who had optic neuritis at the first examination at 2 years of age, and who when nearly 4 years old could crawl about and mutter a few words. In the region of the macula there was "a slight veil-like, milky-bluish haze, gradually fading into the colour of the surrounding retina. In the centre of the opacity at the site of the fovea centralis was a cherry-red patch, not very dark, a little smaller than the disk, and with ill-defined outline."

It is well recognized that syphilis is not a factor in the disease; and hitherto no genuine case of amaurotic idiocy has been recorded in any but Jewish children. It has been asserted that the disease is restricted to Polish Jews, but this is not so; German and Polish Jews are likewise affected.

Sachs [13], in his recent paper on the disease, gives it as his opinion that the tendency to the disease is born unquestionably with the child, and that it is not acquired, nor due to a toxic cause. Afflicted children are "possessed of a nervous system so inadequate to the demands imposed upon it that its cells, after having performed their function for a few weeks or months, undergo complete disintegration."

More than one observer has recorded cases which in some degree resemble amaurotic idiocy, but which lack certain characteristic features. The family element may be absent; but it must be remembered that in but few such cases can we be sure that no more children would be born to the parents.

It has been stated above that the true disease is found only among Hebrews. Wandless [19] has recently reported, as atypical examples of amaurotic idiocy, three cases in a family known to be five-sixths Irish. One of the children was 14 years old when he died, and a second was

8 years old at the time of observation. The choroid and retina were atrophic, the latter showing pigmentation; the usual changes in the macula were not present. The autopsy showed complete optic atrophy. The retinal layers were hopelessly degenerated and no ganglion cells could be found. The ganglion cells throughout the whole nervous system were markedly degenerated, and degeneration was observed in the thymus, adrenal bodies and pituitary gland.

Parhon and Goldstein [9] record the first case observed in Roumania. The child was a Jew and was 14 months old.

Spielmeyer [17] describes a special form of the disease in which mental weakness, blindness, and a family character of the illness were present, but the former did not show itself till 6 years of age, and death did not occur until puberty.

Spiller [18] also refers to a patient whose illness was allied to amaurotic idiocy, and who lived until 8 years old.

Gordon [4] reports two cases of Russian Hebrew children, a brother and sister, in whom mental deficiency was noted in infancy, and blindness very early in life. Optic atrophy was present, and an irregular patch of absorption of choroidal pigment was observed in each eye, but there was no cherry-red spot at the fovea. He urges the opinion that anomalies in the structure and function of the ductless glands may be the real cause of the disease.

Kingdon and Russell [6], in 1897, published a paper dealing with "infantile cerebral degeneration with symmetrical changes at the macula." Five children in a family of seven were affected. In one of these cases an autopsy was made, when degeneration of the cortical pyramidal cells was found, with sclerosis of the pyramidal tracts; and a large amount of free fatty material was distributed throughout the sections. There was no evidence that these changes had occurred in other than normally developed tissues. The authors state that "so far as has been discovered the lesion is purely cortical, and it is just possible that the retinal changes are due primarily to a degeneration of the ganglion cells similar to that met with in the pyramidal cells of the cortex, and that the limited ophthalmoscopic appearance is partly due to a much greater abundance of those cells in the macula region."

Schuster [16], in a comprehensive survey of the subject, mentions five types of the disease, but he points out that, although their general similarity is remarkable, it is doubtful whether they can all be correctly included. Some stress is laid upon the fact that the disease is independent of any lesion of the blood-vessels. In the case which he reports the child died at the age of 15 months. The rods and cones were normal in places, but swollen for the most part. In the foveal region the outer nuclear layer showed the following change—namely, the cells were placed in a convex layer as though increased in the direction of the vitreous, whilst the scleral side showed concave formation. The inner nuclear layer was much altered, many cells having perished. The outer molecular and Henle's layer were very much pronounced and less firm than usual. Schuster thinks that from the various changes seen œdema of the macula region was present. He says that no absolutely normal ganglion cells were present in the retina, and he summarizes the changes of individual cells as follows: Increase in volume; nucleus eccentric, dark and surrounded by dense protoplasm; knot-points of meshwork which fills cells thicker than normal; Nissl granules not preserved; gradual vacuolation. In some cells the diseased dendrites were clearly seen.

A few years ago Schaffer, of Budapest [15], contributed an interesting and well-illustrated article on this disease. He characterizes the minute pathology of amaurotic idiocy as a cell swelling. The details are best seen at the ampulla-shaped swellings on the dendrites; the individual fibrils are made to stand out, and their wave-like form is remarkable. Schaffer insists that it is the interfibrillary material which is first attacked, and that the cell degeneration follows. This view is supported by observers who consider that it is the interfibrillary substance or hyaloplasm of the cell and dendrites which carry the nervous impulse. Glia proliferation was well demonstrated. Bielschowsky's method showed that the optic nerve was normal, the inference being that the loss of vision was caused by a central cortical lesion.

From an examination of cells stained by Nissl's method, Schaffer concludes that the cell body is filled with two forms of network—namely, the nervous framework or inner neuro-reticulum, and a non-nervous spongioplasm (Cajal). The latter is more clear in pathological cells because in these the Nissl bodies have disappeared.

It was further observed that the structure of the cell nuclei was peculiar; the nucleus was seen to consist of threads, which possibly corresponded to chromatin, and which formed a network, which occurred in part only of the nucleus. This is not seen in normal nuclei, and Schaffer thinks it possible that disease of the cell causes a temporary greater activity of the nucleus. All parts of the cortex showed a remarkable lack of fibres. Schaffer concludes by expressing the opinion that children with amaurotic idiocy possess an abnormally exhaustible nerve-cell protoplasm, which, becoming paralyzed with the strain of the earliest functions, soon degenerates.

The characteristics of Mott's two autopsies [8] were: Absence of healthy cells in brain and cord; absence of Nissl granules in most of the Betz cells; glia proliferation; sclerosis of pyramidal tracts. No fibrils were seen coursing through the cells, as described by Gordon Holmes. In the case of the second patient (Case IA), Mott found acute inflammation of the liver and pancreas, which suggested a toxic cause for the illness. In the brain there was almost complete disappearance of tangential fibres, with marked diminution of the super-radial and inter-radial fibres. The radial fibres were abundant (Weigert-hæmotoxylin). The anterior and posterior cord roots showed fairly normal bundles of myelinated fibres, indicating that, although profound changes had occurred in the cytoplasm of the ganglion cells, the axons were still capable of function. This fact is important, taken in conjunction with the statement that it is the interfibrillary substance of the cell, and not its fibrillary conducting material, which suffers the primary, and so far invariable, change in the disease.

Mott, in 1907, thought it probable that every nerve-cell in the body was affected by the morbid process, and he concluded that "this extraordinary regressive metamorphosis is brought about by a conspiracy of morbid factors—namely, an inherent racial lack of specific neuronic energy and some general alteration in the chemical composition of the blood, either by the existence in it of a neurotoxin, or by the failure of some chemical substance to form in sufficient quantity, for the building up of the nucleo-proteid substance of the nervous system."

The most important observations which have been made on the disease in England in recent years have been embodied in papers by Mott in the *Archives of Neurology*, 1907 (*vide supra*), and by Poynton, Parsons and Gordon Holmes in *Brain*, 1906 [10]. The following conclusions were arrived at by the latter authors as the result of an elaborate microscopical examination of the nervous system:—

(1) That there is strong evidence that amaurotic family idiocy is a primary disease of the nervous elements, and that the neuroglia proliferation is secondary to this degeneration.

(2) That inasmuch as the nerve-cells are relatively more affected than the fibres, and as in certain tracts there may be no visible change in the fibres, the affection may be considered a primary cell disease.

(3) That the primary change is disease of the interfibrillary protoplasm, because this is very much more severely affected than are the neurofibrils. They also conclude that: (*a*) The disease is not due to arrested development, because there is no reason why, if this were the case, such an arrest should cause a progressive and invariably fatal

disease; and because, if it were so, the symptoms would probably be evident from birth. There is also little anatomical evidence of maldevelopment. "The most easily obtained evidence of the completed development of the central nervous system—namely, myelination of the fibres—proves that the final development of the different parts of the brain is completed at different periods in a fairly long space of time, and is not ended until a few months after birth. But the examination of these brains does not indicate greater abnormalities in the regions which develop late than in those where development is completed early in intra-uterine life—e.g., the visual cortex, which is myelinated very early, is quite as severely affected as the prefrontal region in which the myelinated fibres appear late. If, however, the disease dates from the earlier months of extra-uterine life, the development of fibres which myelinate late may be checked, owing to deficiency of trophic influence from the diseased cells." (*b*) The disease is not due to bacterial toxins, but to (*c*) some inherent biochemical property of the protoplasm of the cells, as the result of which it undergoes certain changes which result in its degeneration."

The authors further state that, on pathological grounds, the disease is one "sui generis," and must be separated from the class of diplegias. In this disease the nerve-cells are reduced in number, and those which remain are shrunken and atrophic. The myelinated fibres are those most greatly affected, and, unlike amaurotic family idiocy, the disease is often associated with gross defects or macroscopical changes in the brain.

REFERENCES.

[1] Allbutt and Rolleston. "System of Medicine," 1910, viii, pp. 468-73.
[2] Apert. *La Semaine Méd.*, Par., 1908, xxviii, p. 25.
[3] Carter. *Arch. of Ophthal.*, New York, 1894, xxiii, p. 126.
[4] Gordon. *New York Med. Journ.*, 1907, lxxxv, p. 1077.
[5] Kingdon. *Trans. Ophthal. Soc.*, 1894, xiv.
[6] Kingdon and Russell. *Trans. Med.-Chir. Soc.*, lxxx.
[7] Koller. *Med. Rec.*, New York, 1896, l, p. 266.
[8] Mott. *Arch. of Neurol.*, 1907, iii, p. 218.
[9] Parhon and Goldstein. *Rev. Neurol.*, 1909, xvii, p. 895.
[10] Poynton, Parsons, and Holmes. *Brain*, 1906, xxix, p. 180.
[11] Sachs. *Journ. of Nerv. and Ment. Dis.*, New York, 1887, xiv, p. 541.
[12] *Ibid.* Loc. cit., 1903, xxx, p. 1.
[13] *Ibid.* Osler and McCrae, "System of Medicine," 1910, vii, p. 868.
[14] Schaffer. *Zeitschr. f. d. Erforsch. u. Behandl. d. jugendl. Schwachsinns*, Jena, 1909, ii, p. 19.
[15] *Ibid.* *Journ. f. Psychol. u. Neurol.*, 1905; also *Neurol. Centralbl.*, Leipz., 1905, xxiv, pp. 386, 437.
[16] Schuster. *Archiv. f. Augenheilk.*, Wiesb., 1909, lxiv, p. 1.
[17] Spielmeyer. *Neurol. Centralbl.*, 1906, xxv, p. 51.
[18] Spiller. *Amer. Journ. of Med. Sci.*, Philad., 1905, N.S., cxxix, p. 40.
[19] Wandless. *New York Med. Journ.*, 1909, lxxxiv, p. 953.

PART II.—By Dr. Mott.

Histological and Chemical Examination of the Nervous Structure.

Preface.

The histological and chemical investigations contained in this communication were carried out in the Pathological Laboratory of the London County Asylums, Claybury. A portion of the microscopic investigation was made by Dr. Carlyll under my direction, and I wish here to acknowledge my indebtedness to Dr. Fortuyn for his investigation of the cell lamination in the visual and auditory areas; to Dr. Edgar Schuster for three very admirable drawings, and to my assistant, Mr. Sydney Mann, for his chemical investigation, the results of which form an appendix.—F. W. Mott.

Introduction.

I described two cases of this disease very fully in vol. iii, *Archives of Neurology*. I mentioned there my reasons for terming the disease amaurotic dementia rather than amaurotic idiocy. I pointed out that the brains were of normal size or even larger than normal average; moreover, the convolutional pattern was in no respect like that of an idiot's or imbecile's brain; in these two cases the Sylvian fissure did not slope obliquely upwards and backwards as in the simian brain, and the superficial surface of grey matter, owing to the complexity of the convolutions, was by no means deficient in extent; neither did the microscopic examination of the cortex cerebri indicate a deficiency in numbers of the cortical cells. Moreover, I showed that the same characteristic change which is known to affect the cerebro-spinal ganglion cells also affected the sympathetic.

I have since had the opportunity of examining four such brains of children dying of this disease, three of which were from Cases I, III and VII. In all these cases the brains were of normal average weight, and the convolutional pattern complex, and in no way denoting either imbecility or idiocy. Again, I have had the opportunity of examining the central nervous system in all these cases, and I do not find a deficiency of numbers of the cortical cells. Moreover, the sympathetic ganglion cells I found showing the same change, only not so advanced as the cerebrospinal ganglion cells. It may, therefore, be concluded that the disease is an affection of the whole of the neurones of the body. It cannot be present long before birth or the convolutional pattern would not develop to its perfect form. We have to ask ourselves, therefore, what is the cause of this extraordinary disease, in which the microscopic morphological changes in the nervous system, and the clinical phenomena are so characteristic as to be unmistakeable for any other disease? Is it an acquired disease? If so, what conditions of life should limit this disease to the Jewish race. So far, I have been unable to associate it with any condition of food or environment; it appears to occur in both breast-fed and artificially-fed children, and I am inclined to agree with Sachs that there is little evidence to show that conditions of food or environment can account for the disease. Since it affects only the offspring of Jewish parents, and frequently several of the offspring of the same parents suffer with this disease and die of it, it follows that racial and family heredity do play a part, and probably are solely responsible for its occurrence.

MORPHOLOGICAL AND CHEMICAL INVESTIGATIONS IN RELATION TO THE PATHOGENESIS.

The evidence I have adduced shows that all the nervous units are present at birth, but from some cause or other their specific vital energy is so deficient that they are unable to maintain physiological equilibrium; they are unable to store any reserve of the Nissl substance which many authorities regard as the material basis of nervous energy; in consequence of this, and probably also from the swelling of the cell, the conductile mechanism itself (neurofibrils) undergoes destruction, with morphological and bio-chemical changes in the neurones. As the neurones degenerate and die the neuroglial cells proportionally proliferate and increase in size, thus altering the consistency and feel of the brain itself, which has a tough, leathery character. Do the neurones die because of an inborn deficiency of specific vital energy, or do they die because the ambient medium contains some toxic substance or lysin which destroys them, or is it because the ambient medium is lacking in some substance necessary for the development and maturation of the neurones? If it were a toxin or lysin we should rather expect it would act equally on all the cells of the body, and certainly all the nerve-cells, which is not the case. It might be a deficiency in the blood of some substance, for we know that cretinism is due to an absence of the thyroid gland; but cretinism is a condition of obvious defective brain development—in fact, we have an idiot's brain. The thyroid is not affected in Tay-Sachs disease, nor can I find any gland which is affected; and I have examined all the tissues of the body in several cases. We now come to the only other cause—viz., an inborn lack of specific vital energy of the nerve-cells, due to a racial inherited failure of the germinal determinants of the nervous system.

The neurones are perpetual elements, they are all present at birth with all their innate potentialities to respond to stimuli from without; this leads to their acquiring connexions and associations with one another; in no part of the body, excepting the reproductive organs in adolescence, do such important synthetic chemical changes take place as in the central nervous system of the human being in infancy. The Nissl substance has to be accumulated in the nerve-cells, especially in those of later phylogenetic and ontogenetic development; the myelin has to be deposited around the axial fibres of the neurones, particularly in the brain cortex, where there is scarcely any present at birth. How is this accomplished? Although stimulus from without by all the sensory avenues plays some part in accelerating this synthetic process, nevertheless every neurone has a specific autonomic inherited energy apart from stimulus. The experiments of Ross Harrison demonstrate the truth of this fact. The principles of heredity tell us that this autonomic specific energy resides in the nucleus, which is the soul of the neurone; and the experiments of Loeb upon parthenogenesis in Sea Urchin eggs show that the nucleus possesses an auto-katalytic ferment, which in the process of segmentation of the egg-cell in reproduction is capable of decomposing the lipoids of the cytoplasm and recomposing from the products a more highly phosphorized substance, nuclein.

The experiments of Verworn and others tend to show that the Nissl substance is a store of reserve neural energy, and it is contained in the meshwork of the conductile neurofibrillary substance; the observations of Macallum showed that it is a nucleo-protein containing phosphorus, and it is not unreasonable to suppose that the nucleus of the neurone produces a ferment substance which, passing into the cytoplasm, elaborates the Nissl substance out of a phosphorized lipoid obtained from the ambient medium; this basophil chromophilous material contained in the interfibrillary meshwork forming a pattern according to the arrangements of the fibrils. Verworn and his pupils' experiments indicate that this substance unites with oxygen and forms a store of neural energy. Marinesco terms it kinetoplasm.

Now, if we study the microscopic morphological characters of this disease we shall note a very remarkable and characteristic disappearance of the Nissl granules, so far as I am aware, not observable in any experimental conditions, such as ligature of blood-vessels, hyperpyrexia, or toxic conditions in mammals. Nor have I seen such change in any human pathological condition. Generally, when the cell is swollen the nucleus becomes eccentric and the Nissl granules are only found at the periphery of the cell; the chromatolysis is perinuclear, just the converse of what is found in this disease. In alcoholic and lead neuritis the anterior horn cells and the Betz cells of the cortex show perinuclear chromatolysis, which is doubtless due to reaction of injury to the axon combined with toxic conditions of the blood.

In Tay-Sachs disease, as is well known, the Nissl substance disappears from without inwards towards the nucleus, and as the Nissl substance vanishes so the cell swells up as if a process of hydrolysis had taken place. In the later stages no Nissl substance can be seen upon the dendrons, which are also swollen irregularly; no Nissl substance can be seen in the greater part of the cell body, which has undergone a bladder-like distension and often curiously and characteristically distorted into an hour-glass shape. The swelling and distortion of the cell is generally proportional to the disappearance of the Nissl substance, and finally there is only a halo of deeply stained basophil substance around the nucleus, which may now be displaced from the centre of the cell; in the case of the pyramidal cells of the cortex it nearly always takes up a position at the base of the apical dendron (*vide* fig. 13 (1)). If the sections are stained with toluidin blue or polychrome blue it will be found that the bladder-like swollen cells show a fine intracellular network which stains at the nodal points (figs. 13 (4) (5)); the network stains owing to a film of incrustation of basophil chromophilous substance. Later, when the change is more intense, the network is incomplete and unstainable areas are seen. I have, so far, only dealt with the cells stained by basic aniline dyes, but we shall not have a true picture of this disease unless many methods of staining are adopted—methods which are applicable to the demonstration of particular structures.

I shall now advance proofs that the process of decay and death of the neurones is characterized by a fatty degenerative change with destruction of the intracellular neurofibrils; there are also changes occurring in the staining reaction of the nucleus, and eventually the nucleus itself is destroyed, although that is a comparatively infrequent event.

CELLULAR CHANGES.

The nuclear membrane is sometimes stained with basophil dyes deeply; also the nucleolar and intranuclear network. Generally in cells which show a marked degree of swelling, the nucleo-plasm also stains with the basic dye. In the normal cell the nucleo-plasm is unstained by basic dyes. This microchemical basophil reaction suggests a change in the normal biochemical function of the nucleus. Are these cell changes due to a failure in the ambient medium of the necessary materials which may be required by the nuclear ferment to form the nucleo-protein Nissl substance, or is it some progressive failure of the nucleus in its reaction on the cytoplasm, causing the cell to swell up and eventually break up and destroy the intracellular network? The dendrons also show characteristic swellings (*vide* fig. 13 (8) (9), and fig. 15). Although all the nerve-cells of the central nervous system show the fatty change in some degree, including the ganglion cells of the retina, which, as we know, are developed by a bud from the forebrain, yet the neurones are not equally affected in the whole nervous system, nor even in sections of the same part; therefore, specific cell energy does play a part, whichever view is taken, regarding the pathogenesis. The chemical analysis of the brain as compared with a normal child's brain of the same age shows a deficiency in organic phosphorus and sulphur, and an increase of water-soluble extractives, containing phosphorus and sulphur. Consequently we cannot suppose that there is an increase of lipoids in the cells, due to accumulation; but it rather shows that the nucleus provides a splitting ferment, whereby the cell-plasm is decomposed and broken up, but the nuclear activity does not complete the vital process by a synthetic action; thus there may be a chemical decomposition on the way to a fatty acid, e.g., choline, glycerophosphoric acid and stearic acid, and no recomposition. A fatty acid or soap, or some lipoid, which takes the Scharlach stain in various degrees of intensity, may account for this reaction. The cells are also stained by the Marchi-Pal and Heidenhain methods in varying degrees of intensity; in all cases taking the form of very minute particles, which is additional evidence of its fatty degeneration. In the cortex of the cerebrum and in the cerebellum, where the degeneration is most intense, an immense number of granule cells (Körnchenzellen) are seen; these contain larger ruby red granules, and are seen scattered throughout the whole of the cortical grey matter of both the cerebrum and the cerebellum. At first I thought these were ganglion cells, but when Scharlach-stained specimens are counter-stained with methyl violet, with methyl blue, or logwood, it is then seen that these cells have a nucleus in the middle, and that several together lie on a ganglion cell, and almost, or completely, obscure it; sometimes two of these granule cells, joined together, occupy the position of a degenerated ganglion cell, and take on the characteristic hour-glass appearance. It may generally be said that the nerve-cells very seldom indeed show any coarse granules. It looks as if the neuroglia cells possessed a phagocytic function, as was first pointed out by Bevan Lewis, and I have seen in both silver preparations and preparations stained by other methods (photomicrograph 5) appearances suggesting phagocytic activity of the neuroglia cells.

Two of the three brains examined showed numbers of granule cells in the white matter as well as the grey, this showing that these cannot therefore be ganglion cells which have undergone this degeneration. Numbers of cells filled with deep red stained fat granules are also to be seen in the perivascular lymph sheaths, as if the endothelial cells or cells of the adventitia had taken up the fatty matter. Curiously enough, not much change was found in the cerebrospinal fluid withdrawn during life by lumbar puncture.

All the facts show that in this disease there is a fatty degeneration of the cytoplasm of the neurones, which I find Alzheimer describes in a recent publication. The satellite cells found in the perineuronal spaces, or neuroglia cells, show a fatty change, which is probably of the nature of an infiltration; that is to say, these cells have devoured the fat produced by the degeneration of the neurones; the fatty contents of these cells indicate a further process of change, judging from the deeper coloration by the Scharlach dye, and the larger size of the globules (*vide* Plate I). The appearance of the nerve-cells of the cortex makes it possible that the late stages of hydrolysis convert the cytoplasm into a thick emulsion, giving the cell that peculiar appearance which is not unlike that of the cortical cells of the suprarenal body, which we know contains a phosphorized fatty substance that stains deeply with Scharlach R. Moreover, I have noticed that when there is a considerable amount of glia fibrillation of the superficial layers of the cortex, and the microscopic examination of sections of the cortex shows a marked condition of fatty change in the cells (*vide* photomicrograph 8), that the brain substance is semifluid, like cream, so that the tough, leathery, fibrillated superficial layer of the cortex can be peeled off from the subjacent semifluid, creamy grey matter.

DETAILED DESCRIPTION OF FIGURES AND PHOTOMICROGRAPHS.

I will now describe, with the aid of the accompanying figures and photomicrographs, the cell changes which occur when sections have been stained by particular methods.

Fig. 13, a number of cells showing morphological changes.

(1) A large pyramidal cell of the cortex from a Scharlach-stained section counterstained with hæmatoxylin; the whole cytoplasm is stained an orange-pink; it presents a fine granular appearance. The cell is swollen, the nucleus is pushed up towards the base of the apical dendron, and from the base of the cell there is an oval, bladder-like swelling. (Magnification 500.)

(2) A similar pyramidal cell stained with Weigert-Pal; the cytoplasm is filled with minute blue-stained globules. The cortex stained by Marchi method would show similar globules stained blackish-grey. Again, Heidenhain's hæmatoxylin stains these fine globules blue. These fine granules thus stained may be protoplasmic particles covered with a film of fat or soap; they are the same as the fine granules seen in (1). (Magnification 500.)

(3) A cell stained by Scharlach R or Sudan III, showing much larger granules stained deep red. Most of these, and they are very abundant in the cortex, are Alzheimer's Körnchenzellen, and are either neuroglia cells or satellite cells filled with fatty droplets. It is very difficult to say whether any of the ganglion cells ever present such an appearance. (Magnification 500.)

(4) A large cortical pyramidal cell stained with toluidin blue as recommended by Schaffer, to demonstrate the intracellular network; it also shows the swelling and characteristic almost hour-glass distortion; the Nissl substance has almost entirely disappeared; there is still a little incrustation of the nodal points of the intracellular network around the nucleus. (Magnification 500.)

(5) A large spinal anterior horn cell; the same description applies as above in 4. (Magnification 500.)

(6, 7, 8, 9) Various pyramidal cells of the cortex stained by Cajal's neurofibril method. Sections were also stained by Bielschowsky's method, but the results as regards the points to be described are the same. (6) shows the basal neurofibril process split, the fibrils passing down each side of a pale substance which appears to consist of globules or round particles; the nucleus is connected with fibres coming in from the apical dendron. In (7) an appearance which is very rarely seen is

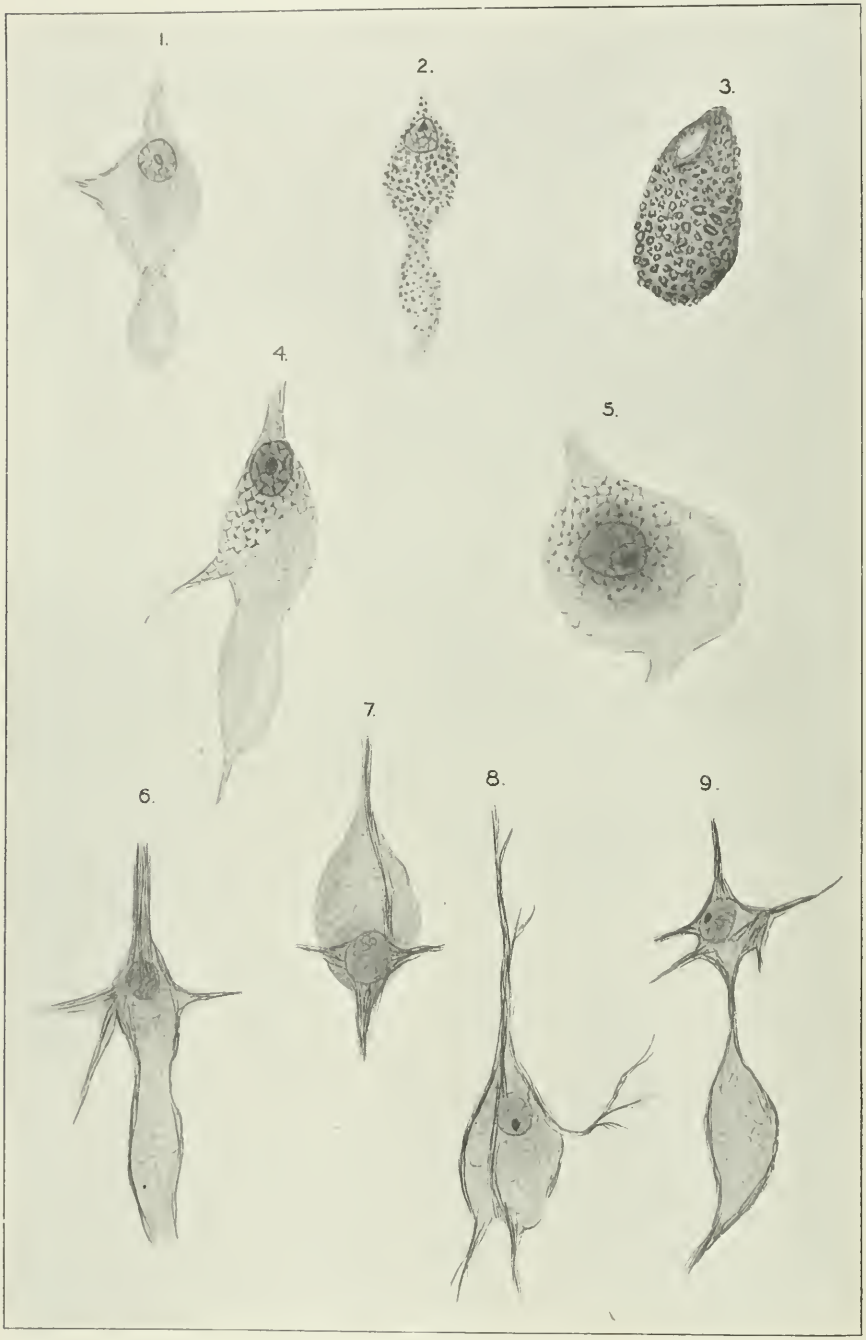
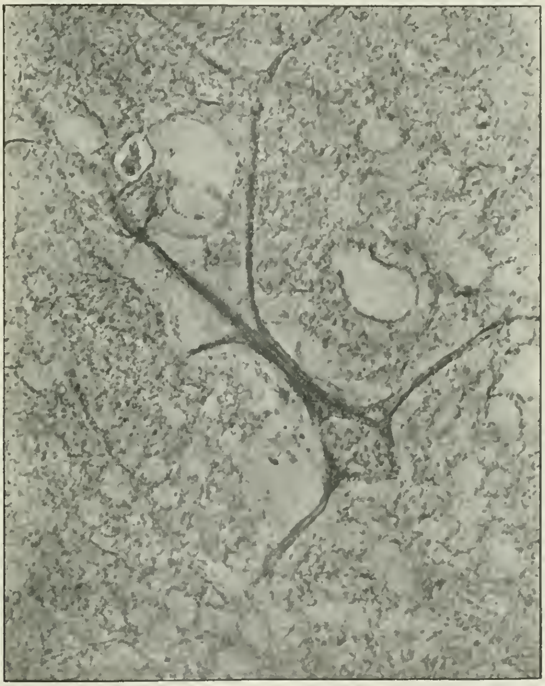


FIG. 13.

shown; here the nucleus is apparently held in position by intact neurofibrils passing across the cell from one lateral dendron to the other. (8) shows an appearance not infrequently seen of the fibrils of the apical dendron passing along the periphery of the cell to other processes. (9) shows a large ovoid swelling on one of the processes of a ganglion cell as if the hydrolytic process had commenced in the process instead of the body of the cell. (Magnification 400.)

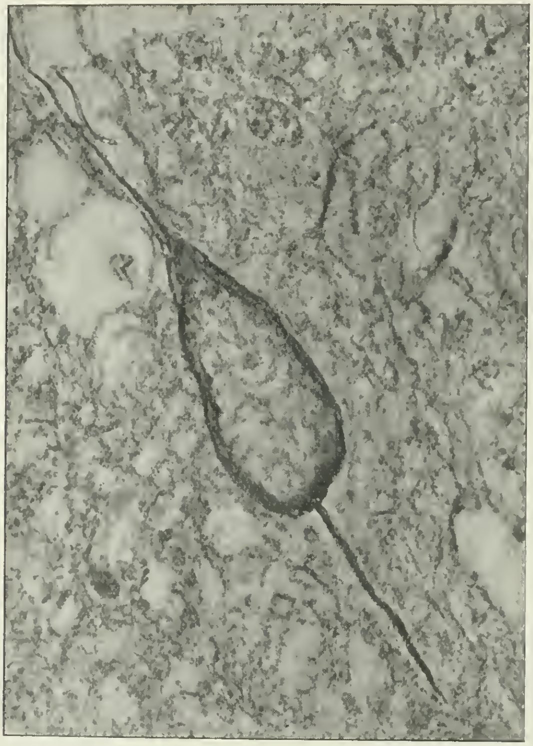
Photomicrograph 1.—A nearly normal cortical pyramidal cell showing abundant neurofibrils in the processes; this was the only one seen in a section showing hundreds of cells in a state of degenerative decay. (Magnification 1,080.)



PHOTOMICROGRAPH 1.

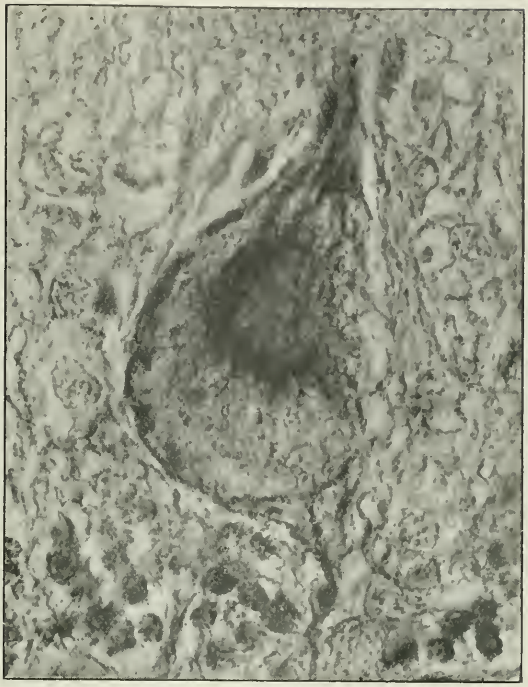
Photomicrograph 2.—A pyramidal cell in which the only fibrils are seen at the periphery and in the two processes. The fibrils seem to be continuous between the apical dendrons and the axon. The whole central portion of the cytoplasm has a coagulated structureless appearance. (Magnification 1,180.)

Photomicrograph 3.—A cell of Purkinje stained by the neurofibril method. The fibrils can be seen passing from the apical dendron to the nucleus and around the periphery of the cell continuous with the fibrils of what appears to be the axon, arising from the lower portion of the distorted, flask-shaped cell. It will be observed that all around the nucleus there is a complete absence of fibrils. I attribute this to the disappearance of the basket-like terminal arborization of the stellate cells, which a subsequent illustration will show are degenerated earlier than the Purkinje cells. (Magnification 1,260.)

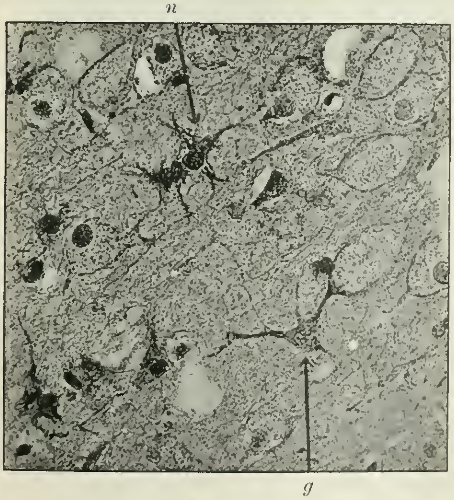


PHOTOMICROGRAPH 2.

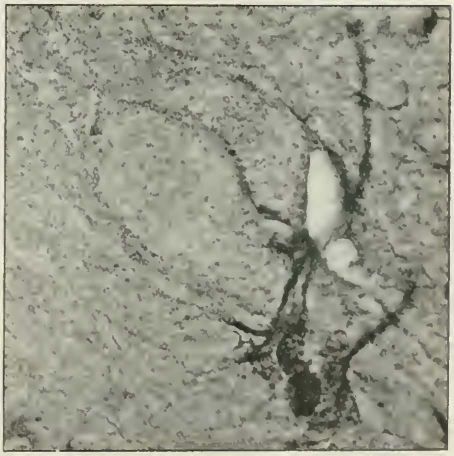
Photomicrograph 4.—Cortex stained by neurofibril method. Numbers of bladder-like cells are seen with a fine granular central portion and externally surrounded by dark-stained fibrils; *g* shows a ganglion cell which apparently is intact as regards its neurofibrils except that one process appears to be surrounding a degenerated cell. It will be observed that this nerve-cell differs markedly from the black-stained



Photomicrograph 3.



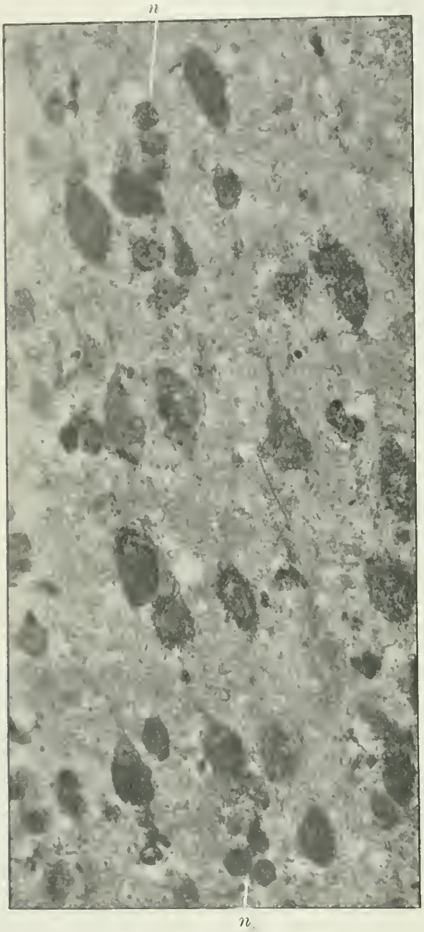
Photomicrograph 4. (× 360.)



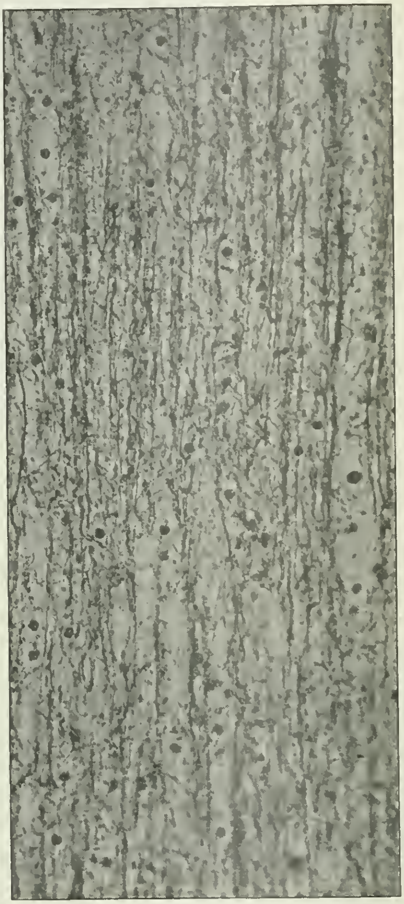
Photomicrograph 5. (× 810.)

neuroglia cells *n*, of which there are great numbers undergoing proliferation. (Magnification 360.)

Photomicrograph 5.—A large neuroglia cell, showing two nuclei and large stout processes, apparently stuck on to and grasping with its processes a large degenerated ganglion cell, but some of the fibrils which seem to come from the glia cell can, by close observation, be seen to be the undestroyed peripheral neurofibrils of the ganglion cell. (Magnification 810.)

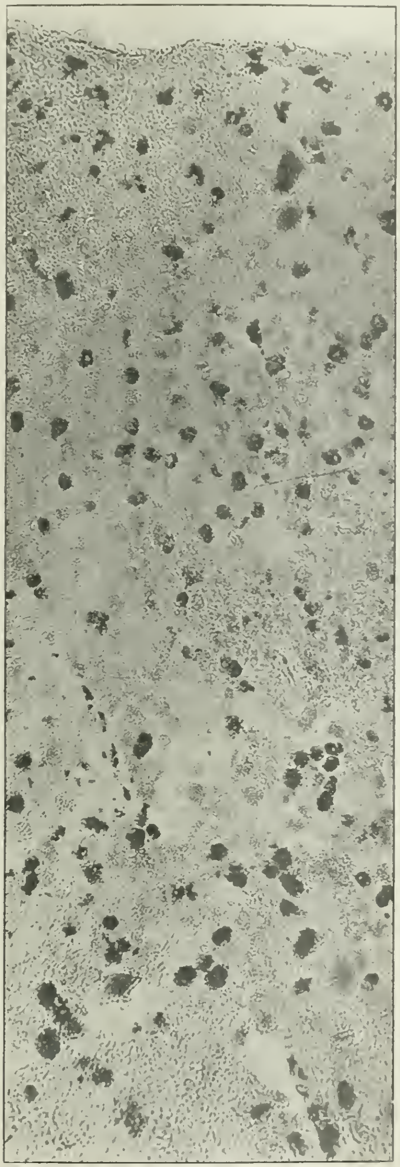


Photomicrograph 6.

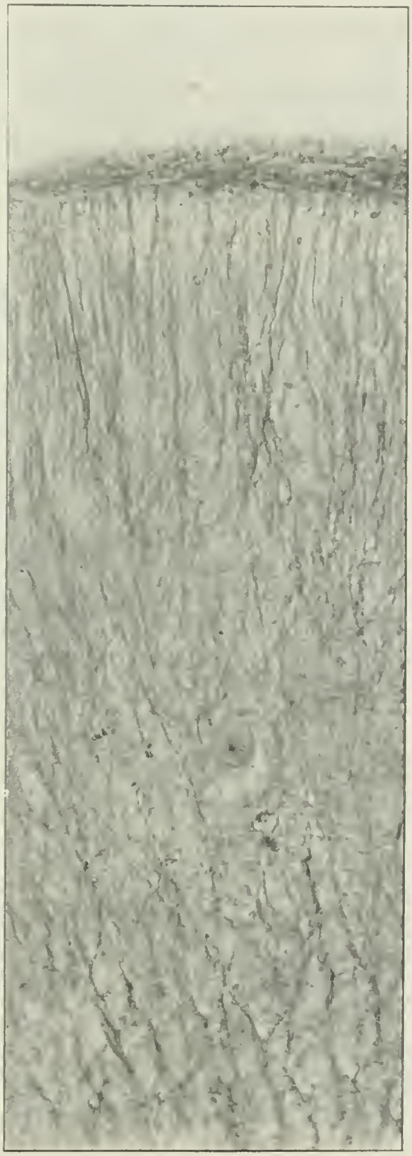


Photomicrograph 7.

Photomicrograph 6.—Cortex stained by Weigert (neuroglia method), subsequently by iron hæmatoxylin (Heidenhain's method). The swollen, distorted, bladder-shaped pyramidal cells are stained purple owing to the fine fatty globules. The nucleus is not stained deeply and it can be seen pushed up to the apical dendron. At the bottom of the picture and at the top can be seen three cells in groups *n*, round in form; these are Körnchenzellen, or granule cells, and with Scharlach or acid fuchsin are stained deep red. In the middle of the picture several can



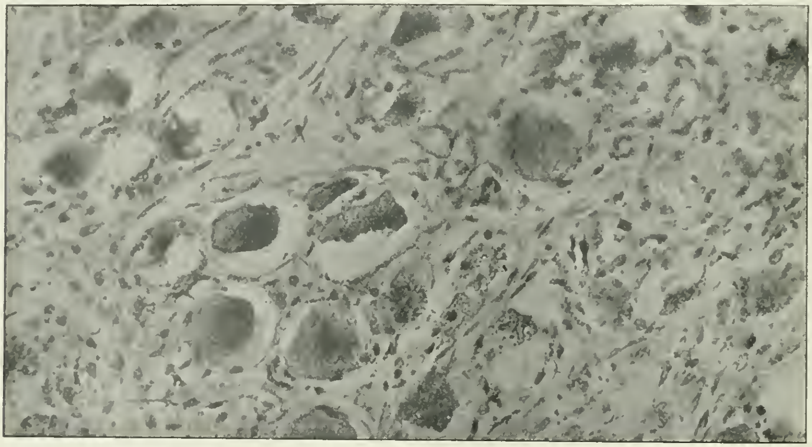
Photomicrograph 8.



Photomicrograph 9.

be seen sticking on the degenerated ganglion cells. No axis cylinder processes can be seen. (Magnification 380.)

Photomicrograph 7.—A Weigert-Pal-stained section of the subcortical white matter of the ascending frontal convolution; it will be observed that there are no coarse fibres characteristic of this cortical area present. This indicates that the large coarse fibres which form the pyramidal system are absent; this fact may be correlated with an absence of fibres in the crossed and direct pyramidal tracts of the spinal cord. All the fibres appear to be attenuated and most of them show degenerative varicosities. These fibres are all radial fibres and it will be observed that there is a complete absence of inter-radial association fibres. (Magnification 330.)



Photomicrograph 10.

Photomicrograph 8.—Section of the cortex stained by Scharlach R. The deeply stained cells are Körnchenzellen, the fainter stained cells with fine granules are the ganglion cells (*vide* also Plate I). (Magnification 180.)

Photomicrograph 9.—Section of the cerebellum stained by Ranke's Victoria blue method. Enormous increase of Bergmann's neuroglia fibrils is seen and a dense felting of the surface. (Magnification 450.)

Photomicrograph 10.—Section of the sympathetic (inferior cervical) ganglion showing peripheral chromatolysis of many of the cells; the change in the sympathetic ganglion is not so advanced in this case as in others that I have examined.

Fig. 14.—Section of cortex stained by Weigert-Pal method showing the terminal arborization of degenerated fibrils around cells, the bodies

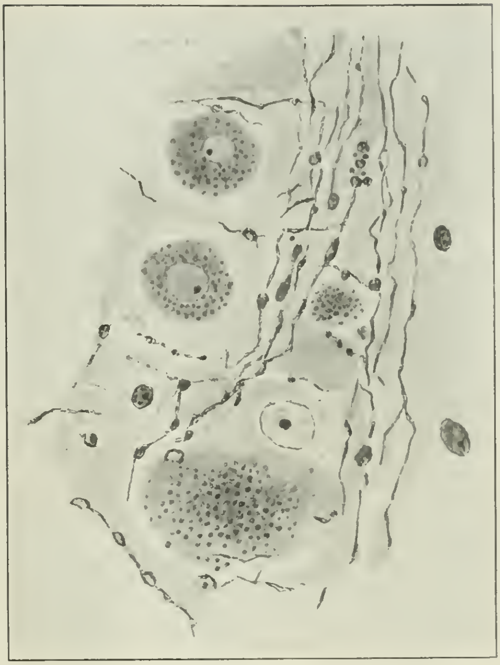


FIG. 14.

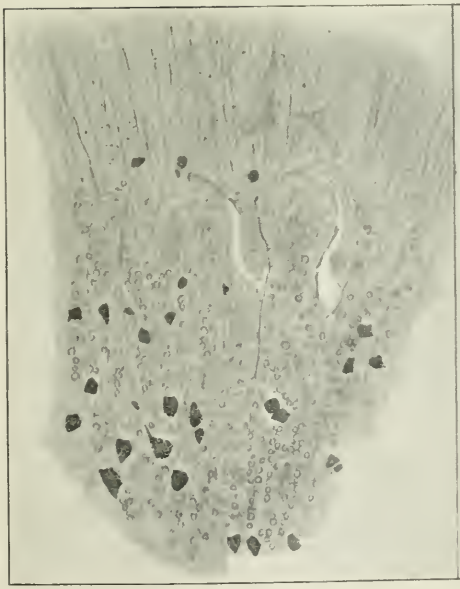


FIG. 15.

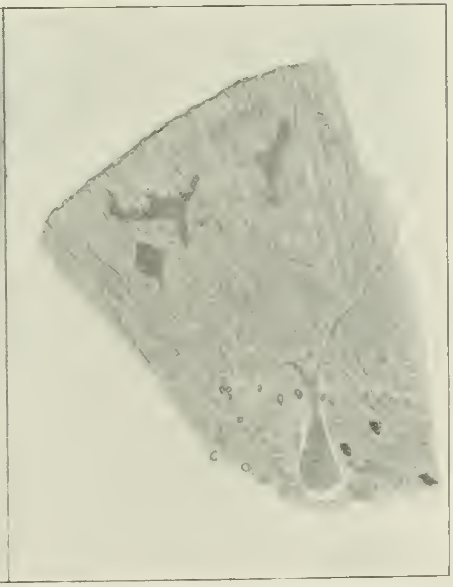


FIG. 16.

of which show minute globules stained by the dye in the same manner as the degenerated nerve fibrils. (Magnification 800.)

Fig. 15.—Frozen section of cerebellum, after hardening in formalin a few days, stained with Scharlach. A number of deeply stained smaller cells are seen amidst the granules which are unstained, many of them doubtless are Körnchenzellen; some are apparently degenerated stellate cells—viz., the cells, the axon of which form a basket-work around the Purkinje cells; the degeneration of these cells would account for the absence of fibrils seen in the greater part of the body of the Purkinje cells (photomicrograph 3). The Purkinje cells are visible because stained a pinkish orange, and the dendrons are swollen and similarly stained owing to fine granules of fatty substances. (Magnification 200.)

Fig. 16.—Another portion of the same section as fig. 15, but showing more distinctly the fine particles of fatty substance in fragments of the swollen dendrons near the surface.

As a control sections of brain of a normal child of the same age were prepared and stained by Scharlach R and Sudan III; the cells are not visible, but the white matter is stained more intensely a deep orange red.

Examination of the Retina.

Portions of the retina were removed from one eye, Case VII (*vide* p. 165), and preserved for a few days in 10 per cent. formol solution. They were then floated into distilled water, and transferred to an alcoholic solution of Scharlach R, or Sudan III, in which they were left for some hours. They were again carefully floated into weak alcohol to wash away the excess of stain, caught on a cover-glass with the ganglionic layer upwards, and mounted in Farrant's solution.

Fig. 17.—Drawing by Schuster. (Magnification 240.)

The ganglion cells in the retina can be distinctly seen, owing to their deeper staining. They appear to be much more numerous in some places than others. They vary considerably in size, as the drawing (fig. 17) shows. As this method shows the whole cell it must be either assumed that as in the spinal ganglion there are cells of varying size in the retina or that the cells differ in size owing to the pathological change. Against this is the fact they are all uniformly stained a deep orange colour, in many places on an unstained background. Moreover, under an apochromatic 2 mm. 140, 4 comp. ocular, the pathological change appears to be the same. I am therefore led to believe that the difference in size is due to different-sized ganglion cells in the retina, a fact which sections would not show.

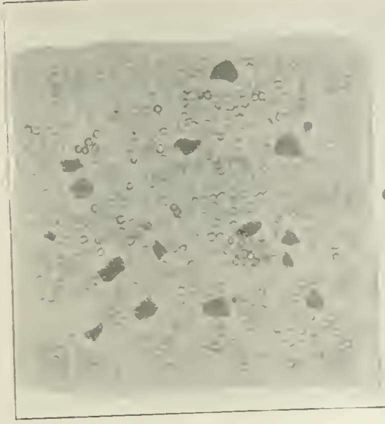


FIG. 17.

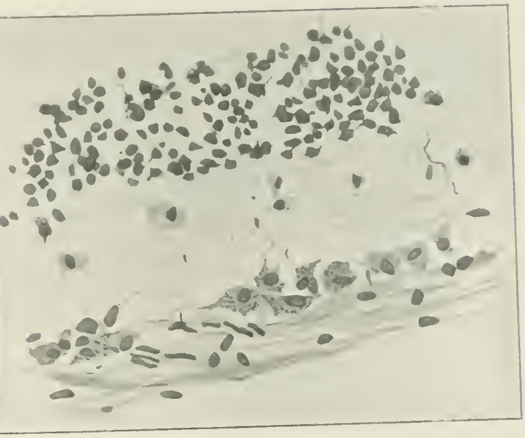


FIG. 18.

Fig. 18.—Stratum opticum. Ganglionic layer, inner molecular and inner layer of granules. (Magnification 450. Schuster.)

The other eye, after hardening in 10 per cent. formol, was embedded in celloidin, and sections through the whole eye were cut and stained by polychrome blue, Nissl, Giemsa, and Van Gieson methods. The only definite observable changes were in the ganglion layer. The cells appear abnormal in shape, the processes are either indistinctly seen or obscure. The cells lie in confused clumps, in places appearing as if joined together. The nuclei and nucleoli are well stained, but the cytoplasm is poorly stained, and appears to consist of a fine intracellular network faintly incrusted with basophil substance. No Nissl granules are seen. Here and there ganglion cells can be seen with a swollen process.

The observations of Dr. Fortuyn show that the whole cortex is similarly affected.

DESCRIPTION OF THE COLOURED PLATES.

The coloured Plates I and II are reproductions of drawings by Miss Kelley to show the appearances presented by Scharlach-stained specimens. It will be observed that the ganglion cells, wherever they are taken from, are stained a reddish-orange or orange colour. The granule cells are stained a deeper ruby-red. I may add that I have employed some of the methods of staining recommended by Lorrain Smith,[1] but I have not found that they yielded such good differentiating results as the Scharlach and Sudan III, or the fuchsin method of Alzheimer.

---

[1] Since this paper was completed I have received an important communication explaining the action of these various dyes on fats and lipoids: J. Lorrain Smith and W. Mair, "Fats and Lipoids in relation to Methods of Staining," *Skand. Arch. f. Physiol.*, Leipz., 1911, xxv, p. 247.

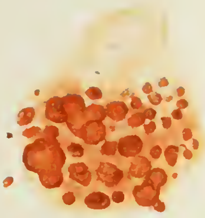
Explanation of Plate I.

Fig. 1 shows a cortical pyramidal cell swollen up, and filled with large red-stained fat globules of uneven size, and some coalescing. At the upper part of the cell is a pale unstained portion, which is apparently the nucleus. I found only very rarely ganglion cells showing this appearance. The vast majority of cells with red-stained large globules were the so-called granule cells (Körnchenzellen).

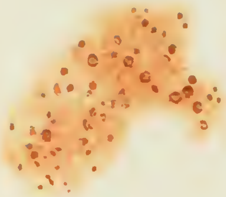
Fig. 2.—This is a distorted pyramidal cell, in which there are scattered small red globules. These, again, are rarely met with, and, as I have previously explained, many cells look like ganglion cells when only stained with the Scharlach, but which when counterstained by logwood or methyl violet prove to be one or several granule cells lying on the decayed ganglion cell, and I would not deny the possibility of this being the case in respect to both this (fig. 2) and that of fig. 1.

Fig. 3.—This is a ganglion cell in an advanced stage of fatty degeneration. The whole cytoplasm is permeated with fine globules of fat. (Magnification 600.)

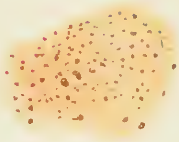
Fig. 4.—A group of small cells of anterior cornu of spinal cord, stained by Scharlach. The cells are stained a pinkish-orange colour. One or two show a very fine granulation appearance. (Magnification 600.)



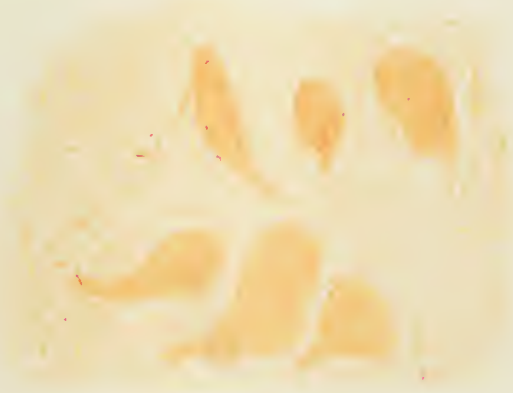
1



2



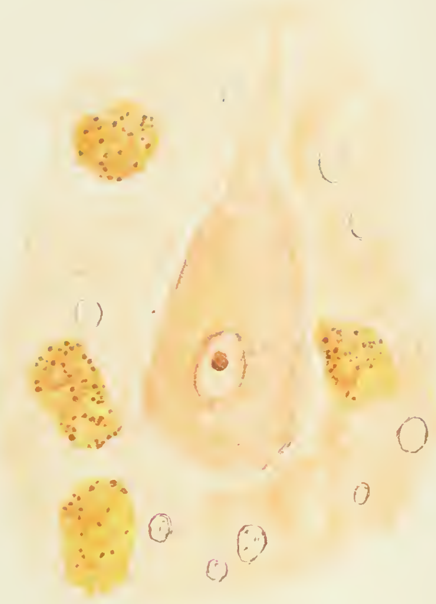
3



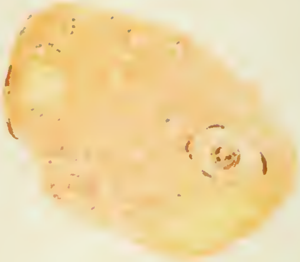
4

CARLYLL & MOTT: *Seven Cases of Tay-Sachs Disease.* Plate I.

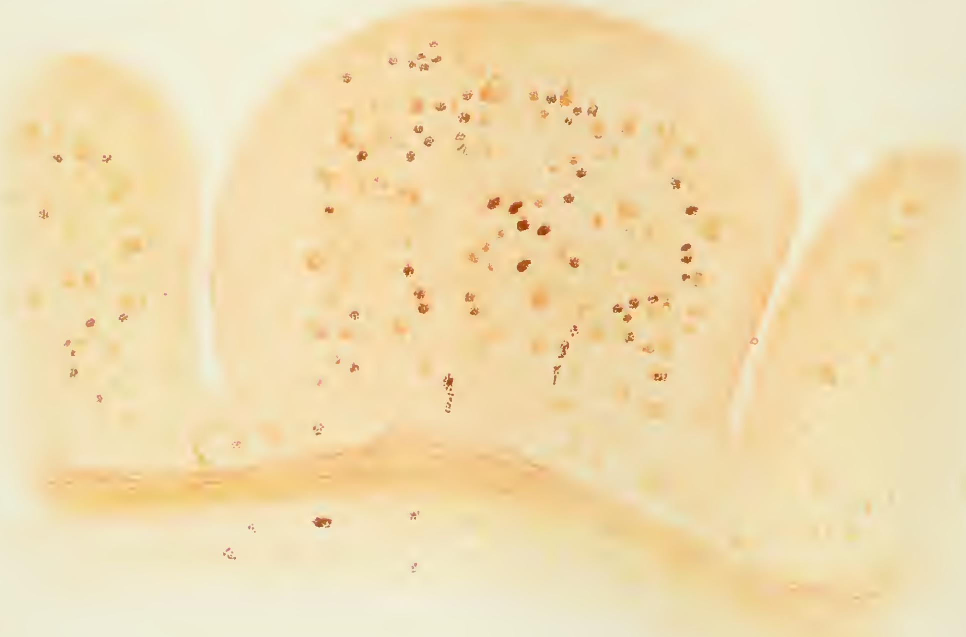
PROC. ROY. SOC. MED
Vol. IV. No. 5.
Pathological Section.



2



3



1

CARLYLL & MOTT: Seven Cases of Tay-Sachs Disease. Plate II.

Explanation of Plate II.

Fig 1.—Section of cerebellum stained by Scharlach R. The cells of Purkinje are seen stained a light orange-red; also a number of much smaller cells are similarly stained. But besides these paler-stained cells, which are degenerated ganglion cells (stellate cells, second type of Golgi), there are scattered about a number of deep ruby-red cells arranged in groups and sometimes in rows. These are granulation cells—amœboid neuroglia cells filled with fat globules. This particular preparation from Case VII does not show any of these granule cells in the white matter; but in the other two cases which I have examined, numbers of these deeply stained granulation cells (Körnchenzellen) exist in the white matter; especially are they found in numbers in the perivascular sheaths. The small granules (nerve-cells) which form such a prominent histological feature of the cerebellum did not take the Scharlach stain. (Magnification 50.)

Fig. 2.—A Purkinje cell in an early stage of degeneration. It is covered all over with very fine pinkish-orange granules. In the immediate neighbourhood are four cells containing coarse red globules. I believe they are ganglion cells, but they may be granulation cells (Körnchenzellen) in an early stage of fatty change.

Fig. 3.—A Purkinje cell showing a rather more advanced stage of fatty change; it is more deeply stained red. (Magnification 600.)

It will be observed that the ganglion cells vary in depth of coloration by the Scharlach dye, and the deeper the colour the more obvious do the particles of fatty substance become. There is also a parallelism between the distortion in shape of the ganglion cell and the evidences of the fatty change. The fact that all the ganglion cells, with the exception of the granules, are degenerated in the cerebellum may account for the apparent absence of the basket of fibrils around the base of the cells of Purkinje; this absence of fibrils is shown in photomicrograph 3. If sections are placed first in alcohol, then in ether for a short time, the cells no longer stain, all the fat having been dissolved out.

Dr. Carlyll, in his historical summary, has given a résumé of previous observations on the morphology of this disease, but it is necessary for me to refer to some important observations by Alzheimer concerning the fatty change in ganglion cells in this disease, and in the amaurotic idiocy described by Spielmeyer and Vogt. These observations I had no knowledge of when I wrote this paper, but I have since found a description very similar to the above in Alzheimer's[1] valuable work, which he kindly sent me. He also gives a number of valuable methods of staining which I shall adopt in the study of a case that has recently died, under the care of Dr. Hume, of Newcastle, who has forwarded me the brain and the eye for examination. This will form the subject of a joint paper on the histology by the most recent methods.

CONCLUSIONS.

(1) Reasons are given why the term "idiocy" should be abandoned; it will be better to adopt the name "Tay-Sachs disease" until the pathogenesis is known.

(2) Reasons are stated why it is probably a failure in the germinal determinants of the nervous system peculiar to the Jewish race.

(3) The morphological and chemical investigations in relation to the pathogenesis are discussed, also the hypothesis is put forward that it may be due to a failure in the nuclear material of the neurones to build up the nucleo-protein Nissl substance out of lipoid substances contained in the cytoplasm, which first have to be decomposed by a nuclear ferment. The autokatalytic ferment action of the nuclear

---

[1] "Beiträge zur Kenntnis der pathologischen Neuroglia und ihrer Beziehungen zu den Abbauvorgängen im Nervengewebe." Nissl und Alzheimer, "Histologische und Histopathologische Arbeiten über die Grosshirnrinde," Jena, 1910, iii, pp. 401-554.

material of the fertilized ovum described by Loeb is considered as affording a somewhat analogous chemical process.

(4) Evidence to show that there is a progressive failure of Nissl substance proceeding from without inwards towards the nucleus and a corresponding accumulation of a fatty substance of the nature of a lipoid, which, accompanied by a process of hydrolysis, would cause a swelling of the cell and destruction of the intracellular neurofibrillary network.

(5) The chemical analysis does not throw much light upon the question; the diminution of the lipoid forms of phosphorus and sulphur is probably due to the diminution of myelin, owing to failure of development of the myelinated fibres. The corresponding increase of extractive forms of phosphorus and sulphur may be possibly due to a breaking down of the more complex to simpler forms of lipoids.

(6) The morphological changes are quite characteristic of the disease. All the ganglion cells stain with Scharlach in varying degrees of intensity, more or less intense in proportion to the degree of swelling and obvious morphological change; they also stain with Marchi, Weigert-Pal, Heidenhain—in fact, all the methods which stain the myelin sheath or fat. They do not, however, stain satisfactorily by Marchi, like degenerated myelin does when the process of decomposition has been complete to choline, glycerophosphoric and oleic acid. Consequently it is more correct to say that the cytoplasm *may be* on *the way* to this complete decomposition.

(7) Whereas the ganglion cells very rarely show *coarse* ruby-red globules of stained fatty substances, there are, especially in advanced cases, immense numbers of cells containing these coarse globules, and forming what Alzheimer terms Körnchenzellen; they are neuroglia cells which have taken up the fat from the dead and decayed ganglion cells, to which they may be seen sticking sometimes in little, closely aggregated groups indicative of active proliferation. It is probable that they have the power of decomposing this lipoid of the dead ganglion cells and possibly of recomposing nuclear substance necessary for their proliferation out of it.

(8) Other methods of staining—e.g., toluidin blue, Cajal silver, or Bielschowsky—show that the intracellular fibrils are ruptured and destroyed by the swelling leaving only the peripheral neurofibrils, which can be followed from the dendrons in their course around the swollen cell to other dendrons or to the axon. In the cortex, the fibrils of the apical dendrons are seen proceeding to the nucleus, which is usually

forced up into the apex of the pyramid. It is possible that the cytoplasm is of the nature of a thick emulsion, each particle consisting of a plasm covered with a film of fat or soap.

(9) The cells of the retina, when this structure is stained with Scharlach, show a similar change to the nerve-cells of the central nervous system: the cells are of varied size, apparently.

(10) In two of the three brains examined there was an accumulation of granulation cells (Körnchenzellen) along the course of the blood-vessels, also endothelial and connective tissue cells of the perivascular sheath could often be seen filled with the dark, red-stained fat globules.

(11) Any of the methods employed for demonstrating neuroglia shows an enormous overgrowth of fibrils, especially in the superficial layers, where it forms a dense feltwork both in the cerebrum and the cerebellum. This overgrowth is proportional to the duration of the disease. Throughout the grey and white matter the proliferated neuroglia cells of large size, with coarse and branching fibres, are seen in great abundance embracing and sticking to the ganglion cells, out of which they appear to be absorbing the phosphorized substances necessary for nuclear proliferation.

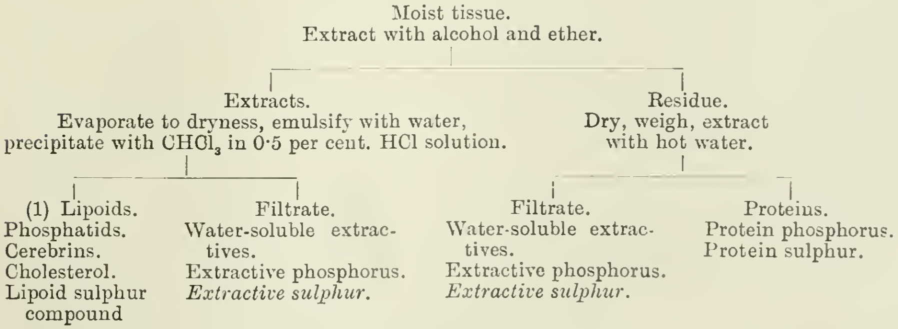
APPENDIX.

THE CHEMICAL EXAMINATION OF THE BRAIN IN TWO CASES OF AMAUROTIC IDIOCY, AND COMPARISON WITH THE NORMAL BRAIN.

By SYDNEY A. MANN.

IN a previous investigation[1] of the brain in two cases of this disease it was shown that in the more advanced case there was a decrease of nucleo-protein which might be associated with the disappearance of the Nissl substance in the neurones, and an increase of simple protein corresponding to an increase of glia fibrils; also in both cases the lipoid sulphur and phosphorus showed a diminution, but the smallness of the size of the samples of brain matter used in that investigation made it necessary that further analyses should be made on larger samples before any conclusions could be based on the latter results.

Since that time the methods for the chemical investigation of the brain have been revised and elaborated[2] and the following research has been made on larger samples, using the methods in their amended form. The methods employed, with full analytical technique, have been already described[2] but it appears necessary to give a résumé of the scheme of analysis. By means of solvents the chemical constituents of the brain tissue are separated into four general groups: (1) lipoids, (2) extractives, (3) inorganic constituents, (4) proteins. The following scheme explains this separation:—



Moist tissue.
Extract with alcohol and ether.

Extracts.
Evaporate to dryness, emulsify with water, precipitate with $CHCl_3$ in 0·5 per cent. HCl solution.

Residue.
Dry, weigh, extract with hot water.

(1) Lipoids.
Phosphatids.
Cerebrins.
Cholesterol.
Lipoid sulphur compound

Filtrate.
Water-soluble extractives.
Extractive phosphorus.
Extractive sulphur.

Filtrate.
Water-soluble extractives.
Extractive phosphorus.
Extractive sulphur.

Proteins.
Protein phosphorus.
Protein sulphur.

---

[1] *Archives of Neurology*, 1907, iii, p. 218.

[2] *Archives of Neurology*, 1909, iv, p. 174.

The variations of the elements phosphorus and sulphur in these groups were investigated for the following reasons. Phosphorus is the radical which seems to play the rôle in the building up of the most complex constituents of the cell, the nucleins and phosphatids, and the variation in its distribution between these and the water-soluble forms should therefore give an indication of the amount of formation or destruction of these important cell constituents. Sulphur occurs in the body in varied stages of oxidation, and the variations in the different stages probably offer a means of estimating the extent to which oxidizing reactions are taking place in the tissues. Sulphur occurs as –SH or cystin sulphur in proteins, as sulphonate or taurin like sulphur, as ethereal sulphates and inorganic sulphates; it enters the organism mainly as unoxidized or cystin sulphur and leaves in an oxidized form as inorganic sulphates.

The following forms of phosphorus and sulphur have been estimated in this investigation.

(1) *Lipoid Phosphorus.*—Phosphorus attached to the phosphatid molecules (lecithins, kephalins, sphingomyelin).

(2) *Extractive Phosphorus.*—Water-soluble inorganic and organic phosphorus compounds; previous experiments have tended to show that the water-soluble phosphorus compounds extracted from the protein fraction are mainly inorganic, but it is difficult to say how far the organic forms may not have been split up in the process of estimation.

(3) *Protein Phosphorus.*—Phosphorus in combination with proteins, nucleo-proteid.

(1) *Lipoid Sulphur.*—Sulphur in combination with lipoids; cannot be removed by cold dilute hydrochloric acid, but splits off as sulphates on prolonged boiling with dilute hydrochloric acid.

(2) *Neutral Sulphur.*—Does not split off as sulphuric acid on prolonged boiling with dilute hydrochloric acid. Does not form lead sulphide on treatment with alkali and lead acetate. Is not precipitated by phosphotungstic acid except to a very slight extent (about 5 per cent). Reacts with *a* naphthyl isocyanate like an amino acid, and represents therefore an intermediary state in the oxidation of cystin to sulphuric acid or ethereal sulphates, probably of the nature of taurin.

(3) *Inorganic Sulphur.*—Precipitated directly by barium chloride in acid solution. In this investigation the inorganic and neutral sulphur have been classed together as extractive sulphur, previous work having shown that the amounts of inorganic sulphur are so small that their investigation is a difficult matter.

(4) *Protein Sulphur.*—Sulphur in combination with proteins.

One hundred gramme samples of the minced whole brain from two cases of amaurotic idiocy, with a similar sample of the normal brain of a child of about the same age, have been investigated according to the above scheme, with the following results. Table I shows an approximation of the main groups of constituents of the brain tissue in percentage of total solids :—

TABLE I.

	Age 18 months Normal brain	Age 20 months Amaurotic idiocy Case III	Age 17 months Amaurotic idiocy Case IV
Simple protein	35·0	29·6	30·8
Nucleo-protein	10·3	13·7	10·0
Phosphatids	28·0	21·9	19·8
Cerebrins	13·9	15·9	12·2
Cholesterol	11·0	11·8	8·9
Extractives	Not estimated.		
Ash			
Total phosphorus	1·503	1·357	1·318
Total sulphur	0·49	0·396	0·47
MOISTURE	81·52	83·44	79·31

The analysis made on 100 grm. samples of the uniformly minced brain confirm the results previously obtained from the analysis of smaller samples of the grey and white matter separately, that there is a decrease of the phosphatids in the brain of these cases, with a diminution of the total phosphorus and sulphur. The figures for the normal brain are in agreement with former analyses made on brain from children of about the same age with the exception that the nucleo-protein approximation is somewhat low.

The results of one of the cases previously reported showed a diminution of nucleo-protein with a corresponding increase of simple protein in the grey matter; this change is not apparent in the above cases in which uniform mixtures of the grey and white matter were analysed. This does not, however, negative the former result as the amount of phosphorus in the protein fraction is very small, and when the white matter (which has not been found deficient in nucleo-protein) is included, the departure from the normal is greatly increased, and unless it is very marked may be beyond the limits of chemical analysis. As further cases are obtainable this point will be further investigated on large samples of the grey matter separately.

Except in the case of phosphatids, the slight variations in the other constituents—cerebrins, cholesterol, &c.—may be explained as the result of nutritive changes. The above figures, however, are approximations of the general groups of constituents, and a more accurate idea of the chemical abnormalities met with in the pathological brains is shown by the following Table II giving the percentage distribution of the elements phosphorus and sulphur. Tables II and III show the distribution of phosphorus and sulphur in percentage of total phosphorus and sulphur.

TABLE II.

	Normal	Case III	Case IV
Protein phosphorus	3·9	5·7	4·3
Lipoid ,,	72·2	62·5	58·4
Extractive ,,	23·7	31·7	37·2

TABLE III.

	Normal	Case III	Case IV
Protein sulphur	59·2	61·2	59·4
Lipoid ,,	19·4	14·8	12·5
Extractive ,,	21·4	24·0	28·1

The above table shows that there is *a marked relative diminution of the lipoid forms of phosphorus and sulphur with a corresponding increase of the extractive forms*, the protein and sulphur and phosphorus showing no marked change.

The Pathological Anatomy of a Case of Amaurotic Family Idiocy.

By GORDON M. HOLMES.

ABOUT three years ago I received from Dr. Parkes Weber for microscopical examination several small pieces of the cortex and the adjacent white matter of the brain, as well as two pieces of the cerebellum, from a male child who had died in the German Hospital while under the care of his colleague, Dr. Karl Fürth.

The history I received from Dr. Parkes Weber was that the child (J. M.), who died when 2 years and 10 months of age, was admitted in a very weak condition in which he had been for a considerable time. He lay constantly in a position resembling that of tetany, but the hands were in the "fist" rather than in the "accoucheur" position. There was also rigidity of the neck, and he had some difficulty in swallowing. The degree of spasm varied from time to time. He died about a month after he was admitted to hospital from an attack of acute pneumonia. (The family history may be obtained from Dr. Carlyll's account of the case of the boy's sister, who died later in the Evelina Hospital for Children, *see* p. 156.)

At the autopsy it was found that the brain was abnormally large: it weighed 1,450 grm., which exceeds the average normal weight of the adult male brain. It was also very firm and hard to touch, but there was no gross abnormality visible in it to the naked eye.

The gyri of the pieces of brain I received were well developed, and the attached portions of the meninges were apparently normal. The cortex was also broad and well developed, indeed, it appeared broader than that of a normal child's brain, but the tissues were found to be very tough on cutting. The folia of the two pieces of cerebellum were very slender and wasted, as much so indeed as I have ever seen in either primary or secondary atrophy of this organ. All the pieces I received had been fixed in alcohol, and consequently only the ordinary tissue stains—hæmatoxylin with Van Gieson's counterstain or eosin—and Nissl's method could be employed in their examination.

The most characteristic changes in the cerebral cortex were best visible in sections stained by Nissl's method; they were present in both the nerve-cells and the neuroglial matrix. The number of cells in the different layers of the cortex was apparently diminished, more so in the superficial than in the deeper layers, but this diminution was probably not greater than could be accounted for by the increased depth of the grey matter. All the cells from all portions examined were similarly affected, and not a single cell of normal structure could be found. They appeared swollen and inflated, their normal angles had disappeared, and, as a rule, the usual number of dendrites could not be detected by this method (fig. 1). Their nuclei were almost always displaced from the centre of the cell and frequently lay near the base of the apical dentrite. In well-differentiated Nissl preparations the greater part of the cell body was unstained and colourless, but some finely granular chromophilic material, evidently not identical with the normal tigroid, as it stained equally deeply with hæmatoxylin, lay around the nucleus. A few tigroid masses could be frequently seen in the apical dendrites of the larger cells ; the nucleus was usually irregular in shape and appeared relatively too small for the cells, probably owing to the swelling of the latter. Further, irregular globose or club-shaped appendages could be occasionally seen attached to the basal margins of the larger cells, but it was impossible to study their relation to these by the methods at my disposal.

All the cortical cells were affected by these changes, though they were more prominent in the larger than in the smaller. They were identical in every particular with the changes that characterized the cases of amaurotic family idiocy that I had previously examined [1], and those which had been described in detail by Risien Russell, Spiller, Mott, Schaffer, and others. The changes in the supporting tissue of the cortex were equally pronounced. There was an enormous increase of the neuroglial elements, both fibrillar and cellular, so that under a medium magnification the tissue appeared a dense feltwork of neuroglial fibrils. This sclerosis was, on the whole, greater in superficial than in the deeper layers of the cortex, and it was not equally marked in the different regions I examined. In addition to this fibrillar proliferation there was a remarkable increase, greater than I have ever seen in a diffuse pathological disease of the cerebral cortex, of the neuroglial cellular elements; the ordinary nuclei were increased in number and larger and more prominent than normal, while others were surrounded by homogeneous cytoplasm which spread out into tapering and branching processes so as to form typical "spider cells." Other neuroglial nuclei lay in spherical masses of protoplasm, and in places a considerable number of granular cells (Körnchenzellen) evidently of neuroglial origin lay scattered in the tissue. This neuroglial proliferation was almost as pronounced in the white as in the grey matter of the brain, and it is unquestionably to it that the abnormal size and weight of the brain is to be attributed.

Another feature was a considerable thickening of the walls of many of the larger cortical vessels, and an infiltration of their sheaths by small round cells of the lymphocyte type. No definite plasma cells could be recognized among them (fig. 2). This perivascular infiltration, which lay chiefly in the position of the Virchow-Robin space, was found, however, round only a relatively small number of vessels.

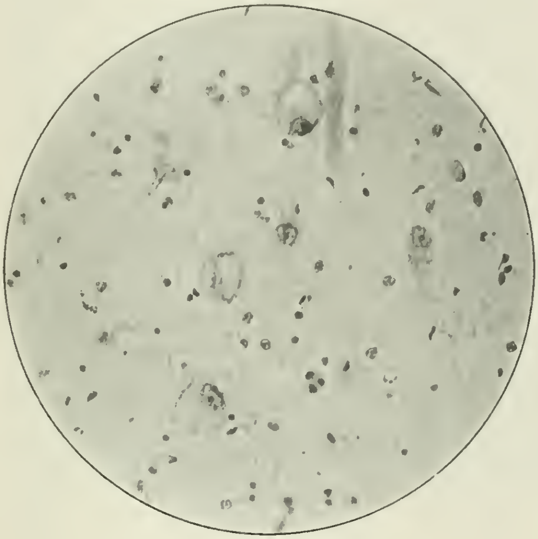


Fig. 1.

A photomicrograph of the cerebral cortex to illustrate the characteristic affection of the cells.

Microscopical examination of the cerebellum confirmed the naked-eye appearance of atrophy; the folia were small and stunted, and, though the molecular layer was normal in breadth, the cells of the granular layer were very sparse and reduced, in places, at least, to a small proportion of their normal number. In many folia, too, there were very few, or even no, Purkinje cells, and those that persisted presented the same pathological changes as those of the cerebral cortex (fig. 3). Their increase in size was very prominent, and similar globose appendages could be seen attached to the basal surfaces of many (fig. 4). The neuroglial sclerosis was less prominent than in the fore-brain, but there was undoubtedly a fibrillar increase in the molecular layer, while a band of proliferated neuroglial nuclei, such as may be seen in most conditions of cerebellar atrophy, lay in the intermediate zone of the cortex, or the zone of the Purkinje cells.

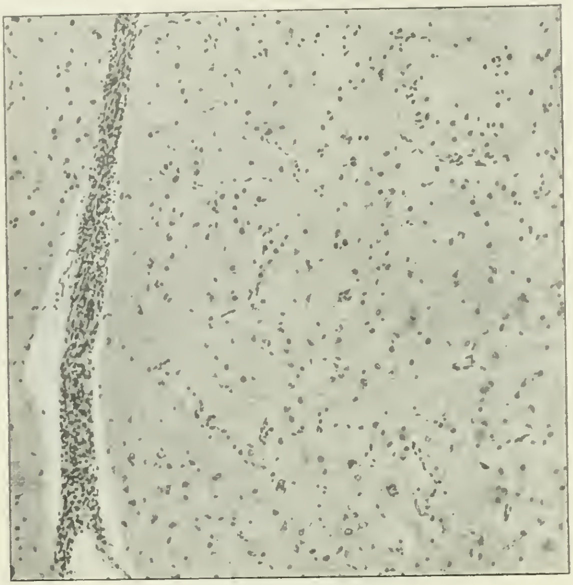


FIG. 2.

A lower magnification of the cerebral cortex, to show the general sclerosis and a vessel with perivascular small round-cell infiltration.

When I first examined my sections the affection of the nerve-cells was so similar to that which is characteristic of amaurotic family idiocy that I was inclined at once to regard the case as an instance of this disease, the more so as such cellular changes are found only in this condition, or in forms allied to it. There were, however, certain features that made me hesitate in definitely coming to this conclusion, especially in the absence of a full clinical history; as the abnormal size and weight of the brain, which was much impressed on me by the notes I received with the specimens and by the original request to examine

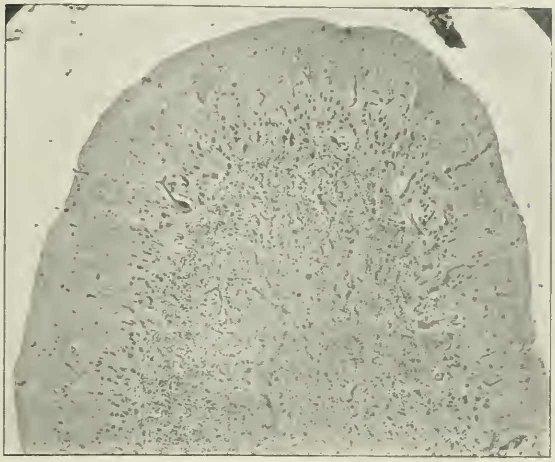


Fig. 3.

A folium of the cerebellum to show the general atrophy, especially of the granular layer.

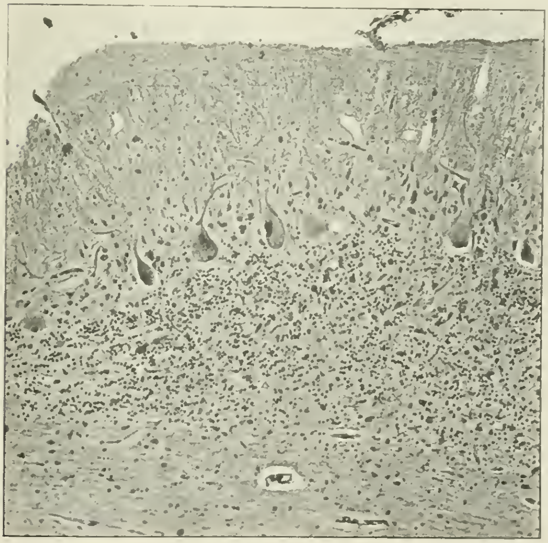


Fig. 4.

The cortex of the cerebellum to show the characteristic changes of the Purkinje cells.

a case of "megalocephaly"; the enormous amount of neuroglial proliferation, which was quite absent in my previously examined cases, and had never been very considerable in any of the cases already reported; the perivascular cellular infiltration in the cerebral cortex, which had, as far as I knew, not been found in other cases; and finally, the considerable atrophy of the cortex of the cerebellum.

The fact, however, that a later member of the family presented the characteristic clinical features and ocular changes, and that the same cellular affection, which is pathognomonic of amaurotic family idiocy, was found throughout the central and sympathetic nervous systems by Drs. Mott and Carlyll, makes it imperative to include this case in the disease. The neuroglial proliferation and the consequent increase in the size of the brain may be regarded as only an extreme instance of the sclerosis that has been previously observed; it is noteworthy that in the case of the sister, Dr. Mott found a considerable sclerosis. While the cerebellar atrophy may be looked upon as a coincidence. The perivascular cellular infiltration is suggestive of congenital syphilis, but, though this cannot be now determined with certainty, it is possible that it was in this instance associated with the congenital tendency to the disease.

REFERENCE.

[1] Poynton, Parsons and Holmes. *Brain*, 1906, xxix, p. 180.

Pathological Section.

February 21, 1911.

Dr. F. W. Mott, F.R.S., President of the Section, in the Chair.

Asphyxia under Chloroform.

By A. Goodman Levy.[1]

This research was undertaken to investigate the effect of asphyxia in animals under the influence of chloroform in view of a prevalent tendency to attribute a lethal importance to a condition of partial asphyxia induced accidentally in the course of chloroform anæsthesia and termed "intercurrent asphyxia." The apparent importance of this condition is emphasized in the Report of the Hyderabad Commission [15], and passing references to it are found in papers upon chloroform by Embley [3], and by Schäfer and Scharlieb [17]. Sherrington and Sowton [19] found that chloroform depressed the isolated mammalian heart more when it was perfused in a saline solution under asyphyxial conditions than when the solution was oxygenated; this work, as the authors state, lacks controls and completion. F. W. Hewitt [8] has been a frequent exponent, from a clinical standpoint, of this view, and more recently R. Gill [7] has published a treatise on the subject of a partial asphyxia which he infers is inherent to chloroform inhalation.[2] With the exception of Sherrington and Sowton's work no systematic investigation of the subject appears to have been undertaken, but general surmise has led to an extensive adoption of a plan of administering oxygen with chloroform for clinical purposes.

---

[1] From the Research Laboratories, University College Hospital Medical School.

[2] Since writing the above, Gardner and Buckmaster [6] have shown that the oxygen content of cats' blood may be reduced to 60 per cent. of the normal in full chloroform anæsthesia.

There would appear to be good reasons for taking this subject into serious consideration, for it is generally accepted that asphyxia has an exciting effect upon the cardio-inhibitory centre, and as it is also held that chloroform itself determines its activity (Embley [3], McWilliam [12], &c.), it might be anticipated that the combined action of chloroform and asphyxia would result in a strongly reinforced inhibitory action.

In the course of my experiments it became evident that it was necessary to adopt a standard of comparison, and this was afforded by parallel experiments conducted under the influence of ether. It is an obvious fact, judging by clinical experience, that intercurrent asphyxia under ether, however objectionable, is not of any serious importance, at any rate in so far as it affects the occurrence of sudden death. A degree of cyanosis (frequently, in fact, considerable) accompanies the administration of ether by the usual "closed" method, and has even been regarded (mistakenly so, as is now known) as a necessary adjunct, yet notwithstanding this it has never been suggested that such rare accidents as occur under ether may be attributable to intercurrent asphyxia. Further, ether does not of itself appear to excite the cardio-inhibitory centre, and therefore experimental asphyxia under ether provides a good standard by which to judge of experimental asphyxia under chloroform.

This investigation has been resolved into two separate lines of research, considered here in separate sections.

SECTION A.—HEART FAILURE FROM REFLEX CARDIAC INHIBITION.

The influence of chloroform upon the cardio-inhibitory reflex is similar to that which it exercises upon most other reflexes—i.e., progressive depth of anæsthesia progressively obscures the reflex. This fact was pointed out by François-Franck [4], and I have frequently confirmed his observation under the graduated administration of chloroform. The influence of asphyxia alone upon the cardio-inhibitory reflex has been studied also by François-Franck [5], who found that the cardio-inhibitory reflex induced by stimulating the infra-orbital nerve is abolished in an advanced stage of asphyxia. In this connexion also attention must be drawn to the observation of C. S. Sherrington [18] that asphyxia in the spinal dog first intensifies limb reflexes and finally suppresses them.

To study the combined effect of chloroform and asphyxia upon the cardio-inhibitory reflex I proceeded as follows: The animal, a cat, was tracheotomized under chloroform anæsthesia. The distal end of the tracheotomy tube was arranged to lie in a narrow-necked bottle into which a continuous current of air mixed with chloroform was pumped in excess of the animal's respiratory requirements. The percentage of vapour was carefully regulated in percentage terms. The blood-pressure was registered by a mercurial manometer connected with a carotid artery. The respiration was recorded from the thoracic respiratory movements, for the respirations are mainly abdominal in the cat under normal chloroform anæsthesia and are little affected by asphyxia, whereas the thoracic respirations, normally very slight, are intensely exaggerated by asphyxia and afford a very graphic index of the progress of the latter. The vagal reflex was produced by stimulation of the central end of a cut vagus nerve with a faradic current, the other vagus remaining intact. It frequently happens that the inhibitory element of this complex reflex is unobtainable or insufficiently marked, but it is more constantly obtained from the vagus than from the superior laryngeal nerve; the superior laryngeal reflex is furthermore frequently evanescent. When the inhibitory reflex is obtainable very similar results follow consecutive stimulations of the same intensity, provided the anæsthesia is maintained at an unaltered level during the intervals.

Asphyxia was produced by causing the animal to respire a confined atmosphere. A rubber bag was partially inflated with the same chloroform atmosphere that the animal had been inhaling for some time previously, and was attached to the trachea tube. The extent of inflation was made consistent with the desired rapidity of the asphyxial process. The vagal reflex could thus be tested in any stage of asphyxia, rapidly or slowly produced, and the results obtained compared with those taken immediately prior to the attachment of the bag.

In order that the state of anæsthesia should be maintained at practically the same level throughout the experiment, a somewhat prolonged administration of vapour at a fixed percentage was given beforehand, so as to reduce the rate of absorption of chloroform before attaching the asphyxiating bag. The quantity of chloroform vapour contained in the bag was too small, even if largely absorbed, to influence the degree of narcosis to any considerable extent. There being, further, no tangible evidence of the destruction of chloroform in the body, there would not appear to be any probability of the production of a lessened anæsthesia during the process of asphyxia.

According to Morat and Doyon [13], Embley, and others, it is in the induction period of chloroform administration that the vagus centre manifests its greatest activity, both spontaneous and reflex. Unfortunately this stage of anæsthesia does not lend itself readily to experiments such as these, and therefore the condition of fully induced anæsthesia alone has been considered in this research.

Two series of experiments of this nature were performed, twelve under chloroform and eight under ether. The ether vapour was administered in a similar fashion to the chloroform and was controlled to a constant concentration, but the actual percentage value of it was not ascertained.

TABLE I.—EXPERIMENTS UNDER CHLOROFORM ASPHYXIA.

No.	1910	Percentage of chloroform	Stimulation		Reflex	Latent period of reflex
			Strength	Duration		
Experiments in which the Reflex was Diminished only.						
1	July 19	1·5 per cent.	—	9 sec.	Progressive diminution and final suppression	Increased
2	,, 21	1·0 ,,	15 cm.	8 ,,	Marked diminution in final stages	Diminished
3	,, 22	0·5 ,,	Strong	10 ,,	Progressive diminution in latter half of tracing	Increased
4	,, 25	1·0 ,,	Strong	6 ,,	Early progressive diminution	,,
5	Aug. 19	0·8 ,,	20 cm.	5 ,,	Progressive diminution and final suppression	Unchanged
6	Sept. 9	1·5 ,,	6·5 cm.	7 ,,	Early and persistent diminution	Increased
7	,, 12	1·5 ,,	14 cm.	5 ,,	Early diminution and almost total suppression	,,
Experiments in which the Reflex was Increased.						
8	Sept. 1	1·0 per cent.	6 cm.	—	Marked progressive increase—diminution in final stage	Increased
9	,, 5	(*a*) 0·5 ,,	6 ,,	4 sec.	Marked progressive increase (asphyxia not carried to final stage)	Diminished
		(*b*) 2·0 ,,	6 ,,	2 ,,	Progressive increase in the first part—diminution in final stage	,,
10	,, 15	1·5 ,,	Light	14 ,,	Marked increase ...	Unchanged
11	July 28	1·0 ,,		6 ,,	Slightly diminished at first, marked increase in middle stages. Asphyxia stopped when oxygen in bag reduced to 9·2 per cent.	Increased
12	Sept. 8	1·5 ,,	19 cm.	5 ,,	Slightly increased at first, diminution later	,,

An analysis of the above table shows that asphyxia under chloroform has a general tendency to cause diminution of the reflex under consideration. In the first seven of the experiments asphyxia caused diminution alone, most marked in the advanced stages, but sometimes quite evident in the earlier stages also (figs. 1 and 2). In every case (including the following five) in which the asphyxia was carried to completion some degree of diminution could be detected in the concluding stage—that is to say, at about the time of primary failure of respiration. In the remaining five cases there was evidence of exaggeration of the reflex under asphyxia. Of these three showed increase only, except in the terminal phase, and the increase was progressive in two of them. In the last two cats both increased and decreased effects were observed antecedent to the terminal phase. Figs. 3 and 4 show well-marked instances of exaggeration of the reflex. The latent

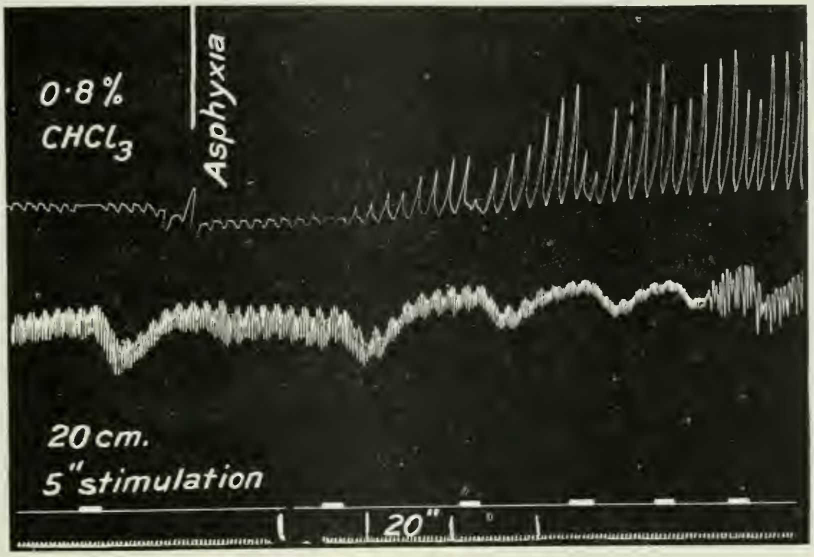


Fig. 1.

Asphyxia under chloroform, somewhat rapidly induced. Progressive diminution of the vagal reflex. The signal line also represents the blood-pressure zero. Time marked in seconds.

[*Note.*—All tracings to read from left to right. The upper tracing in each case represents the respirations. Where no abscissa line has been introduced the stimulation-signal line (in fig. 8 the time-line) serves to indicate the pressure zero.]

period of the reflex is generally lengthened in asphyxia under chloroform independently of changes in the intensity of the reflex.[1]

As a control to the foregoing results I may briefly mention a series of experiments in which asphyxia was induced by causing the animal to inhale an irrespirable gas, either pure or diluted. The gases employed were hydrogen, nitrogen, and carbon dioxide, and chloroform vapour

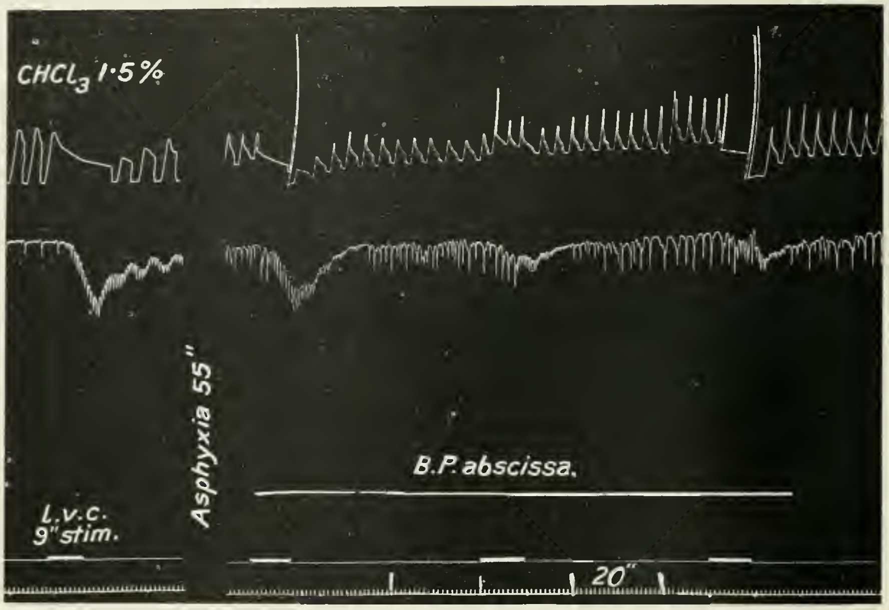


Fig. 2.

Asphyxia under chloroform. Progressive diminution of the vagal reflex. In the interval represented by the gap in the tracing, the asphyxiating bag was connected to the trachea tube, and the following vagal stimulation was made 55 seconds later. Time marked in seconds.

was given in the same proportion per atmosphere as was present in the previously inspired air. The administration was never conducted for a long period, and therefore no question of progressive anæsthesia could

---

[1] It is noteworthy that the simultaneous respiratory reflex undergoes a change in asphyxia under chloroform. Normally the arrest of respiration takes place in *inspiration*, but it gradually changes to well-marked *expiratory* arrest as asphyxia advances (fig. 3). This effect is not generally so well observed under ether anæsthesia.

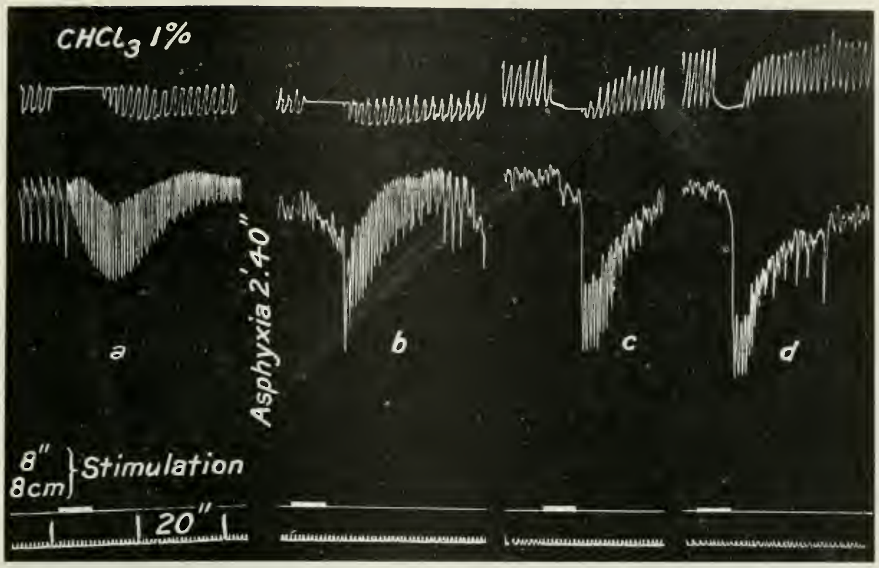


FIG. 3.
Asphyxia under chloroform, slowly induced. Progressive increase of the vagal reflex. The signal line also indicates the blood-pressure zero. *a*, normal reflex; *b*, 2 min. 42 sec. after commencing asphyxia; *c*, 5 min. 30 sec. after *b*. *d*, 2 min. after *c*. Time in seconds.

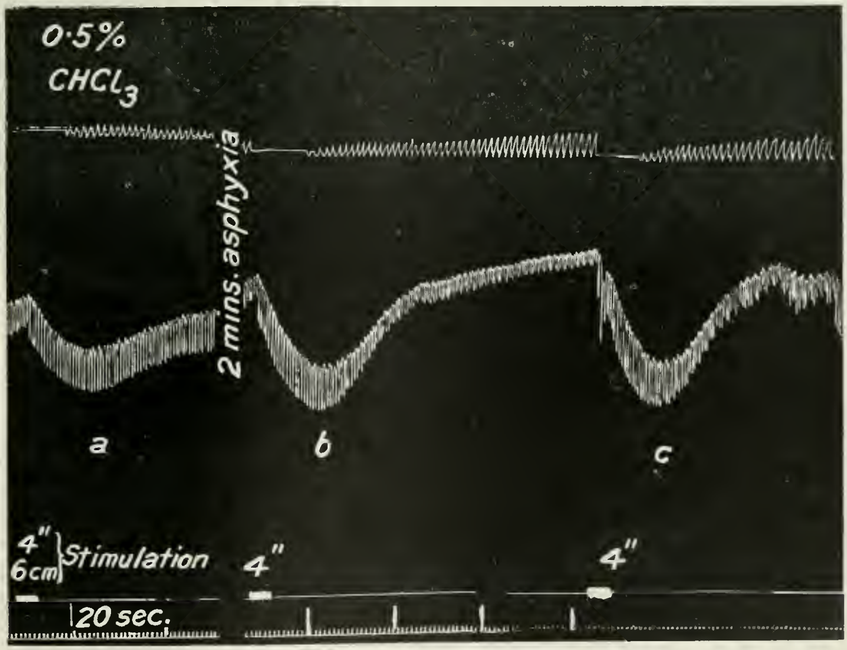


FIG. 4.
Asphyxia under chloroform. Progressive increase of vagal reflex. *a*, normal reflex *b*, 2 min. after bag connected; *c*, 1 min. 10 sec. after *b*. Time marked in seconds.

arise; further, artificial respiration being generally employed, variations in absorption, such as might be induced by the exaggerated respirations of asphyxia or by the reflex cessation of respiration during stimulation of the nerve were eradicated. These particular experiments were not uniformly satisfactory, but they served to substantiate, under the somewhat different conditions of asphyxia and anæsthetic administration, the previously described observations that the vagus reflex is variably affected by asphyxial factors, but more commonly in the direction of decrease. In fig. 5 is shown a tracing of an exceptionally intense increase of the reflex under an atmosphere of CO_2 25 per cent., $CHCl_3$ 0·5 per cent., air 74·5 per cent. This particular experiment was repeated three times on the same animal, thus excluding the possible influence of fortuitous conditions.

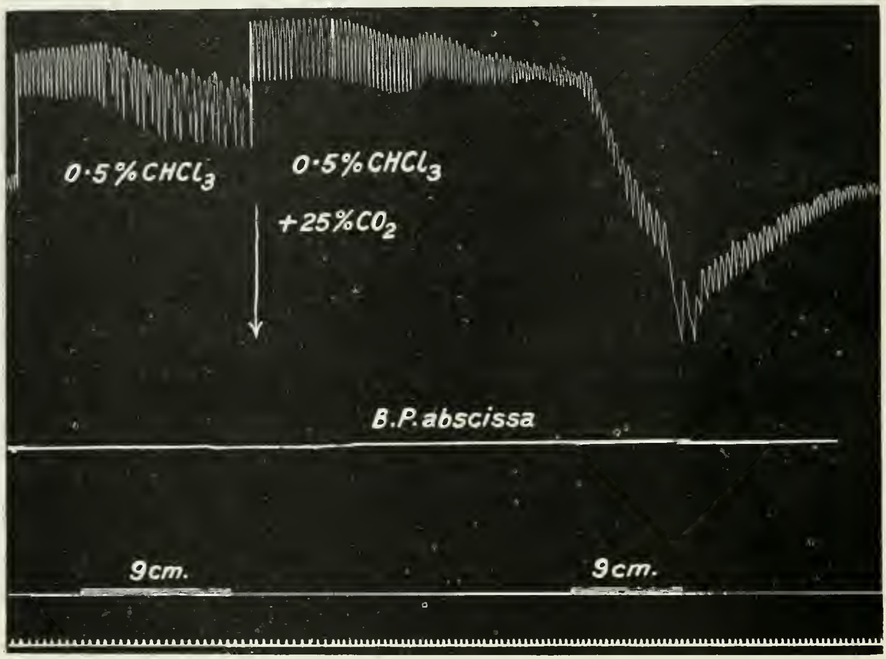


Fig. 5.

Increase of vagal reflex under 25 per cent. CO_2. Artificial respiration. Time marked in seconds.

There do not appear to be any diverse factors in the foregoing experiments to account for the irregular results, which are doubtless, therefore, due to interacting physiological processes. The respective parts played by the nerve-centres, the cardiac and general circulatory mechanism in modifying the inhibitory reflex during asphyxia, cannot be unravelled in the absence of further knowledge. It is, for instance, highly essential to ascertain the influence of asphyxia upon direct cardiac inhibitions caused by stimulation of the peripheral end of the cut vagus, but the difficulty of obtaining this reaction in the cat[1] has caused the postponement of further investigation.

TABLE II.—EXPERIMENTS UNDER ETHER ASPHYXIA.

No.	1910	STIMULATION		Reflex	Latent period of reflex
		Strength	Duration		
			Experiments in which the Reflex was Diminished.		
1	Sept. 19	9 cm.	7 sec.	Early total suppression	Unaltered
2	,, 21	6 ,,	4 ,,	Progressive diminution	Diminished
3	,, 30	5 ,,	4 ,,	Marked progressive diminution	,,
4	Oct. 4	10 ,,	4 ,,	Progressive diminution	Unaltered
			Experiments in which the Reflex was Increased.		
5	Sept. 28	6 cm.	5 sec.	Slight progressive increase	Unchanged
6	,, 28	6 ,,	5 ,,	,, ,,	,,
7	,, 29	5 ,,	4 ,,	Marked progressive increase	Diminished
8	Oct. 5	5 ,,	4 ,,	Slight progressive increase	,,

The ether asphyxia series is analysed in Table II. Here the instances of increase and decrease of the reflex are seen to occur in equal frequency, and in fig. 6 is shown a well-marked instance of the former variety. The latent period is more generally reduced in ether asphyxia, and there is not so marked a tendency towards total suppression in the final stage, otherwise the phenomena are very similar to those observed under chloroform asphyxia.

---

[1] *See* Hough, *Journ. Physiol.*, 1895, xviii, p. 163, in confirmation of this.

I may add that I have never observed any indication that a fatal result may be caused by a cardio-inhibitory reflex of any intensity, under either anæsthetic.

The conclusions arrived at from a consideration of this section are as follows:—

(1) Under chloroform the cardio-inhibitory reflex is obscured or suppressed by complete asphyxia.

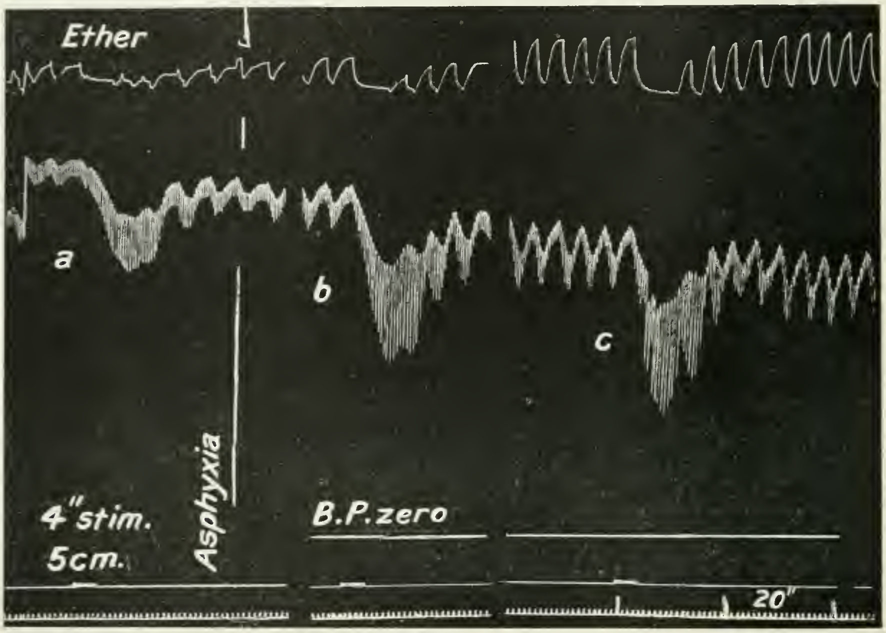


Fig. 6.

Asphyxia under ether. Progressive increase of the vagal reflex. The first gap represents an interval of 40 sec., the second gap an interval of 60 sec. Time marked in seconds.

(2) In a condition of partial asphyxia under chloroform the cardio-inhibitory reflex is variably affected, for it may be either diminished or increased in an apparently fortuitous manner, the intensified form of reaction being, on occasion, of a marked and significant character.

(3) Under ether, partial asphyxia also affects the cardio-inhibitory reflex in a similar diverse fashion, and the intensified reaction would appear to be no less frequent than under chloroform.

SECTION B.—SPONTANEOUS CARDIAC AND RESPIRATORY FAILURE IN ASPHYXIA UNDER CHLOROFORM.

The form of asphyxia employed in the experiments considered in this section was, as before, non-obstructive, and hence all blood-pressure effects such as might be due to variations in intrathoracic pressure were avoided. The period of fully induced anæsthesia was alone studied, the stage of induction not being taken into consideration.

(I) Cardiac Failure.

Asphyxia under both chloroform and ether alike seldom causes more than a slight rise of blood-pressure, and frequently either no rise at all occurs or else an early decline may be found. The asphyxia manifests itself in a fall of pressure, which is generally late in onset and rapid in course, and appears to be conditioned simply by want of oxygen, as in asphyxia without anæsthetics. The slowing of the heart usually seen in the course of this fall is well ascertained to be due to concurrent asphyxial impulses from the cardio-inhibitory centre, and to be an accompaniment, and not a determining cause, of fall of pressure. In experiments under general anæsthesia the characteristic slow and regular vagal beats only occur when the asphyxia is practically complete, with a rapidly falling blood-pressure and failing respiration. Under ether this vagal beat is generally better marked than under chloroform.

Slowing of the heart-beat in asphyxia due to heart-block has recently been described by Lewis [10] and Mathison [11]. This appears from Mathison's paper to be late and irregular in onset in the intact animal, and, so far as I can judge from the data given, enters more into the consideration of those events following the stage in which asphyxia has become sufficiently acute to cause cessation of regular respiration.

Irregularities of the heart-beat, generally ascribed to vagal action on account of their non-occurrence when the vagi are cut (Hill and Flack [9], Mathison), are sometimes found in the presence of a partial deprivation of oxygen under chloroform, as in the stages of a gradually induced asphyxia prior to the respiratory and vascular climax (*see* fig. 7). Such irregularities are not infrequently observed, apart from asphyxia, in uncomplicated chloroform narcosis, but their onset does appear to be determined in some instances by partial asphyxia, and hence they must be considered as asphyxial phenomena. The first part of this tracing shows irregularities which are in all probability due to vagal influence, but the second part, characterized by very rapid and small heart-beats, resembles the form which results when vagal action is cut out. In the course of this latter type of tracing, on rare occasions a sudden stoppage of the heart's action has been observed when the oxygen supply has become very limited, giving rise to a not inconsiderable fall of blood-pressure. This fall is not permanent, but is subject to rapid recovery, complete or partial. The most pronounced instance of this kind which I have come across occurred in a cat which was being slowly asphyxiated under 0·8 per cent. chloroform; at the end of six minutes the pressure rapidly fell 58 mm. (from 146 mm. to 88 mm.), but soon recovered to

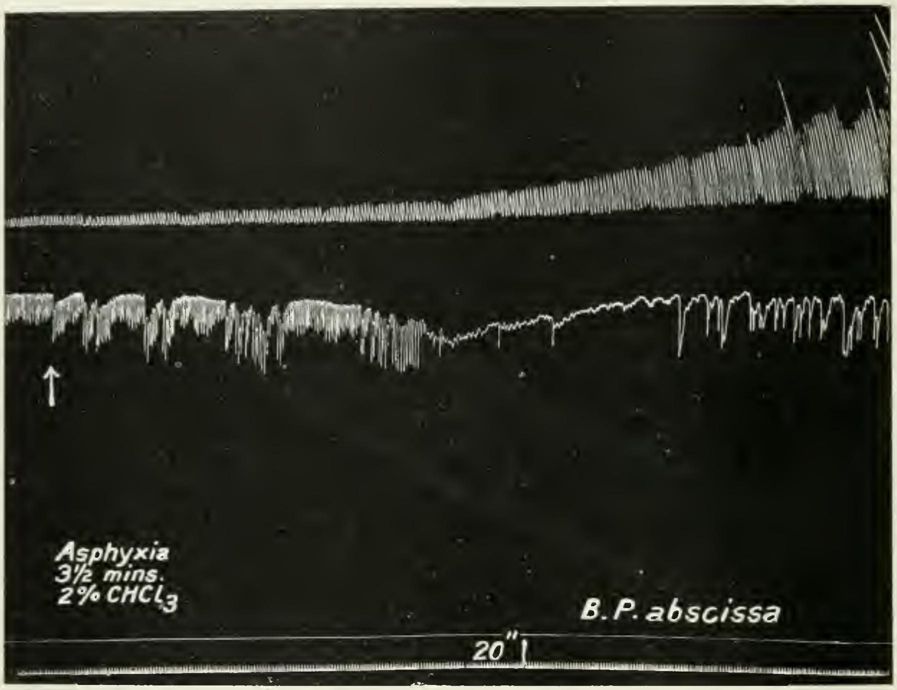


Fig. 7.

Forms of irregularity of the heart-beat in slow asphyxia under 2 per cent. chloroform. The asphyxiating bag was connected to the trachea tube 3 min. 30 sec. before the arrow mark. Respirations ceased very shortly after the terminal portions of the tracing shown. Time marked in seconds.

126 mm., the breathing ceasing in the final asphyxial stage two minutes later. It is impossible to define the cause of this form of irregularity from the blood-pressure tracings alone, but it is probable that it is not so much a sign of heart failure as an indication of vascular adjustment.

It does not appear, from the foregoing observations, that any serious permanent lowering of the mean blood-pressure follows from the irregularities of heart-beat which may be found in partial asphyxia, and these are probably therefore of little significance from a clinical point of view.

I have not met with any of the irregularities just described in the case of asphyxia under ether, the ether pressure tracing presenting a singularly even form up to the final stage. To anticipate later matter, it is in this freedom from irregularities alone that ether asphyxia compares with chloroform asphyxia to the disadvantage of the latter.

(II) The Arrest of Respiration.

The moment at which regular respiration ceased was adopted as an indication of the point when the oxygen in the blood became insufficient to support life and the asphyxial process might be considered complete. This primary arrest of regular respiration is a well-marked phase under anæsthetics (fig. 8); it follows very shortly after the asphyxial spasm, when this occurs, and is coterminous or nearly so with the asphyxial fall of pressure, although the heart does not actually cease beating until considerably later. The respiratory arrest is followed by a prolonged pause, which is succeeded in turn by a series of convulsive asphyxial gasps at long intervals. Two methods—(*a*) and (*b*)—were adopted for the investigation of this subject:—

(*a*) *Observations upon the Minimum Partial Pressure of Oxygen necessary to maintain Respiration.*—Cats were asphyxiated in the usual fashion by causing them to inhale an atmosphere containing either chloroform or ether vapour confined in a bag. The bag was withdrawn at the moment complete suppression of regular respiration supervened, and its contents analysed. The percentage of residual oxygen was taken as a measure of the animal's resistance to asphyxia under the respective anæsthetics. The time taken to induce complete asphyxia varied naturally with the extent to which the bag was inflated, which was not exactly ascertained, and therefore not taken into account in these experiments. Table III includes four experiments of this nature, the animals being tracheotomized but not subjected to any further operative procedure.

TABLE III.—PROLONGED SIMPLE ASPHYXIA.

1910	Anæsthetic	Arrest of respiration in[1]	Analysis	
			O_2	CO_2
Oct. 7	Chloroform, 1·8 per cent.	13 min. 40 sec.	4·5 per cent.	11·3 per cent.
,, 10	,, 1·8 ,,	9 ,, 20 ,,	4·2 ,,	12·0 ,,
,, 7	Ether	10 ,, 10 ,,	4·6 ,,	8·3 ,,
,, 11	,,	6 ,, 20 ,,	5·5 ,,	9·4 ,,

[1] The time taken to arrest respiration was dependent on the extent of inflation of the bag, which was not controlled.

The two results obtained under chloroform, and the first of the two under ether, agree with the observation of Paul Bert [1] that the lowest respirable percentage of oxygen for the cat, when not under the influence of an anæsthetic, is an average of 4·4 per cent. In the second ether case the respiration failed at a somewhat higher percentage (5·5 per cent.). It is, therefore, apparent that the respiratory centre is neither more nor less resistant to uncomplicated asphyxia under chloroform than in its absence. Under ether, asphyxia would appear from the above results to be somewhat more lethal in its effects.

It is interesting to note that a greater excretion of CO_2 occurs under chloroform asphyxia than under ether asphyxia. This is a constant phenomenon, and there is a good deal of collateral evidence regarding the increased production and output of CO_2 under chloroform (Buckmaster and Gardner [6], Collingwood [2], Glasgow Committee [14]). This is not, however, a matter for present discussion, inasmuch as the highest of these percentages is non-lethal, and consequently the differences noted can have no significance in relation to the subject under consideration.

In addition to the above, other analyses were made of the contents of the bag in the case of some of the experiments described under Section A, and the results must be considered separately, for the conditions represent more closely those of a subject undergoing manipulations in the course of a severe operation. As previously described, the animal was tracheotomized, a cannula inserted in a carotid artery, one vagus divided and its central end stimulated at intervals. The analysis was made, as before, as soon as respiration ceased.

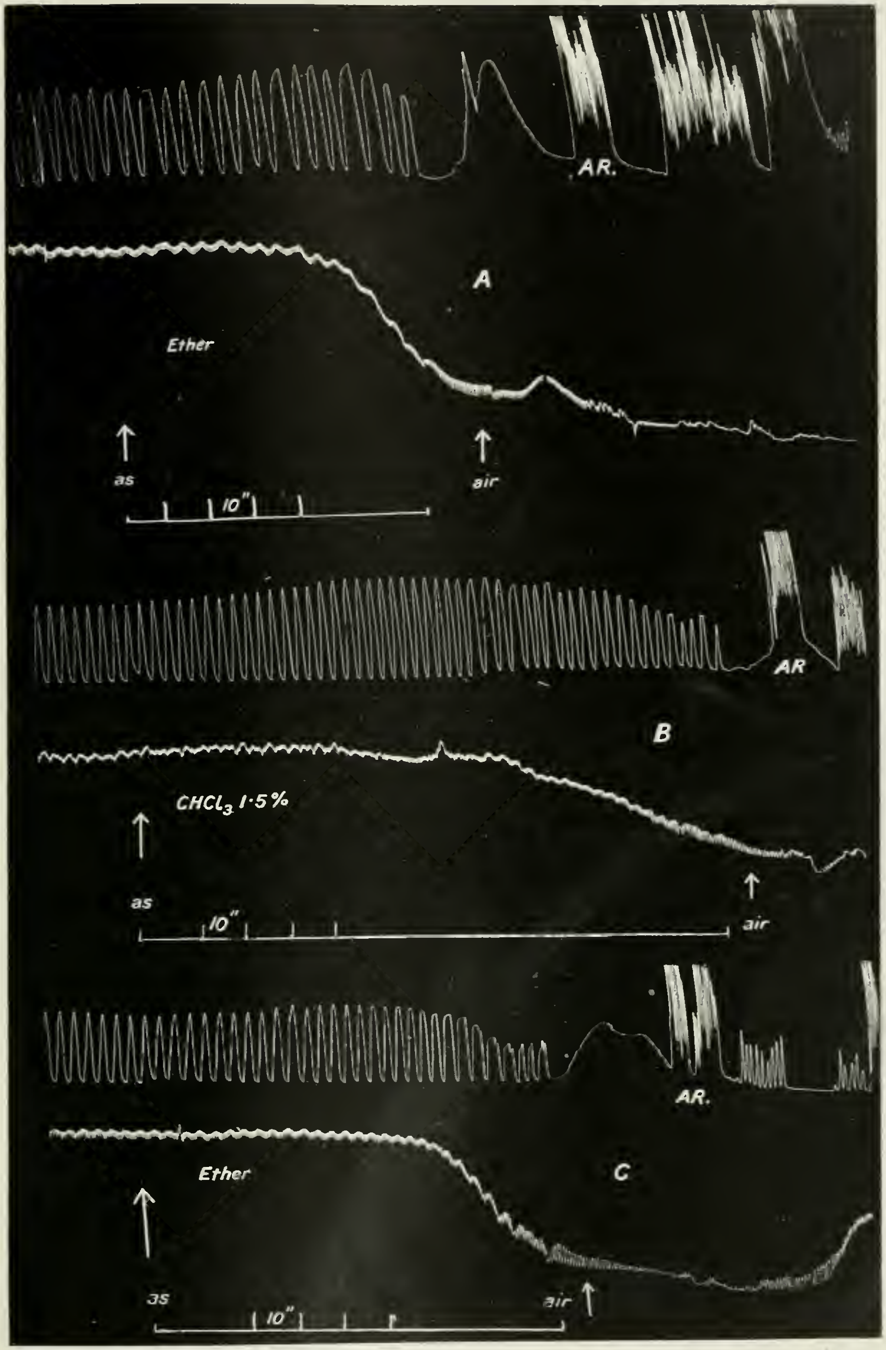


FIG. 8.

Asphyxia under chloroform compared with asphyxia under ether in the same animal. A and C are tracings of asphyxia under ether. B is a tracing of asphyxia under chloroform. The horizontal lines, bearing time markings in periods of 10 sec., represent the blood-pressure zero and also, by their length, the duration of the asphyxia up to the moment of the cessation of regular respiration. A R = artificial respiration. Respirations recorded from abdomen.

TABLE IV.—PROLONGED COMPLICATED ASPHYXIA.

1910	Anæsthetic	Arrest of respiration in[1]	Analysis	
			O_2	CO_2
Oct. 12	Chloroform, 2·0 per cent.	8 min. 40 sec.	4·5 per cent.	10·0 per cent.
Aug. 19	,, 0·8 ,,	6 ,, 40 ,,	4·0 ,,	10·4 ,,
Sept. 21	Ether	7 ,, 0 ,,	5·3 ,,	8·5 ,,
,, 28	,,	4 ,, 30 ,,	6·6 ,,	7·6 ,,
,, 29	,,	6 ,, 50 ,,	7·0 ,,	8·8 ,,
,, 30	,,	3 ,, 10 ,,	6·6 ,,	7·0 ,,
Oct. 5	,,	3 ,, 30 ,,	6·5 ,,	6·4 ,,

[1] See footnote to Table III.

The two chloroform records in the above table show an oxygen percentage remaining in the bag which is very much the same as was found under uncomplicated asphyxial conditions (Table III). The average of the oxygen percentages in the experiments under ether is notably raised, being 6·4 per cent., as against 5·0 per cent. shown in Table III. The amount of CO_2 found under both anæsthetics averages somewhat less than in simple asphyxia. The average CO_2 under ether is again found to be less than that under chloroform, but it is possible that, in this series, the notably larger remnant of O_2 in the ether cases may be partially responsible for the difference.

The foregoing figures demonstrate that a smaller quantity of oxygen will generally maintain respiration under chloroform in the presence of severe operative measures than will suffice under ether with similar conditions, and that therefore asphyxia under ether may prove more detrimental than chloroform asphyxia.

(*b*) *Observations on the Time Incidence of Cessation of Respiration.*—The factor of time incidence was investigated by attaching to the trachea a thin rubber bag of about 20-c.c. capacity—i.e., just large enough to allow free respiratory movements to take place. The animal being put under one of the two anæsthetics under consideration, the bag, fully inflated with air only, was attached and left on until the respiration failed; it was then restored by artificial respiration and re-subjected to asphyxia in the same way under the other anæsthetic. There is no positive standard of depth of anæsthesia, but this was adjusted so as to be equal, as far as could be judged, under the two anæsthetics, and corresponding to that of ordinary surgical anæsthesia.

TABLE V.

Number of experiment	Anæsthetic	Initial blood-pressure	Breathing ceases in	Remarks
No. 1	(1) Ether	128 mm.	2 min. 12 sec.	Restored by artificial respiration
	(2) Chloroform, 0·5 per cent.	76 ,,	2 ,, 12 ,,	,, ,,
No. 2	(1) ,, 1·0 ,,	132 ,,	2 ,, 5 ,,	,, ,,
	(2) Ether	126 ,,	1 ,, 38 ,,	Failed to restore animal
No. 3 (fig. 8)	(1) ,, ...	126 ,,	1 ,, 8 ,,	Restored by artificial respiration
	(2) Chloroform, 1·5 per cent.	86 ,,	2 ,, 14 ,,	,, ,,
	(3) Ether	90 ,,	1 ,, 32 ,,	,, ,,

The numbers in parentheses indicate the order in which the anæsthetics were administered.

It is evident, from the three experiments in the table, that the respiratory centre may be affected more rapidly by asphyxia under ether than under chloroform, for in two of them the ether asphyxia is a more acute process than is the chloroform asphyxia, and it is further noticeable that, whereas all the animals were restored by artificial respiration after chloroform, one of them could not be restored by these means after ether. Asphyxia is, therefore, certainly not in this respect a more serious complication under chloroform than it is under ether, when it has the clinical reputation of being comparatively harmless.

A final word may be added in relation to the incidence of fatal suffocation arising from an accidental complete occlusion of the air-passages during anæsthesia. It is stated by Richet [16] that chloroformed animals support asphyxia as well as, if not better than, animals which have not been anæsthetized, and that it is always possible to restore them for some minutes after the cessation of respiration. This observation does not accord with my own experience, for I have met with cases, both under chloroform and under ether (but more frequently under the latter), in which I failed to restore the heart-beat. This subject is, I think, worthy of investigation, for some of the asphyxial symptoms are not strongly evinced under chloroform, and so there must exist a suspicion that the nature of a suffocative process may not be properly recognized. The following considerations have to be borne in mind in connexion with this matter :—

(1) In complete obstructive asphyxia there is a greater privation of air than in the case of any of the non-obstructive experiments described

in the foregoing part of this paper, and hence the cessation of respiration comes about within a shorter period.

(2) Asphyxial spasm is practically suppressed at any higher concentration of chloroform than 1 per cent., and indeed this important sign may be altogether lacking. I have never found it entirely wanting under ether.

(3) The respiratory movements may be restrained by the obstruction, when full anæsthesia is maintained, from showing their asphyxial exaggeration.

(4) The mechanical influence of the asphyxial respirations may induce an earlier fall of blood-pressure than in the non-obstructive form of asphyxia.

These remarks are, of course, merely suggestive, and do not lead to any positive conclusion in respect of the possibility of death from suffocation under chloroform in ordinary clinical conditions.

General Conclusions.

So far as the scope of these experiments extends, they do not afford any evidence to justify the assumption that asphyxia, whether partial or complete, is a more serious complication in any important respect under chloroform than under ether narcosis. In both cases asphyxia is evidently undesirable, and of the two there is evidence that asphyxia under ether is the more active in affecting the respiratory centre.

REFERENCES.

[1] Bert, Paul. "La pression barométrique," Par., 1878, p. 577.
[2] Collingwood. *Journ. Physiol.*, 1907-8, xxxvi (*Proc. Physiol. Soc.*, October, 1907, p. xxi).
[3] Embley. *Brit. Med. Journ.*, 1902, i, pp. 817, 885, 951.
[4] François-Franck. *Compt. rend. Soc. de Biol.*, 1883, 7 sér., iv, p. 255.
[5] *Idem.* "Travaux du labor. de M. Marey," Par., 1878, ii, p. 273.
[6] Gardner and Buckmaster. *Journ. Physiol.*, 1910, xli, p. 60.
[7] Gill. "The $CHCl_3$ Problem," 1906.
[8] Hewitt. "Anæsthetics and their Administration," 1907; also *Lancet*, 1904, ii, p. 1497.
[9] Hill and Flack. *Journ. Physiol.*, 1908, xxxvii, p. 77.
[10] Lewis and Mathison. *Heart*, 1910, ii, p. 47.
[11] Mathison. *Heart*, 1910, ii, p. 54.
[12] McWilliam. *Brit. Med. Journ.*, 1890, ii, pp. 831, 890, 948; also *Journ. Physiol.*, 1899. 1900, xxv, p. 233.
[13] Morat and Doyon. "Traité de physiologie," Par., 1899, i, p. 108.
[14] "Report of the Glasgow Committee," *Brit. Med. Journ.*, 1880, ii, p. 957.
[15] "Report of the Hyderabad Commission," 1891.
[16] Richet, C. "Anesthésie," "Dictionnaire de physiologie," Par., 1895, p. 506.
[17] Schäfer and Scharlieb. *Trans. Roy. Soc. Edin.*, 1901, xli, p. 311.
[18] Sherrington. *Journ. Physiol.*, 1909, xxxviii, p. 378.
[19] Sherrington and Sowton. "Report of the Special Chloroform Committee of the British Medical Association," *Brit. Med. Journ.*, 1906, ii, p. 85.

DISCUSSION.

Mr. RICHARD GILL said: Most of us will assent to much in the excellent paper which Dr. Levy has favoured us with to-night. But there are some points in it which are open to criticism, and I will draw attention to two of them. The first refers to those instances of respiratory arrest which were not recovered by artificial respiration. Dr. Levy ascribes the fatal result to failure of the respiratory centre. Now, in all examples of asphyxia, pulmonic congestion is present in an intense degree. That this abnormal condition of the lungs enters as one of the factors in the causation of arrest of the action of the respiratory machine is shown as follows: by assisting the function of aëration artificially (I presume that the heart has not ceased to functionate, and that it is capable of being restored to its normal function), we observe that as the respiratory muscles are becoming increasingly supplied with oxygen, these manifest signs of activity, but irregularly, and that when they are supplied with an efficient amount of oxygen they resume their normal function. Thus, failure of the respiration in asphyxia is invariably associated with exhaustion of the respiratory muscles, emanating from two sources: a rapidly diminishing supply of oxygen, and the increased and increasing amount of work which they have to do in order to overcome the resistance of congested lungs. But, assuming respiratory failure in asphyxia to be due, not to a temporary exhaustion of the respiratory muscles in examples of recovery, but to some abnormal condition of the respiratory centre, how is this prominent characteristic of the attitude of the respiratory centre explained, viz., the rapid recovery of its function after having been subjected, hypothetically, to a preformed degree of depression?

The second feature I desire to allude to is the absence of distinction of the different degrees of chloroform-action in the experiments which Dr. Levy conducted. We have the lowest degree of chloroform-action, associated with unconsciousness, and I think it would be well if we confined the term "anæsthesia" to that degree. We have also the higher degrees of chloroform-action, which should, for purposes of clearness and accuracy, be designated by the term "chloroform-narcosis." The exact degree of narcosis is measured by the size of the pupil. Now, the sets of phenomena are different in anæsthesia and narcosis. The reason of this is obvious, the encroachment upon the air in the chloroform air atmosphere is greater in narcosis than in anæsthesia. Now what new results or changes in the normal results are produced by the negative action of chloroform when present in excessive intensity? I am inclined to think that Dr. Levy has not laid enough stress on the negative action of chloroform, as providing the underlying conditions, from which he draws his conclusions. Supposing, however, that chloroform is responsible in a positive way for those conditions, during the terminal stage of asphyxia induced by it, how does Dr. Levy explain the fact that we do not witness them in an incipient stage during chloroform anæsthesia? At the beginning of the latter we find the pulse at its natural frequency, and a certain increase in respiratory frequency. It is true that in the course of chloroform anæsthesia respiratory frequency tends to become accelerated, and the pulse shows a slight increase in the number of beats. No one, however, can call these changes manifestations of inhibition or of depression.

Now, I should like to call attention to a very important point which has only an indirect bearing on Dr. Levy's investigation. Everyone is aware of the remarkable rapidity with which the complexion recovers its natural colour after the administration of nitrous oxide gas. Let us suppose a patient who presents in a state of chloroform narcosis an equal degree of duskiness to that which is induced by nitrous oxide gas. What period will elapse before he recovers his natural colour after the withdrawal of chloroform? About fifty seconds; whereas in the case of nitrous oxide gas it is a matter of four or five seconds. What, then, is the explanation of that difference? It will be remarked at once that in chloroform narcosis more oxygen is required, and observation informs us that the return to the normal is gradual and progressive. The conclusion, therefore, is that something impedes the oxygenation of the blood. But there is no other agent but chloroform present in the blood. Thus, after the oxidation of chloroform by the pure or nascent oxygen in the blood the latter is restored to its normal condition, and without manifesting the least sign of injury to any of the components of it. A consideration of what I have just expounded will, I think, lead to the explanation of Dr. Levy's failure to recover by artificial means some of the cats he asphyxiated. An undue excess of chloroform in the blood was present in these instances, and the heart failed from exhaustion, before enough oxygen could be got into the blood.

Uterine Tumours in Rabbits.

By A. E. Boycott.[1]

In the spring of 1909 four cases of epithelial tumour of the uterus were found in rabbits which were killed for various experimental purposes. Though a careful watch has since been kept, no further examples have been met with, and, though our knowledge of their nature and history is still far from complete, it seems worth while to place the present series on record. As far as I can ascertain, similar tumours have not been previously described. Lack[2] records the discovery of a carcinoma widely disseminated all over the body of a rabbit in which, about a year previously, he had cut open the ovary and scattered the ovarian cells about the peritoneal cavity. One of his figures (Pl. ix, fig. 2) shows a very similar structure to that found in my own cases, though in these latter the growth was strictly local, and showed no evidence of extension beyond the uterus. In the absence of repetition and confirmation, the view that Lack's tumours arose from the misplaced ovarian cells must be regarded with suspicion. Shattock[3] considers that the growth was a spontaneous carcinoma of uterus, and records a case in which multiple nodular growths occurred in both halves of the uterus with a few nodules in the peritoneal cavity.

Case I (fig. 1, A, fig. 2, C, and figs. 6 and 7).—One-half of the uterus is occupied by a large solid mass about 8 cm. by 2 cm. by 3 cm., and weighing 26 grm. On transverse section (fig. 2, C) the cavity of the uterus is reduced to a lenticular slit lined with a single layer of epithelium; over the surface of the tumour this is thinned out into a simple layer of low cubical cells, but on the fundus side the cells are columnar, and grow in small, irregular papillæ. The tumour itself (fig. 6) is composed of a loose connective tissue stroma in which irregular epithelial growths are embedded, forming for the most part solid masses of cells, but with a good number of alveoli of varying size lined with cubical epithelium. On the mesometric side the epithelial masses deeply infiltrate the muscular coat (fig. 7). The other half of the uterus is

---

[1] From the Pathological Department, Guy's Hospital.

[2] *Journ. of Path. and Bact.*, 1900, vi, p. 154.

[3] *Trans. Path. Soc. Lond.*, 1900, li, p. 56.

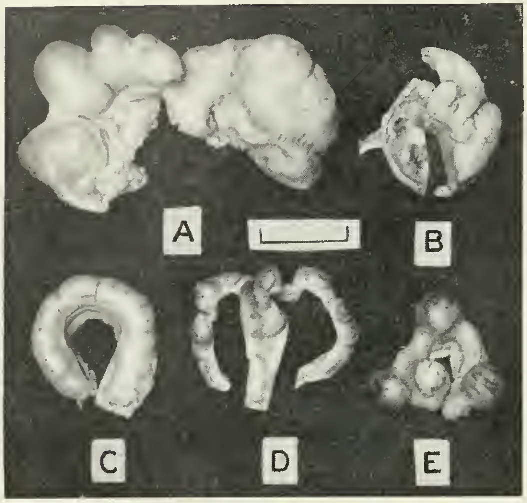


Fig. 1.

A, about two-thirds of the uterus of Case I. B, Case III. C, Case IV. D, normal rabbit uterus. E, normal early pregnancy. (Scale 1 in.)

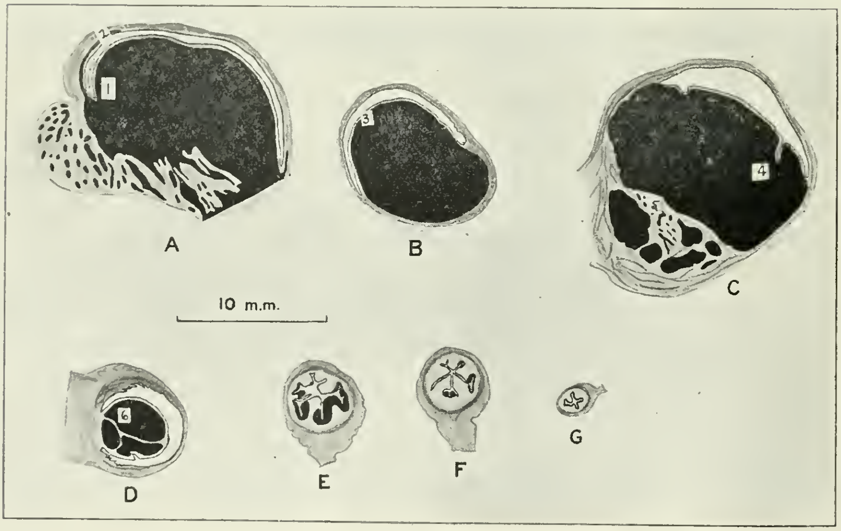


Fig. 2.

Transverse sections to show extent of epithelium. A, Case III. B, Case II. C, Case I. D, Case IV. E, F, and G, normal uteri.

occupied with five pregnancy cysts containing fœtuses about 10 mm. long. It is probably significant that the placentation of these fœtuses is abnormal, for, though the overgrowth of the maternal epithelium and adjacent connective tissue occupies about the normal area, the actual placenta is not more than one-quarter the proper size, and is growing only at one side of the maternal pad.

Case II (figs. 2, B, and 5).—The uterus is empty except for a single tumour mass about 2 cm. by $1\frac{1}{2}$ cm. The epithelial part of the tumour is formed of solid masses and alveoli in about equal proportions; some of the alveoli seem to be much distended by pressure, and the epithelium is stretched out into flat, instead of cubical, cells (fig. 5, which also shows the epithelium lining both sides of the uterine cavity). There are a good many papillary intra-alveolar growths.

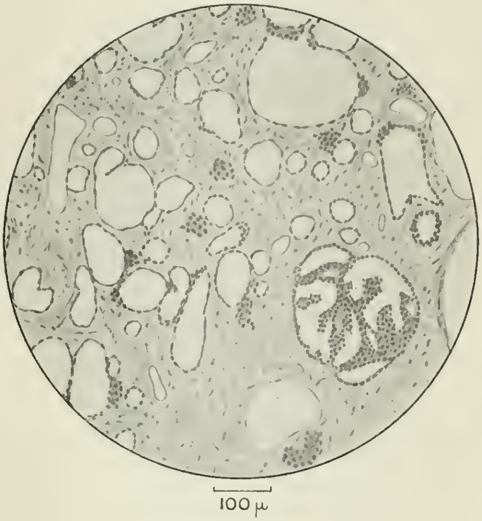


Fig. 3.

Case III (fig. 2, A, 1).

Case III (figs. 1, B, 2, A, 3 and 4).—The uterus is empty except for a single tumour mass about $6\frac{1}{2}$ cm. by 2 cm. by $1\frac{1}{2}$ cm., weighing about 12 grm. The tumour is almost entirely alveolar; many alveoli are lined with much flattened epithelium, and there are a number of intra-alveolar growths (fig. 3). The tumour in this case also extends into the wall of the uterine cavity over the fundus, and is spreading along between the deep ends of the glands and the muscular coat in the form of irregular alveoli lined with columnar epithelium (fig. 4).

Case IV (figs. 1, C, 2, D, and 8).—One-half of the uterus has five, the other six, small pedunculated tumours, arranged at approximately equal distances from one another. The epithelium is mostly arranged in small alveoli (fig. 8); there are some solid masses and a good many intra-alveolar growths. In one place there is hæmorrhage into the stroma, with some necrosis of both connective tissue and epithelium.

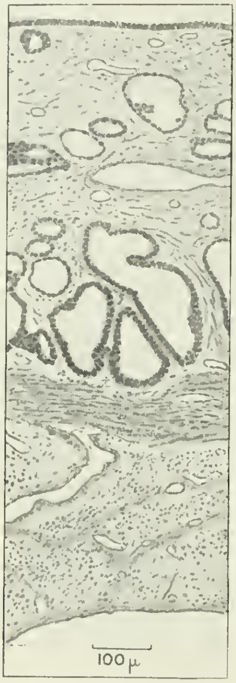


Fig. 4.
Case III (fig. 2, A, 2).

The tumours in all four cases, therefore, consist of a delicate stroma containing epithelium growing in an irregular way, either as alveoli suggesting an origin for tubular glands or as solid masses of cells closely packed together. In many instances the epithelium lining the alveoli has formed papillary intracystic growths and the extreme flattening of the lining epithelium which is seen in varying degrees indicate the retention of some secretion, though the alveoli in question have no obvious microscopical contents. All the tumours have a free blood supply; the vessels are quite large but have simple endothelial walls of only capillary thickness.

All the tumours appear to have arisen on the mesometric side of the uterus, that is, in the site of normal placentation. The uterine cavity lies on the side of the fundus and is completely lined with mucous membrane which does not differ materially from the normal. Different examples of the mucous membrane found in the non-pregnant uterus of normal rabbits are shown in fig. 9, A, B, and C; the epithelium of the uterus in one of the tumour cases is shown in fig. 4, and that lining both sides of the uterine cavity in another case in fig. 5. In no case have I been able to trace any continuity between the epithelium lining the uterine cavity and that forming the tumour.

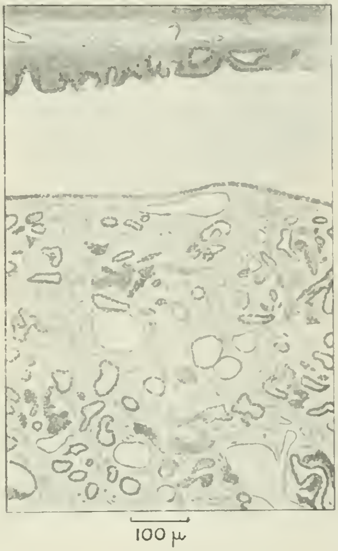


FIG. 5.

Case II (fig. 2, B, 3).

I conceive that these tumours have arisen in sequence to abortions. In normal pregnancy in rabbits[1] there is an overgrowth of the uterine

---

[1] For help here I am much indebted to Dr. J. W. Jenkinson, Lecturer on Embryology in the University of Oxford. Some information will be found in Duval, *Journ. de l'Anat. et de la Physiol.*, 1889, xxv, pp. 309, 573, and Maximow, *Arch. f. mikr. Anat.*, 1898, li, p. 68, and 1900, lvi, p. 699.

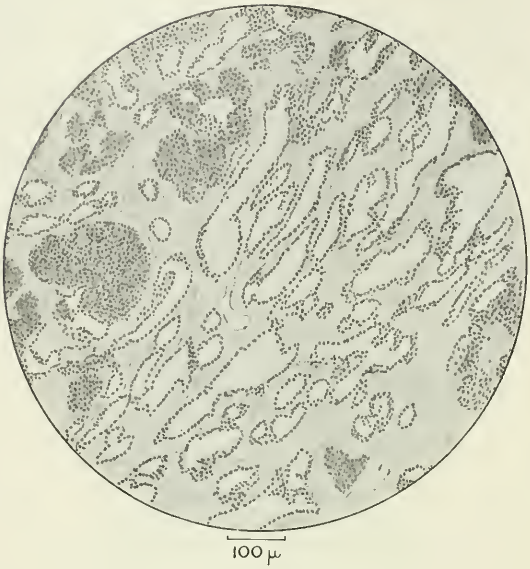


FIG. 6.

Case I (fig. 2, C, 4).

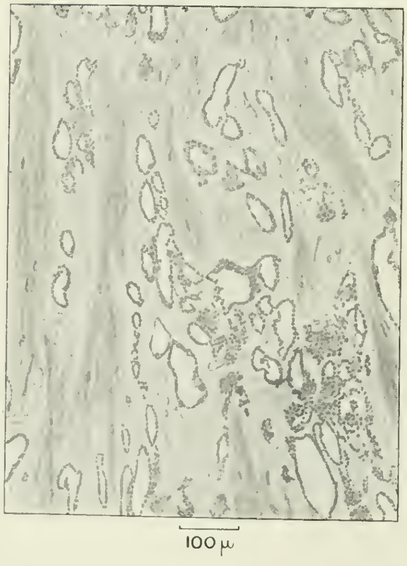


FIG. 7.

Case I (fig. 2, C, 5).

mucosa at and immediately surrounding the area of placentation. The epithelium grows out into papillæ (fig. 9, D) and the glands, which are imbedded in loose connective tissue, become much larger and more complex in structure (fig. 9, E); in both parts the epithelial cells are obviously much larger than normal. At the site of placentation the uterine ends of the glands degenerate (fig. 9, E) and are destroyed by the growth of the placenta from the fœtus. The deeper parts remain embedded in the overgrown mass of connective tissue (maternal glycogenic tissue) which forms the bed for the placenta. It seems possible that, if at this stage the fœtus and placenta come to a bad end, the uterine epithelium might grow across from the edges of the site of placentation and so leave the deep ends of the overgrown glands isolated and with no openings into the uterine cavity. Under these circumstances it would not be surprising if an irregular growth of epithelium in some cases arose.

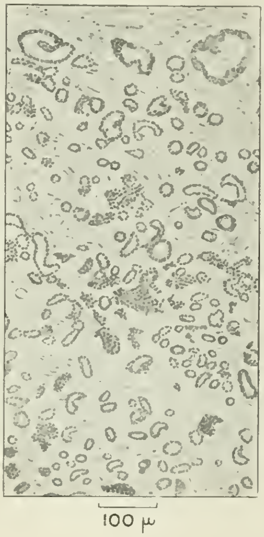


Fig. 8.

Case IV (fig. 2, D, 6).

In no case could I find any remains of fœtus in the tumours, but the explanation suggested receives some support from the facts that in Case IV the eleven tumours were arranged in a row in both halves of the uterus just as are normal placentations, and in Case I the half of the uterus which had no tumours contained fœtuses with abnormal placentations of a kind which would appear likely to lead to abortion. It is also of interest to note that all four cases occurred during the breeding season and in a series of between 140 and 150 female rabbits, so that, at that time, it was quite a common condition. If this interpretation is correct, the tumour would be a real "deciduoma."

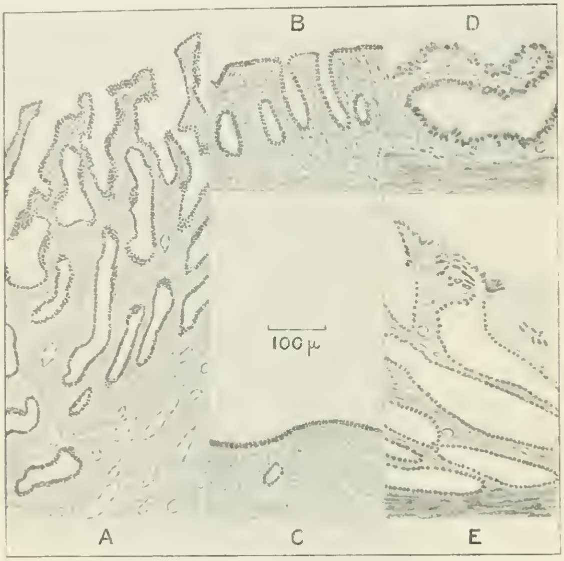


Fig. 9.

A, B, C, mucosa in three different normal uteri. D, mucosa in normal pregnancy close to edge of placenta. E, mucosa in a more advanced normal pregnancy at edge of placenta. All the figures are drawn to the same scale.

Further information is evidently wanted in these directions: whether the local infiltration seen in Case I ever extends to general malignancy, whether the tumours are transplantable, whether they can be produced experimentally by interference with early pregnancies.

Action of Pro- and Anti-thyroid Preparations.

By WALTER EDMUNDS.

IN a former communication[1] the results of certain experiments with the feeding of rats on thyroideum siccum and antithyroid preparations were given: the conclusion arrived at being that the antithyroid preparations did no good in mitigating the effects of chronic thyroid poisoning. The experiments were few in number and have therefore since been continued.

Dr. MacConkey, of the Lister Institute's Serum Branch at Elstree, kindly prepared an "antithyroid" powder from the blood of certain goats from which I had previously removed the thyroid gland. The blood was drawn and whipped to remove the fibrin; the serum, containing the corpuscles, was then evaporated to dryness—not by heat, which it was feared might decompose the antibody, but by a low barometric pressure; the residue was then powdered: 10 c.c. of blood gave 1 grm. of powder.

To two rats were administered, three times a day, 10 gr. (0·64 grm.) of Elstree antithyroid, and to two other rats was given the same dose and also 10 gr., three times a day, of thyroideum siccum B.P. In all four cases the experiment had to be stopped on the eighth day because the supply of antithyroid powder had run short, but the two rats which had this only remained well, whereas the other two rats which had thyroideum in addition had lost flesh considerably.

Another experiment was made: to one rat was given 5 gr. of thyroideum siccum three times a day, and to another the same, and in addition 10 gr. of the antithyroid powder, also three times a day; both rats died on the tenth day. A second pair of experiments were made: to one rat was given thyroideum, 2½ gr. three times a day, and to another the same dose, and in addition the antithyroid powder, 10 gr. three times a day; both rats died on the eighteenth day. These results seem to show that not much good can be got from the antithyroid preparation in the treatment of continued thyroid poisoning in the doses here given.

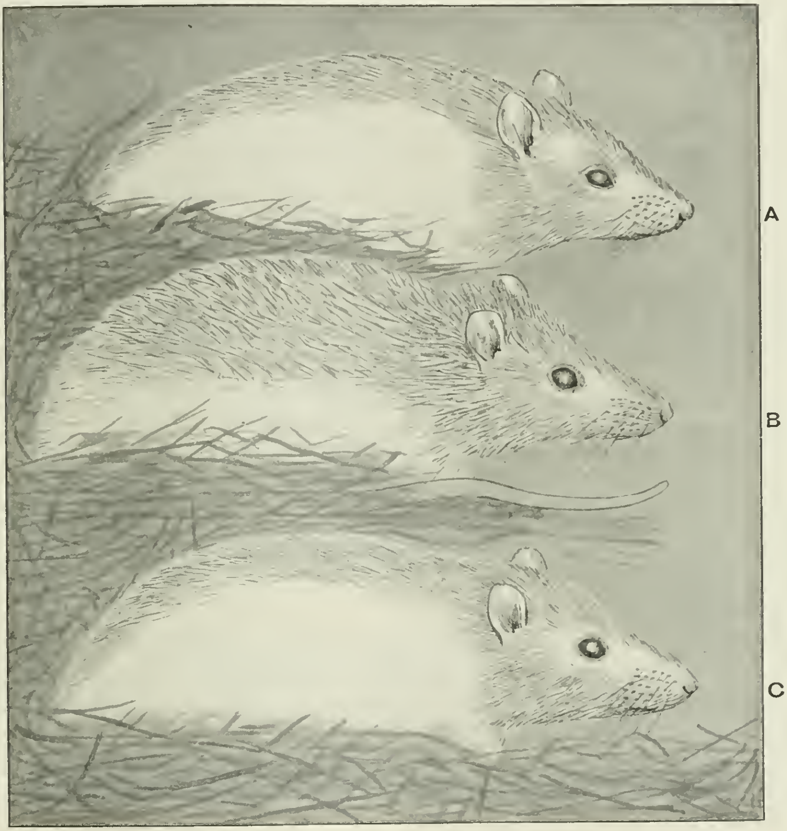
The doses of thyroid used seemed very large, especially the 10 gr. three times a day, and accordingly smaller doses were given: to one rat was given 1 gr. of thyroid three times a day, and to another the same, and in addition 10 gr. of a commercial antithyroid preparation three times a day; the first rat died in seven days and the second in twenty-two days. Both rats showed changes post mortem pointing to the thyroid poisoning being the cause of death, therefore, on this occasion, the antithyroid seemed to be of some benefit.

---

[1] January 18, 1910; see *Proceedings*, 1910, iii, p. 100.

Experiments were now made with thyroid feeding, and the administration of the milk of thyroidless goats, and also, as a control, of a normal goat. A rat was fed on the milk of a thyroidless goat, and, when it was seen that the rat remained well on this diet, there was given 10 gr. of thyroideum three times a day; the rat died in thirty-two days. A series of experiments were now made, giving to rats ½ gr. of thyroideum twice or thrice a day and feeding them exclusively on fresh milk from goats kept for that purpose at the Brown Institution; three of the goats were thyroidless and one normal: in all the experiments the dose of thyroid was ½ gr., but in the first series it was given three times a day and in the second twice. The results in the first series were: Fed on the milk of thyroidless goats (eight experiments), the average duration of life under thyroid feeding was twenty-one days, the shortest being nine days and the longest thirty-six days; fed on normal goats' milk (eight experiments) the average duration of life under thyroid feeding was thirty-nine days, the shortest being nine days and the longest sixty-seven days; the longest was probably more than sixty-seven days, for the thyroid feeding was then stopped: in all cases the post-mortem results pointed to continued thyroid feeding being the cause of death. In the second series of cases in six rats fed on the milk of thyroidless goats and given ½ gr. of thyroideum twice (instead of thrice) a day the average duration of life was thirty-two days, the shortest being ten days and the longest fifty-one days. Two rats were similarly treated and fed on the milk of a normal goat: one died in nineteen days and the other in sixty-six days (for the last week this rat was fed on normal cows' milk instead of normal goats' milk). The only fallacy one can think of in the above experiments is that the milk of the normal goat, which was an Anglo-Nubian, may have been richer than that of the other goats, but any difference could have been made up for by the rats taking a little more milk.

When one remembers that, as Andersson [2] has shown, there are secretory nerves to the thyroid gland, it seems probable that the output of thyroid secretion is proportional to the demand and that there is no material excess to be excreted in the milk or urine. The normal kid has a thyroid of its own, presumably adequate to its needs; there is therefore no such good reason why the milk should contain thyroid as why it should contain calcium, as it does in large quantities, for the formation of the kid's bones. We will conclude, therefore, that the milk of normal goats contains no thyroid to speak of. In the case of thyroidless goats the matter is different: two opposite views can be taken as to the effect of thyroidectomy on the milk of a goat. It may be suggested that the thyroid is a poisonous body and that its poison is counteracted by another body to be called antithyroid; that when the



A, a normal white rat. B, white rat after being fed for fifty-three days on thyroideum, ½ gr. three times a day; notice width of the palpebral fissure; the eyes also were prominent. C, the same rat twenty-one days after stopping the thyroid feeding; the eyes were still prominent; they remain so now thirty-one days later.

thyroid is excised the antithyroid remains free in the blood and can be obtained in the blood serum or milk and used to counteract the excess of thyroid of Graves's disease: this is the principle underlying the more usual methods of "antithyroid" treatment of that disease. The other view is this: Excision of the thyroid often causes myxœdema, this is an undoubted fact; three of the goats on which I operated died of myxœdema, and very possibly the others did not die because an antibody formed and protected them. Under this view this antibody must be an anti-myxœdema body, and therefore a pro-thyroid body; further, it must be formed in excess if it is to be found in the blood-serum or the milk. This would not be different to what occurs with other antitoxins. The antitoxin of diphtheria must be in excess in the blood of the immune horse, and a syphilitic infant is said to have been cured by the milk from a syphilitic mother under treatment by salvarsan, although that milk contained no arsenic [1]. It was on this principle that Stradiotti [6] injected sheep with a thyroid preparation and administered the sheep's serum to patients suffering from Graves's disease; the treatment was followed by improvement of the symptoms. Similar experiments were made by MacCallum [4], Rogers [5], and Beebe [3].

It is this latter theory that is supported by the experiments here related: the hyperthyroidic rats died sooner when taking the milk from thyroidless goats (milk which should contain an anti-myxœdema body—that is to say, a pro-thyroid body) than when taking the milk from a normal goat.

It was stated that in one experiment the administration of thyroid to the rat was stopped and the normal goats' milk continued. The loss of flesh quickly got much better, but the exophthalmos only very slowly improved, suggesting some anatomical alteration; this is a condition which often occurs in Graves's disease, the exophthalmos being the last symptom to improve (*see* fig.).

REFERENCES.

[1] Alston, H. *Brit. Med. Journ.*, 1911, i, p. 360.
[2] Andersson. *Arch. f. Anat. u. Phys.* (Anat. Abth.), 1894, p. 177.
[3] Beebe, S. P. *Journ. Amer. Med. Assoc.*, Chicago, 1906, xlvii, p. 661.
[4] MacCallum. *Med. News*, New York, 1903, lxxxiii, p. 820.
[5] Rogers, J. *Journ. Amer. Med. Assoc.*, 1906, xlvii, p. 655.
[6] Stradiotti. *Brit. Med. Journ.*, 1907, i (Epitome), p. 95.

Pathological Section.

March 21, 1911.

Dr. F. W. Mott, F.R.S., President of the Section, in the Chair.

A Case of Resection of the Cæcum and Ascending Colon, showing Chronic Hypertrophy of the Mucous Membrane, with Stenosis simulating Cancer.

By C. Gordon Watson.

The following is a short history of the case. A. C., a woman aged 46, was admitted to St. Bartholomew's Hospital on September 4, 1910, with a history of frequent attacks of colicky pains in the abdomen, and the occasional passage of dark blood *per rectum* during the past six months or so. During the last few months the patient had lost weight rapidly. She had not suffered from diarrhœa or constipation.

Condition on admission: The patient was somewhat wasted and anæmic; the abdomen not distended; nothing abnormal could be felt either *per abdomen* or *per rectum*. On examination under an anæsthetic a well-defined sausage-shaped tumour could be felt in the region of the cæcum. The abdomen was opened on September 12, and the tumour felt was found to involve the cæcum and part of the ascending colon, and at first sight suggested an intussusception. On further examination it was found that the bowel wall in this region was considerably thickened, and extremely resistant to pressure. The infiltrated area was sharply defined both above and below. A few enlarged glands were seen in the neighbourhood of the cæcum. A lateral anastomosis was made between ileum and the transverse colon, close to the hepatic flexure, and the abdomen closed.

On September 29, seventeen days later, the abdomen was again opened, and the portion of gut now shown resected. The patient made a good recovery from the operation, although the recovery was delayed by suppuration in the abdominal wall.

Since the operation the patient has gained weight, and is now in very good health.

The following is a description of the specimen as drawn up by the Assistant Curator of St. Bartholomew's Hospital Museum :—

" A resection of the intestine, which includes 2½ in. of ileum, cæcum, appendix, and 8½ in. of ascending colon. The ileum appears normal. The mucous membrane lining the posterior wall of the cæcum appears healthy, but that of the anterior wall has a great number of polypoid villi varying in length from ⅛ in. to ¼ in. The muscular wall is hypertrophied, and there appears to be more fat than normal. In the colon a similar condition is seen, except that the polypi are longer up to a point 3 in. above the ileo-cæcal valve, where a stricture partially occludes the lumen of the intestine. From this point, and extending 5 in. along the colon, the mucous membrane is covered with large flat tag-like processes in great numbers. An especially long and broad example is to be noted springing from the posterior wall. This appearance of the mucous membrane ceases abruptly at a point ½ in. below the end of the specimen, and the mucous membrane beyond this is normal. The line of demarcation at both ends between the normal and abnormal mucous membrane is very definite and almost annular. There seems to be more fat than normal in the outer wall and in the appendices epiploicæ. The muscular coat is greatly hypertrophied. Some small glands were found on the posterior aspect."

The following is a microscopical report from Mr. Roberts, of the Pathological Department, St. Bartholomew's Hospital :—

(1) "*Section of Colon.*—The mucosa is thickened, the glandular tubules, which are lined by regular columnar epithelium, are widely separated by a cellular infiltration, the elements of which are small round (lymphoid) cells, fibroblasts, and plasma cells. The surface is necrotic in some places. The inner muscular coat is hypertrophied, and is broken up into short segments by small bands of fibrous tissue, and cells running at right angles to the axis of the gut; the cells are largely plasma cells. The outer coats also contain cells and fibrous tissue, but are not so much affected. Nowhere is there any histological evidence of tubercle; there are no giant cells and no endothelial tubercles; tubercle bacilli could not be demonstrated in the sections.

(2) "*Sections of Five Lymphatic Glands.*—Sections of the glands show no evidence of tuberculosis or of malignant growth. No tubercle bacilli were demonstrated in them."

It seems clear that we are dealing with a case of chronic inflammation involving a portion of the cæcum and ascending colon. This inflammation involves all the coats of the bowel except the serous coat, and has resulted in a considerable hypertrophy of the mucous membrane, and has at one point produced some stenosis of the lumen of the bowel. The diseased area is sharply defined from the normal, both above and below.

To describe this case as one of hyperplastic pericolitis, colitis polyposa, or colitis fibromatosa does not help in the elucidation of its pathology. Can this specimen be regarded as one of hyperplastic tuberculosis? We know that tubercle is met with in the colon in three distinct forms—the miliary, ulcerative, and hyperplastic. The hyperplastic form is usually localized, gives rise to a definite tumour, and has frequently been mistaken for malignant new growth. It is not often associated with ulceration of the mucosa, and quite commonly produces some stricture of the bowel. The neighbouring glands are usually enlarged, and sometimes definitely tuberculous. It usually involves the cæcum or sigmoid—more often the former.

From published accounts there seem to be two distinct types, the submucous and subserous, but more often there is a combination of the two. The former to the naked eye closely resembles a neoplasm, the latter is more readily distinguished as an inflammatory lesion. The thickening of the wall of the intestine consists of firm connective tissue and fat, and though in some cases definite tubercles and a few bacilli are seen, in the majority, which in other respects resemble the specimens containing tubercle, no tubercles are found. The wall of the cæcum is usually firm and unyielding. This was so marked in my case as to resemble an intussusception. With but a single exception in the recorded cases, there has been a more or less marked stricture at or near the ileocæcal valve. The hyperplasia of the coats is just as marked below as above the stricture, and is not caused by the stricture. In the case in which no stricture existed (recorded by Caussade and Charrier) the hyperplasia was well marked. With regard to the mucous membrane, Crowder, in describing a typical case, says "the mucous membrane is much thickened and beset with little papillomatous masses of variable size, which constitute one of the striking and almost constant features of the pathology of this condition."

Conrath [1], in 1898, collected eighty-five cases, chiefly from German literature, and a drawing which he reproduces of one specimen (which I now show) is not unlike the specimen under discussion. Doubtless in the past many such cases have been classed as carcinoma, and so lost to the literature. Crowder [2], in 1900, published two cases with a good account of the literature of the subject up to date. The specimen I show corresponds very closely to the description given of his first case, though with this important difference, that tubercles were present in portions of his specimen, and are not seen in mine. F. S. Kidd [3], in the *Lancet*, 1907, describes three cases as hyperplastic tuberculosis, which bear a resemblance to the specimen now shown, and in which no tubercles were demonstrated. In Kidd's cases the subserous coat was mainly involved, and the mucous membrane was but little affected, although in one case it was slightly ulcerated. Lockhart Mummery [4] showed a specimen at the Clinical Section last year, which he described as hyperplastic tuberculosis of the sigmoid. I saw the specimen and the section. The specimen certainly resembled the one now shown, although there was more extensive hyperplasia. In most of the recorded cases a more extensive hyperplasia has existed than in the specimen shown, but it is reasonable to suppose that in this case the hyperplasia would have increased if the operation had been delayed.

At the last meeting of the British Medical Association, Alexis Thomson read a paper on "Fibromatosis of the Stomach," with the object of showing that many cases of leather-bottle stomach, previously regarded as malignant, are in reality inflammatory. On reading his account of the microscopical appearances one is struck with the resemblance to the cases described as hyperplastic tuberculosis of the colon.

Several cases are recorded of hyperplastic tuberculosis of the colon associated with malignant disease, and it seems reasonable to suppose that such a condition, whether in the stomach or colon, would predispose to carcinoma.

With regard to treatment, it might be argued that resection is too severe a measure for a disease which is not malignant. On the other hand, a certainty in diagnosis can seldom be made unless a gland reveals either growth or tubercle. More information is required as to the value of a simple short circuit without resection. If the cæcum is put out of circuit, will the condition retrogress or progress? If it progresses and the stricture, which in nearly every recorded case exists, increases, then perforation of the cæcum may occur despite the short circuit, because the ileo-cæcal valve allows of no backflow. In some cases suppuration with general peritonitis had occurred, as in D'Arcy Power's case and in one of Kidd's; in others intestinal obstruction has resulted from adhesions to the small intestine. It seems not unlikely that some of the cases of reputed disappearance of malignant tumours of the bowel may be explained on the assumption that the condition was really hyperplastic tuberculosis, which retrogressed either with or without a short circuit.

I do not like to draw conclusions from one successful case, but I think that if I met with a similar case I should adopt a similar mode of treatment rather than risk having to resect at a later date under more urgent or more difficult conditions.

REFERENCES.

[1] v. Conrath. "Ueber die lokale chronische Cœcumtuberkulose," *Beitr. z. klin. Chir.*, Tüb., 1898, xxi, pp. 1-110.

[2] Crowder, T. R. "Hyperplastic Tuberculosis of the Cæcum," *Amer. Journ. Med. Sci.*, Philad., 1900, N.S., cxix, p. 668, with references.

[3] Kidd, F. S. *Lancet*, 1907, i, p. 9.

[4] Mummery, P. L. *Proc. Roy. Soc. Med.*, 1910, iii (Clin. Sect.), p. 201.

On the Relation of the Meningococcus Intracellularis to Pseudoglioma.[1]

By GEORGE COATS and J. GRAHAM FORBES.

THAT metastatic ophthalmitis occurs with some frequency in epidemic cerebrospinal meningitis has long been known; that the meningococcus is capable of causing in children a metastatic ophthalmitis of pseudogliomatous type, without associated symptoms of meningitis, appears to have escaped recognition. We present some illustrative cases.

CASE I.

Simultaneous onset, without constitutional symptoms, of metastatic ophthalmitis and arthritis of the left wrist-joint. No history of specific infectious disease. Temporary improvement of the ocular symptoms under treatment, followed by retrogression. Excision. Primary seat of metastasis, the retina. Meningococci recovered from the vitreous.

I. C., aged 4½, was admitted to the Hospital for Sick Children on October 7, 1910. On October 6, without signs of general illness, the left wrist-joint became swollen and painful. A doctor who was consulted observed pus in the anterior chamber of the left eye, and sent the child to the hospital. She had had none of the infectious fevers, the two other members of the same family were quite well; no fevers (including "spotted fever") were known to be prevalent in the neighbourhood. The right eye was externally normal. In the left there was moderate conjunctival and episcleral congestion. The cornea was clear, the pupil fairly well dilated, and pus was coming forward through it into the anterior chamber. The child seemed to suffer little or no pain, and appeared to be in perfect health, head symptoms being entirely absent. The left wrist-joint was swollen, but with little tenderness, and no redness of the skin. The arthritis subsided in about a week. On admission the temperature was 102° F.; it dropped at once to normal, and never subsequently rose above 100° F. There was no history or evidence of vaginal discharge. Under atropine and hot bathing the congestion of the eye decreased, and the pus in the anterior chamber became partially absorbed. After about a week, however, the hypopyon increased once more, and the iris became very vascular, a large staphyloma gradually formed in the intercalary region above, and the pupil became vertically oval. The eye was accordingly excised on October 19.

---

[1] This term is used by clinicians to indicate appearances suggestive of glioma, but unconnected with new growth. Such appearances are most commonly due to a chronic metastatic ophthalmitis, and it is in this sense that we employ the word, leaving out of consideration other rarer conditions which may also be mistaken for glioma—e.g., persistent hyaloid artery, tubercle of the choroid, exudative retinal disease, &c.

Pathological examination : The primary seat of metastasis was probably the equatorial region of the retina. In this situation the retina is deeply buried in purulent infiltration and practically destroyed. The vessels are greatly congested, and there is some laying down of organizing material in their walls. Elsewhere also the retina is deeply infiltrated, and coagula and hæmorrhages are present in the subretinal space. The vitreous is full of pus and coagulum, and organization is commencing in the vicinity of the ciliary body. The papilla is moderately swollen, and the nerve slightly hypercellular for a short distance above the lamina cribrosa. There is no inflammatory infiltration in the intervaginal space. The choroid is practically free from infiltration, the ciliary body only moderately inflamed, and chiefly in the pars plana. The iris is much destroyed and deeply infiltrated. At the sclero-corneal margin above there is a large perforation of the sclera into which the iris and anterior part of the ciliary body are prolapsed. The lens is ruptured at its upper equator. The cornea shows moderate infiltration and œdema.

Bacteriological examination : Immediately after excision a specimen of the pus in the vitreous was obtained under aseptic precautions. It was very viscid, and yielded in film preparations a scanty number of Gram-negative diplococci, both intra- and extra-cellular, but chiefly the latter. By cultivation at 37° C. a pure growth was obtained on serum of a Gram-negative diplococcus, indistinguishable from the meningococcus. Growth failed to develop at 22° C. Fermentation tests applied to the diplococcus yielded acid reactions with galactose, glucose, and maltose, but failed to ferment saccharose. They were therefore in accordance with the behaviour of the meningococcus.

CASE II.

Metastatic ophthalmitis first noticed a week after the onset of arthritis in the left wrist-joint. No history of specific infectious disease. Perforation of the globe at the equator. Excision. Metastatic retinitis. Meningococcus in the vitreous.

B. W., aged 4 months, was admitted to the Hospital for Sick Children under the care of Mr. H. A. T. Fairbank, to whom we are indebted for permission to make use of the case. There was no history of specific infectious disease. No vaginal discharge. Three weeks previously a swelling had appeared on the dorsum of the left wrist ; this had disappeared before the child was brought to hospital. A week later the left eye was noticed to be swollen. When seen a fortnight afterwards there was considerable proptosis, and a little chemosis. The globe was soft, the pupil contracted and bound down, the iris pushed forward against the back of the cornea. There was a yellowish reflex from behind the lens. Orbital incisions were made without result, and as the globe was beginning to become staphylomatous on the outer side, it was excised. In doing so an extrascleral abscess was opened into at the equator.

Pathological examination: This case also seems to be one of metastatic retinitis. Some scarcely distinguishable remains of the retina are present close behind the lens; in the posterior part of the globe it seems to have been entirely destroyed. The globe is filled with polymorphonuclear leucocytes. On the inner surface of the choroid there is a thick layer of inflammatory cells, which is becoming invaded from below by numerous new-formed vessels. The choroid itself is congested, and its stroma is more prominent and fibrous than normal; it shows, however, only a moderate amount of infiltration except in its inner layers anteriorly. The anterior portion of the ciliary body is moderately deeply infiltrated; the pars plana is broken up and destroyed, and from it dense masses of young cicatricial tissue stretch inwards, filling up the posterior chamber and replacing the anterior part of the vitreous. The iris is congested and moderately deeply infiltrated, the infiltration being diffuse, not focal. On its posterior aspect the iris is extensively adherent, centrally to the lens, peripherally to organizing membranes in the posterior chamber. A cicatricial membrane stretches across the pupil. The cornea is practically free from inflammation. Coagulum and leucocytes are present in the anterior chamber. At the site of perforation the pus forms an extra-ocular abscess which is partially isolated by a condensation of the surrounding orbital tissues. Elsewhere the sclera is but little infiltrated.

Bacteriological examination: The pus is very viscid. Films were prepared with difficulty, and showed Gram-negative intra- and extra-cellular diplococci in small numbers. Cultivations from the pus were made on serum and agar, and yielded at 37° C. a Gram-negative diplococcus culturally and morphologically indistinguishable from the meningococcus. Subcultures were planted on serum and agar, but failed to grow on the latter medium. At 22° C. a very feeble growth was obtained on serum, not developing, however, in further subculture. Fermentation tests yielded the following results at 37° C.:—

Saccharose	−	Galactose	+
Lactose	−	Maltose	+
Glucose	+	Lævulose	+

(− = no change in reaction. + = acid reaction.)

Controls planted from a meningococcus obtained from the cerebrospinal fluid of a case of post-basic meningitis yielded precisely similar acid reactions with glucose, galactose, maltose, lævulose, but no change with saccharose and lactose.

Further confirmation of the identity of the organism obtained in this case was kindly provided by Dr. J. A. Arkwright, of the Lister Institute, who submitted the diplococcus to cultural, fermentation, and agglutination tests. From these he concluded that the organism was a typical meningococcus. It produced acid in liquid media from glucose and maltose, and *not* from cane sugar.

The agglutination with meningococcus serum was fair in 1 in 200 dilution, and partial in 1 in 500 dilution. With normal horse serum control no agglutination was obtained.

CASE III.

Metastatic ophthalmitis of the right eye following measles. Eyes buphthalmic. Excision of right eye. Metastatic retinitis and ? choroiditis. Gram-negative diplococci with the morphological characters of the meningococcus found in the vicinity of the retina at the equator.

A. M., aged 9 months, was admitted on July 31, 1907, to the Royal London Ophthalmic Hospital, under the care of Mr. J. Herbert Parsons, who has kindly permitted us to use the case. The right eye had been bad for six days, following upon an attack of measles. Both eyes were buphthalmic. In the right the eyelids were swollen, the conjunctiva injected, the cornea dull, the anterior chamber shallow. The iris pattern was blurred and the pupil occluded. Under atropine the pupil failed to dilate, and on August 6 marked chemosis set in. The eye was excised the next day, and was found to be slightly adherent to the surrounding tissues.

Pathological examination: The globe measured antero-posteriorly 25 mm., vertically 23 mm., transversely 23·5 mm., the cornea 13 mm. by 11·5 mm. The retina is very deeply infiltrated throughout, but more especially about the equator, where it is entirely broken up and buried in dense collections of polymorphonuclear leucocytes. A considerable amount of pus is present in the subretinal space posteriorly. The choroid is not so much inflamed as the retina, but shows a good deal of congestion and infiltration of rather diffuse type; the infiltration is most dense at the equator on the temporal side, and in this situation the sclera is also slightly infiltrated. The nerve is somewhat hypercellular for some distance above the lamina cribrosa, but not so far as the cut end. The intervaginal space is normal. The ciliary body is moderately inflamed, chiefly in the pars plana. An extensive cyclitic membrane in an early stage of organization is present in the posterior chamber. It completely invests the equator and anterior surface of the lens; the iris is adherent to it in its entire extent, and the pigment epithelium is dragged upon and distorted. The contents of the vitreous are still purulent and show no organization, except a very slight amount on the surface of the papilla. The iris is congested and shows a moderate amount of diffuse infiltration, an inflammatory membrane is present on its anterior surface. The corneo-iridic angle is occluded, and the root of the iris is atrophic: these changes probably have to do with the buphthalmos, not with the panophthalmitis. The cornea is normal. The anterior chamber contains coagulum and a few leucocytes.

Bacteriological examination: In this and the following case the eyes were unfortunately fixed before cultures could be made. In the sections diplococci are found in moderate numbers in the pus of the vitreous. They are especially concentrated in the vicinity of the retina at the equator, and are not found with certainty in the choroid. Morphologically they show the typical characters of the gonococcus-meningococcus group. They are almost all intracellular, and some leucocytes are crowded with them. Swollen and degenerate forms are uncommon. They are exquisitely Gram-negative, giving up their colour even when the gentian violet is very imperfectly washed out of the nuclei.

CASE IV.

Metastatic ophthalmitis of the right eye following the onset of measles at an interval of a fortnight. Excision. Probably metastatic retinitis and choroiditis. Gram-negative diplococci morphologically similar to the meningococcus in the vitreous and subretinal space.

R. C., aged 15 months, was admitted to the Royal London Ophthalmic Hospital on May 12, 1907, under the care of Mr. Lang, to whom we are indebted for the use of the case. The child had suffered from measles three weeks before, and the right eye had been bad one week. The lids were swollen, there was chemosis and proptosis, the cornea was hazy, and a patch of discoloured lymph covered the pupil. The eye was excised the next day.

Pathological examination: The retina is partially detached posteriorly, and is very deeply infiltrated. In places, especially about the equator, it is completely destroyed. Large hæmorrhages are present here and there in its substance and on its surface. The subretinal space is full of pus. The choroid is very deeply infiltrated except over a small area at the posterior pole. The infiltration is uniform and equal in all the layers. Over extensive areas the epithelium is stripped up by exudation, and at the equator the inner layers of the choroid are quite broken up, the choroidal and subretinal exudates being continuous. At the equator also the sclera is slightly infiltrated and loosened in structure. The ciliary body is infiltrated chiefly in the pars plana. The vitreous and posterior chamber are full of pus, and in the posterior chamber there is a considerable amount of hæmorrhage. There is practically no organization of the exudates. The iris is rather densely infiltrated and some hæmorrhages are present in the stroma. The cornea is normal. A fibrinous coagulum, with a few leucocytes and red corpuscles in its meshes, occupies the anterior chamber.

Bacteriological examination: A Gram-negative diplococcus is present in the pus of the vitreous and subretinal space, especially about the equator of the globe. It is doubtful if it is present in the choroid. Its morphological characters are in general those of the gonococcus-meningococcus group, but many swollen degenerate forms occur. No capsule is visible. The cocci are found both within and without the cells, but are probably more frequently extracellular. In the anterior part of the vitreous they are much more scanty, and they are not found in the pus of the anterior chamber, iris, or ciliary body.

In two of our cases an organism was found identical in appearance, staining reactions, and cultural characteristics with the meningococcus. In the vitreous of the other two clinically similar cases, a microbe was present with similar morphological and staining peculiarities; although in these instances no cultures were available, there can be little doubt that the organism in all four was the same.

Since none of these cases showed symptoms of cerebrospinal meningitis, it appears that the meningococcus may give rise to a metastatic ophthalmitis unassociated with constitutional disturbance. Since also, according to our experience, the meningococcus seems to occur with considerable frequency in such cases, we are led to conjecture that it may play a chief, or probably a specific, part in the ætiology of that form of metastatic ophthalmitis in children which goes by the name of "pseudoglioma." In addition to the evidence of our own cases certain other considerations may be adduced in support of this conjecture.

(1) On a priori grounds there is much to be said in favour of regarding pseudoglioma as a clinical and pathological entity, liable to be imitated no doubt by other forms of ophthalmitis, yet in the main well defined in type, and probably due always to the same cause. Our own cases were of somewhat greater severity than is common in pseudoglioma, and in two instances a perforation of the sclera had taken place; had it not been so the eyes would not have been excised, and no opportunity would have been afforded of making an early pathological examination. Yet they conformed well to the general type: in Case I the symptoms, though very threatening at first, receded later, and the eye might perhaps have been saved but for the gradual formation of an intercalary staphyloma; in Case II the changes in the anterior part of the globe were insignificant, and the eye was lost owing to a perforation at the equator; in the other two cases also the anterior changes were not great, and the panophthalmitis was not of the severest type. According to Treacher Collins the usual primary seat of metastasis in pseudoglioma is the retina; so it was also in our cases. It might be supposed that the special clinical characteristics of pseudoglioma are due, not to a specific organism, but to a difference in the vital reaction of the child's tissues; in other words, that pseudoglioma is caused by the ordinary pyogenic organisms, but that the tissues of the child are capable of a more powerful resistance, thus overcoming the virulence of the microbes, and modifying their effects. Cases of metastatic ophthalmitis from the ordinary pyogenic organisms seem to be rare in children, but in one case of streptococcal infection which we

had the opportunity of examining there was no such modified reaction, the panophthalmitis being of a virulent type with commencing ring infiltration of the cornea. It is doubtful also if traumatic infections of the vitreous run a more favourable course in children than in adults.

(2) It is well known that in true epidemic cerebrospinal meningitis a form of metastatic ophthalmitis occurs which has the characteristic clinical features of pseudoglioma.[1] According to the descriptions of Axenfeld[2] and Uhthoff (loc. cit.) the ocular symptoms are first observed simultaneously with, or a few days after, the commencement of the meningitis. In some instances the disease begins insidiously with moderate injection and chemosis in one eye, or much more rarely in both; in other cases the onset is threatening, but the symptoms soon subside. The signs of iritis are present, and a hypopyon, usually transitory, may develop; sometimes, however, the anterior parts of the eye remain entirely free from inflammation. In the vitreous inflammatory exudates are formed, which rapidly become purulent, giving a characteristic yellow reflex from the depths of the eye. There is little or no pain. Perforation of the sclera occasionally occurs, but in general the form of the globe is preserved as the inflammation subsides and the vitreous exudates organize the lens and central portion of the iris are thrust forward, while the periphery of the iris is retracted. The globe becomes soft, and a secondary cataract may develop.

The above description might be applied word for word to an ordinary instance of idiopathic pseudoglioma, and since the meningococcus has been found several times[3] within the globe in these meningeal cases there seems considerable inherent probability of its being also the cause of idiopathic pseudoglioma.

---

[1] The following figures, quoted from the papers of Uhthoff (*Bericht. d. Ophth. Gesellsch.*, Heidelberg, 1905, xxxii, p. 84) and Hanke and Tertsch (*Klin. Monatsbl. f. Augenheilk.*, 1907, xlv, 2, p. 545), show the frequency of this association: Knapp, 4 per cent. to 5 per cent.; Salomon, 2 in 141; Jedrzejewicz, 2 in 28; Schirmer, 3 in 30; Rudnew Burjew, 1 in 10; Göppert, 3 in 44; Radman, 3 in 61; Curtius, 7 in 200; Wilbrand and Sänger, 0 in 25; Heine, 5 in 100; Uhthoff, 4 in 100. Ballantyne (*Brit. Med. Journ.*, 1907, ii, p. 190) saw no instance in 73 cases. According to Uhthoff, the frequency may be stated roughly at from 4 per cent. to 5 per cent., with apparently considerable variation in different epidemics. Lees and Barlow ("Simple Meningitis in Children," Clifford Allbutt's "System of Medicine," 1899, vii, p. 521) describe a case of posterior basic meningitis they had met with, associated with pseudoglioma, affecting the right eye only and dating from the onset of illness, which occurred three weeks after an attack of measles.

[2] *Archiv f. Ophthal.*, 1894, Bd. xl, iii, p. 1 (110), and Uhthoff, loc. cit.

[3] Axenfeld (footnote in a paper by Haglund, *Klin. Monatsbl. f. Augenheilk.*, 1900, xxxviii, Beilageheft, p. 72); Wintersteiner (*Wien. klin. Wochenschr.*, 1901, xiv, p. 996); Uhthoff (loc. cit.); Mayou (*Roy. Lond. Ophthal. Hosp. Reports*, 1906, xvi, p. 565); Hanke and Tertsch (loc. cit.); McKee (*Ophthal. Record*, Chicago, 1908, xvii, p. 438).

(3) Certain of the attributes of pseudoglioma suggest an association with the meningococcus. (*a*) Pseudoglioma is a disease of childhood; so also, in the vast majority of cases, is cerebrospinal meningitis.[1] (*b*) In cases of pseudoglioma, perhaps the commonest history is of antecedent transitory head symptoms, such as screaming, vomiting, headache, convulsions, delirium, apathy, unconsciousness, "water on the brain," "brain fever," &c.[2] It is probable that many of these cases are instances of sporadic or abortive cerebrospinal meningitis, and that the meningeal and ocular symptoms are alike due to the meningococcus. (*c*) A history of one of the exanthemata, especially of measles or whooping-cough, is also common. It is most interesting to observe that though the history was of measles in our third and fourth cases, yet the organism in the vitreous was the meningococcus; this seems very suggestive of a specific causal relation between the meningococcus and pseudoglioma, and tends to show that the action of the exanthemata is preparatory, either by lowering the general resistance or by causing inflammation of the mucous membranes, and so opening up a path of invasion. (*d*) In our first case there was an arthritis of the left wrist-joint, and in our second a history of a former joint affection. The association of arthritis with pseudoglioma has also been described by Nettleship.[3] In epidemic cerebrospinal meningitis and posterior basic meningitis, arthritis or periarthritis is an occasional complication.[4]

(4) The finding of the meningococcus in the vitreous apart from symptoms of cerebrospinal meningitis is not an isolated observation. The same organism has been found in the nasopharyngeal secretion in about 10 per cent. of normal persons who have been in contact with cases of cerebrospinal meningitis.[5] It has also been found in the

---

[1] Hirsch, A., "Handbook of Geographical and Historical Pathology," *New Sydenham Society's Trans.*, 1886. p. 568. Of Ballantyne's cases (loc. cit.), 81·5 per cent. were under 21 years of age. In the Upper Silesian epidemic, 90·5 per cent. of the patients were under the age of 16 years (Uhthoff, loc. cit.).

[2] Nettleship (*Trans. Ophthal. Soc.*, 1883, iii, p. 36) found such symptoms in eight out of twenty-seven cases; Treacher Collins (*Roy. Lond. Ophthal. Hosp. Reports*, 1893, xiii, p. 368) in six out of eleven.

[3] *Med. Times and Gaz.*, 1880, i, p. 63.

[4] Lees and Barlow ("Simple Meningitis in Children," Clifford Allbutt's "System of Medicine," 1899, vii, p. 534); Ormerod ("Epidemic Cerebrospinal Meningitis," *ibid.*, i, p. 669). Still mentions the occurrence of inflammation about the joints in four out of forty consecutive cases of post-basic meningitis (Allchin's "Manual of Medicine," 1900, i, p. 193).

[5] Lingelsheim (*Deutsch. med. Wochenschr.*, 1905, xxxi, pp. 1017, 1217), quoted by Gordon in his "Report to Local Government Board on the Micrococcus of Epidemic Cerebrospinal Meningitis, &c.," 1907. Goodwin and v. Sholly (*Journ. of Infect. Dis.*, Chicago, 1906 [Suppl. No. 2], pp. 21-34), quoted by Gordon.

conjunctival sac of persons who have had no communication with cases of that disease.[1]

SUMMARY.

We have found the meningococcus in four cases of metastatic ophthalmitis of pseudogliomatous type, and are led to believe that a specific causal relation may be established between this organism and pseudoglioma. Apart from our own observations we base this opinion on the following considerations:—

(*a*) Pseudoglioma is a well-defined clinical entity, and is therefore not unlikely to be due always to the same organism.

(*b*) A form of ophthalmitis identical with pseudoglioma occurs in from 4 per cent. to 5 per cent. of cases of epidemic cerebrospinal meningitis; the meningococcus has been recovered from the eye in such cases. Pseudoglioma and cerebrospinal meningitis are both diseases of childhood, and both are sometimes associated with arthritis.

(*c*) In idiopathic pseudoglioma a history of head symptoms, possibly due to meningitis or simply meningismus is very frequent; two of our cases prove that even when the history is not of head symptoms, but of measles, the pseudoglioma may still be due to the meningococcus.

---

[1] Brons (*Klin. Monatsbl. f. Augenheilk.*, 1907, xlv, i, p. 1, associated with the pneumococcus in a case of keratomalacia); McKee (*Ophthal. Record*, 1908, xvii, p. 438, normal conjunctiva); Lutz (*Klin. Monatsbl. f. Augenheilk.*, 1910, xlviii, i, p. 636, case of chronic conjunctivitis).

Studies in Complement Fixation with Strains of Typhoid, Paratyphoid, and Allied Organisms.

By H. R. DEAN.[1]

THE practical applications of the complement-fixation method may be divided under two headings: (1) The recognition of antibody with the aid of a known antigen; (2) the identification of an unknown antigen with the aid of known antibody. Under the second heading fall the very numerous researches which have been undertaken with a view to the finer differentiation of closely allied bacteria. If value is to be set on this differentiation method of classification it must be shown that it is absolutely specific.

Although the majority of workers have come to the conclusion that the complement-fixation method is extremely specific, a considerable number of papers have appeared in which it is claimed that differentiation may be impossible between races of bacteria usually recognized as distinct. Although a few workers have gone so far as to say that the method is useless for purposes of differentiation, the majority have only found difficulty in distinguishing between nearly allied organisms. In such cases reference is very frequently made to the presence of a group reaction which renders a sharp distinction difficult of attainment.

For a full account of the literature of the subject the reader is referred to the article on "Complement Fixation," by Meier (1909), in Weichardt's "Jahresbericht."

The differentiation of *Bacillus typhosus* from *Bacillus coli* was recorded in Bordet and Gengou's (1902) original paper. These authors immunized guinea-pigs with cultures of *Bacillus typhosus* and tested the serum so obtained with emulsions of *Bacillus coli* and emulsions of *Bacillus typhosus*. They found that complement was completely absorbed by relatively small amounts of typhoid bacillary emulsion in the presence of antityphoid serum, but that relatively large amounts of *Bacillus coli* emulsion mixed with the same antityphoid serum were necessary to produce even a partial fixation of complement. They concluded that the sensitizing substances of antityphoid serum were markedly but not absolutely specific. These results were confirmed in the same year by Widal and Le Sourd (1902). The differentiation of *Bacillus typhosus* and *Bacillus coli* by this method is also described by Ballner and Reibmayr (1908), Simnitzky (1908), and H. R. Dean (1910).

---

[1] From the Bacteriological Department of the Lister Institute. (Read at the meeting on January 17.)

The introduction by Wassermann (1905) of bacillary extracts in place of the previously used emulsions was followed by numerous papers dealing with complement fixation. Further improvements in the method of preparation of bacterial extracts were devised by Leuchs (1907), who was able to satisfactorily differentiate *Bacillus typhosus* from *Bacillus paratyphosus*, although his results showed considerable evidence of a strong group reaction. The results of Leuchs, as regards *Bacillus typhosus* and *Bacillus paratyphosus*, were confirmed by Ballner and Reibmayr (1908), who also found that differentiation was rendered difficult by the presence of a marked group reaction. They advised the use of greatly diluted antisera. They were able to distinguish *Bacillus typhosus* from *Bacillus coli*, and either of these organisms from *Bacillus paratyphosus* B or from *Bacillus typhi murium*; they were, however, unable to distinguish between *Bacillus paratyphosus* B and *Bacillus typhi murium*. An *antityphi murium* serum gave a marked reaction with extract of *Bacillus typhi murium* and an almost equally marked reaction with extract of *Bacillus paratyphosus* B. An antityphoid or an anticoli serum was found to give a strong reaction with its homologous extract and a less, but still evident "group reaction" with extracts of *Bacillus typhi murium* and *Bacillus paratyphosus* B. The *Bacillus paratyphosus* B and the *Bacillus typhi murium* extracts were found to give an identical "group reaction" with a heterologous serum. The group reactions observed by the agglutination and complement-fixation methods ran a parallel course. They considered that the complement-fixation method was as sharp and as specific as agglutination, but that it was not to be preferred as it was so much more complicated, and that consequently sources of error were numerous.

Simnitzky (1908) saturated an antityphoid serum with *Bacillus coli* and with *Bacillus paratyphosus* without removing the antibodies specific for *Bacillus typhosus*. If, however, the serum was treated with typhoid bacilli the complement binding properties for *Bacillus typhosus* were removed together with the group antibodies for *Bacillus paratyphosus* and *Bacillus coli*.

Posner (1908) worked with the serum of patients suffering from typhoid and paratyphoid infections. He concluded that the complement-fixation method was unsuitable for the differentiation of typhoid and paratyphoid infections.

Spät (1908), who also made use of clinical material, came to the conclusion that complement fixation did not afford a means of differentiating between the infections caused by members of the typhoid and paratyphoid group.

From these papers it appears that considerable difficulty has been experienced in the differentiation of typhoid from paratyphoid. Specific differences are recorded by Leuchs and Ballner and Reibmayr, who, however, found it necessary to employ very dilute antisera. The presence of a group reaction is described in both these papers and to this group reaction may probably be attributed the negative results published by Spät and Posner.

The experiments published by H. R. Dean (1910) showed that with the low dilutions of antiserum no differentiation was possible between *Bacillus typhosus*, *Bacillus paratyphosus* B and *Bacillus paratyphosus* A. When, however, the antiserum was highly diluted complete differentiation was obtained. Experiments were also published which confirmed the absorption experiments of Simnitzky.

One of the most useful applications of the complement-fixation method has been the attempt to classify the various strains which have been isolated from cases of food-poisoning. Sacquépeé (1907) was able to effect a separation into two groups which he called "Enteritis I" and "Enteritis II." The group Enteritis I comprises *Bacillus Gaertner*, *Bacillus Morseele* and *Bacillus Bruxelles*. The group Enteritis II comprises *Bacillus Aertryck*, *Bacillus Posen*, *Bacillus Düsseldorf*, *Bacillus Sirault*, the bacillus of hog-cholera, *Bacillus psittacosis*, and *Bacillus paratyphosus* B.

A serum prepared by immunization with any organism in Group I gave a marked reaction with any other member of Group I, but no reaction with any member of Group II. In the same way a serum prepared by immunization with any organism of Group II was specific in its action and reacted equally well with any of the members of Group II, but not with the members of Group I.

Pitt (1909) investigated the *Bacillus nodulifaciens bovis* (Langer) and decided that it should be included in the Gaertner group. He was, however, unable to distinguish *Bacillus Gaertner* from *Bacillus typhosus* by the complement-fixation method, and for this reason concluded that it was questionable if complement fixation was more delicate than the previously employed serological tests.

The classification of the organisms forming the food-poisoning group has recently been very fully investigated by Altmann (1910). This author, as a result of agglutination experiments, divided the Salmonella group into two sub-groups: Enteritis I—Paratyphoid B (Schottmüller), the bacillus of hog-cholera, *Bacillus psittacosis*, *Bacillus typhi murium*, the so-called "Schweinepest" bacillus, *Bacillus pseudo-tuberculosis* of guinea-pigs, and food-poisoning bacilli of the type of Flügge, that is to say, *Bacillus Aertryck*. Enteritis II—The rat virus organisms of the type of Danysz, Dunbar, and Issatschenko, food-poisoning bacilli of the type of *Bacillus Morseele*. For the purpose of his complement-fixation experiments Altmann employed seventeen strains from Group I, namely, eight strains of *Bacillus paratyphosus* B (Schottmüller), two strains of so-called "Schweinepest" bacilli, one strain of *Bacillus typhi murium*, one strain of *Bacillus pseudo-tuberculosis*, and one strain of *Bacillus psittacosis*. Five strains of Group II were used, namely, the strains *Morseele* and *Bruges*, and the three rat poisons, *Dunbar*, *Ratin*, and *Danysz*. Extracts of these organisms were made by Leuchs' method. Using a paratyphoid B serum, the author obtained a marked reaction with each of the seventeen strains from Group I, no reaction with the strains of Group II, and no reaction with *Bacillus typhosus* and *Bacillus coli*. An exactly similar result was obtained by the use of a serum derived from an animal immunized with *Bacillus suipestifer*.

Although the author states that no differentiation was obtained between the various strains of Group I, the published table shows that very considerable differences in the intensity of the reaction occurred. By the use of a serum derived from an animal immunized with *Bacillus Morseele*, Altmann obtained a marked reaction with the extracts prepared from the members of Group II, but no reaction with the extracts prepared from the strains included under Group I. He concludes that further differentiation within the two groups is impossible.

Bainbridge (1911) has shown that by the use of Castellani's absorption method it is possible to distinguish *Bacillus paratyphosus* B (Schottmüller) from *Bacillus Aertryck*. *Bacillus paratyphosus* B is associated by Bainbridge with paratyphoid fever, while the typical symptoms of meat-poisoning are associated with a bacillus which is in every respect identical with *Bacillus Aertryck*. No differentiation, however, is possible between *Bacillus Aertryck*, *Bacillus suipestifer*, and the bacillus associated with the epizootic disease of guinea-pigs.

It will be seen that Bainbridge's results are opposed to those of Altmann, who was unable to differentiate between *Bacillus paratyphosus* B and *Bacillus Aertryck*.

From these and other papers on the subject it appears that the complement-fixation method is a means of demonstrating a highly specific reaction between a bacillary extract and its homologous antiserum. The specific reaction is, however, in considerable danger of being masked by the presence of a more or less strong group reaction. The group reaction, while it adds considerably to the difficulties of differentiation, is not without interest, from the insight which it affords as to the degree of relationship between nearly allied organisms.

GENERAL OBJECT OF THE EXPERIMENTS.

The object of the experiments which are to be described has been:—

(1) To determine the conditions under which the most complete differentiation can be affected.

(2) To determine the influence of relative excess of antigen and of relative excess of antibody on the absorption of complement.

(3) To determine if it is possible, by the use of the complement-fixation method, to differentiate *Bacillus paratyphosus* B (Schottmüller) from *Bacillus Aertryck*.

METHODS.

Preparation of Bacterial Extracts.—All the extracts used in these experiments were prepared by the same method. Roux bottles containing nutrient agar were freely inoculated from a broth culture and kept in the incubator at 37° C. for forty-eight hours. The agar surface of such bottles is about 20 cm. by 10 cm. After forty-eight hours, the growth in each bottle was rapidly emulsified with 20 c.c. of distilled water. The bacterial emulsion was then decanted into a sterile flask and subsequently pipetted off into sterile test-tubes, in amounts of about 5 c.c. to each tube. In some cases the emulsion was treated for one hour at 60° C. This preliminary heating was not found to have any effect favourable or unfavourable on the properties of the extract, but it is recommended, as it obviates risk of infection. The tubes containing the bacterial emulsion were, after a thorough shaking, immersed in an ice and salt freezing mixture. The emulsion was allowed to freeze hard, and then to thaw slowly at room temperature. The emulsion was usually prepared in the morning, and after the first freezing was allowed to stand at room temperature, with occasional shaking, until the evening, when the freezing process was repeated. After the emulsion had been thawed for the second time, it was placed in a strong bottle containing glass beads and shaken all night in a shaking-machine. On the second day the emulsion was pipetted in quantities of 5 c.c. into test-tubes, and the freezing and thawing process was repeated in the morning and in the evening. During the second night the emulsion was again thoroughly shaken in a strong bottle containing glass beads. On the third day the emulsion was centrifugalized. The supernatant fluid, which was either quite clear or only very slightly opalescent, was decanted into a sterile flask and stored for use in the cold room.

Antisera.—The antisera were prepared by the intravenous injection of rabbits with saline emulsions of twenty-four-hour agar cultures. Each rabbit received four or five injections at intervals of four or five days, and was tested on the eighth day after the last injection. If it was found to be satisfactory, the animal was bled out on the ninth or tenth day. The serum, which had separated after twenty-four hours, was heated for half an hour at 56° C. The inactivated serum was stored in small sealed glass bottles, each of which contained about 2 c.c.

THE HÆMOLYTIC SYSTEM.

The hæmolytic system consisted of washed sheep corpuscles, hæmolytic serum derived from rabbits which had been injected with sheep corpuscles, and fresh guinea-pig serum. The guinea-pig serum was freshly prepared on the same day as the experiments were carried out. The hæmolytic system was titrated on each occasion, and an amount of hæmolysin was used in the complement-fixation experiment, which contained about four minimal lytic doses.

EXPERIMENTAL METHODS.

The bulk of fluid in every tube was 2·5 c.c., made up of five separate quantities, each of 0·5 c.c. Each tube contained 0·5 c.c. of diluted bacterial extract (antigen), 0·5 c.c. of diluted serum (antibody), and 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum (complement). After one hour's incubation at 37° C., there were added to each tube 0·5 c.c. of the previously determined dilution of hæmolytic serum, and 0·5 c.c. of a 1 in 20 suspension of washed sheep corpuscles. The tubes were then incubated for a period varying between forty-five minutes and two hours. The series of tubes was removed from the incubator, when the controls showed complete hæmolysis.

The strains used in this series of experiments were as follows: Three strains of *Bacillus typhosus* isolated by Dr. Ledingham from typhoid carriers, and designated Math., T., and Mie. Four strains of *Bacillus paratyphosus* B, designated Schott., MacW., Ma, and P.9. One strain of *Bacillus paratyphosus* A, one strain of *Bacillus enteritidis*, Gaertner, three strains from cases of food-poisoning in men, designated Düsseldorf, H.A., and A.3, one strain isolated from food and one strain isolated from a healthy pig.

All the strains except the typhoid and the Gaertner strains were given to me by Dr. Bainbridge. The strains were carefully tested before the commencement and at frequent intervals during the course of the experiments. In all cases the typical sugar reactions were obtained.

ANALYSIS OF RESULTS.

The results of the experiments are recorded in the annexed tables :—

Table I: In the experiment illustrated in Table I an antityphoid serum has been titrated with an extract made from another strain of *Bacillus typhosus*. It will be seen that this serum acts best in a dilution of 1 in 20 or 1 in 40, that in the dilutions of 1 in 80 and in 1 in 160 the reaction is less but still very marked. A very fair result is obtained in a dilution of 1 in 320, while in a dilution of 1 in 640 the fixation of complement is hardly appreciable. The effect of excess of antibody may be seen by comparing the results in the first three rows. It will be seen that a dilution of 1 in 20 is more effective than a dilution of 1 in 10 and that a dilution of 1 in 10 is more effective than a dilution of 1 in 5. This effect is evidenced in the tubes 5, 6 and 7 of the first three rows. In tube 7, row A, hæmolysis was almost complete; in tube 7, row C, there was only the slightest trace of hæmolysis. This result indicates that when the extract is diluted 1 in 320 and the serum 1 in 5, an excess of antibody is present which prevents the fixation of complement. In tube 7 of row C the same amount of antigen is present, but only one-quarter of the amount of antibody. In this tube the complement was almost completely fixed. The effect of excess of antigen is more difficult to demonstrate because it is a difficult matter to prepare extracts rich in antigen and because large doses of bacterial extract have a great tendency to spontaneous absorption of complement. A slight indication of this effect can, however, be made out in row G, tubes 1 and 2. In these tubes the amounts of extract present were 0·1 c.c. and 0·05 c.c. respectively. These amounts appear to represent a slight antigen excess, for a trace of hæmolysis was noticed. When the amount of extract was reduced (tubes 4, 5 and 6) no hæmolysis occurred. The amount of serum

was the same in all tubes of row G. From this experiment it is evident that a quantitative relationship exists between antigen and antibody and that excess of either constituent is unfavourable to the fixation of complement. In working with bacterial extracts the inhibitory influence of excess of antigen is of theoretical rather than of practical interest, for

TABLE I.—TITRATION OF ANTI-

Serum prepared by injecting a rabbit intravenously with *Bacillus typhosus*

	Dilutions of typhoid extract "Math." 0·5 c.c.	DILUTIONS OF ANTITYPHOID			
		A 0·5 c.c. of 1 in 5	B 0·5 c.c. of 1 in 10	C 0·5 c.c. of 1 in 20	D 0·5 c.c. of 1 in 40
1	1 in 5	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis
2	1 in 10	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,
5	1 in 80	Trace hæmolysis	,, ,,	,, ,,	,, ,,
6	1 in 160	Marked hæmolysis	Trace hæmolysis	,, ,,	,, ,,
7	1 in 320	Almost complete hæmolysis	Marked hæmolysis	Trace hæmolysis	Trace hæmolysis
8	1 in 640	Complete hæmolysis	Almost complete hæmolysis	Marked hæmolysis	Marked hæmolysis
9	1 in 1,280	,, ,,	Complete hæmolysis	Almost complete hæmolysis	Complete hæmolysis
10	1 in 2,560	,, ,,	,, ,,	Complete hæmolysis	,, ,,
11	0·5 c.c. normal saline serum control	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis

Nine parallel rows, each consisting of eleven tubes, were set up. In the tubes numbered 1 to 10, in all nine rows, were put successive dilutions of extract. The bulk of extract dilution was 0·5 c.c. in each tube. The tubes numbered 1 in all nine rows contained the same amount of extract, namely, 0·1 c.c. of extract diluted to 0·5 c.c. with normal saline. The tubes numbered 2 in all rows contained half as much extract diluted to the same bulk, that is to say, 0·05 extract diluted with normal saline to 0·5 c.c. and so on. Half a cubic centimetre of normal saline was then added to all tubes in the ninth row, and to the tubes it is a difficult matter to make an extract sufficiently rich in antigen. The possibility of a relative excess of antiserum is a matter of more practical importance. For instance, if the object of the experiment be to detect a very small quantity of antigen, the optimal dilution of the antiserum must be determined by a preliminary test of the antiserum

SERUM WITH HOMOLOGOUS EXTRACT.

(Strain T.). The typhoid extract was prepared from Strain M.

HEATED RABBIT SERUM (STRAIN T.)				I 0·5 c.c. normal saline (extract control)
E 0·5 c.c. of 1 in 80	F 0·5 c.c. of 1 in 160	G 0·5 c.c. of 1 in 320	H 0·5 c.c. of 1 in 640	
No hæmolysis	No hæmolysis	Trace hæmolysis	Slight hæmolysis	Complete hæmolysis
,, ,,	,, ,,	,, ,,	Marked hæmolysis	,, ,,
,, ,,	,, ,,	No hæmolysis	,, ,,	,, ,,
,, ,,	,, ,,	,, ,,	Almost complete hæmolysis	,, ,,
,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
,, ,,	Slight hæmolysis	Half hæmolysed	Complete hæmolysis	,, ,,
Slight hæmolysis	Marked hæmolysis	Marked hæmolysis	,, ,,	,, ,,
Marked hæmolysis	Almost complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,
Complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,	,, ,,
,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	

numbered 11 in all the other rows. The tubes in the ninth row (I) constituted the extract controls. The tubes numbered 11 constituted serum controls. The tubes in rows A to H received each half a cubic centimetre of the serum dilution mentioned at the top of each column. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. The experiment was then incubated for one hour, and then 0·5 c.c. of a 1 in 20 suspension of sheep corpuscles and 0·5 of a 1 in 600 dilution of hæmolysin were added.

with the homologous extract. Unless this is done the antiserum will not be given the best chance of detecting a trace of antigen. Such a preliminary titration also serves the more obvious purpose of ascertaining how far the particular serum may be diluted. For the differentiation of two nearly allied organisms, the highest dilution should be chosen which gives a well-marked reaction with the homologous extract. A knowledge of the antibody content of a serum is best obtained by titrating the serum with the homologous extract in such a way that each dilution of serum is allowed to interact with each dilution of extract. It is always advisable to carry out a preliminary test of this nature with every serum, as the

Table II.—Serum Antityphoid (Strain T.). Extracts of Typhoid (Strain Math.

		Typhoid extract (strain Math.)			Typhoid extract (strain "T.")			Bacillus Aertryck extract (strain AE)		
	Extract dilutions 0·5 c.c. of	+ 0·5 c.c. antityphoid serum 1 in 10	+ 0·5 c.c. antityphoid serum 1 in 100	+ 0·5 c.c. normal saline	+ 0·5 c.c. antityphoid serum 1 in 10	+ 0·5 c.c. antityphoid serum 1 in 100	+ 0·5 c.c. normal saline	+ 0·5 c.c. antityphoid serum 1 in 10	+ 0·5 c.c. antityphoid serum 1 in 100	+ 0·5 c.c. normal saline solution
1	1 in 5	No hæmolysis	No hæmolysis	Complete hæmolysis	No hæmolysis	No hæmolysis	Complete hæmolysis	No hæmolysis	Complete hæmolysis	Complete hæmolysis
2	1 in 10	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
5	1 in 80	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
6	1 in 160	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
7	1 in 320	Trace hæmolysis	,, ,,	,, ,,	Trace hæmolysis	,, ,,	,, ,,	Trace hæmolysis	,, ,,	,, ,,
8	1 in 640	Marked hæmolysis	Trace hæmolysis	,, ,,	Complete hæmolysis	Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
9	1 in 1280	Complete hæmolysis	Marked hæmolysis	,, ,,	,, ,,	Almost complete hæmolysis	,, ,,	Almost complete hæmolysis	,, ,,	,, ,,

0·1 c.c., 0·05 c.c., 0·01 c.c. and 0·005 c.c. of typhoid antiserum were put up without extract as serum controls; complete hæmolysis took place in every tube.

From each extract were made three parallel sets of dilutions. To the first row was added antityphoid serum 1 in 10, to the second row antityphoid serum 1 in 100, to the third row knowledge so gained is of use in determining the dose of antiserum appropriate for use in subsequent experiments.

Table II: This table illustrates the reaction between an antityphoid serum and extracts prepared from strains of *Bacillus typhosus*, *Bacillus paratyphosus*, and a strain of *Bacillus Aertryck*. With each extract three sets of dilutions were made. To one set no serum was added, and this set served the purpose of an extract control. The second set received antityphoid serum in a dilution of 1 in 100, while the third set received antityphoid serum in a dilution of 1 in 10. An examination of the table shows that with a serum dilution of 1 in 10 no differentiation

AND T.), OF PARATYPHOID A AND OF PARATYPHOID B (STRAINS SCHOTT. AND MACW.).

EXTRACT OF PARATYPHOID A			EXTRACT OF PARATYPHOID B (STRAIN SCHOTT.)			EXTRACT OF PARATYPHOID B (STRAIN MACW.)		
+ 0·5 c.c. antityphoid serum 1 in 10	+ 0·5 c.c. antityphoid serum 1 in 100	+ 0·5 c.c. normal saline	+ 0·5 c.c. antityphoid serum 1 in 10	+ 0·5 c.c. antityphoid serum 1 in 100	+ 0·5 c.c. normal saline	+ 0·5 c.c. antityphoid serum 1 in 10	+ 0·5 c.c. antityphoid serum 1 in 100	+ 0·5 c.c. normal saline
No hæmolysis	Almost complete hæmolysis	Almost complete hæmolysis	No hæmolysis	Complete hæmolysis	Complete hæmolysis	No hæmolysis	Almost complete hæmolysis	Almost complete hæmolysis
,, ,,	,, ,,	Complete hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,	Complete hæmolysis	Complete hæmolysis
,, ,,	Complete hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	Trace hæmolysis	,, ,,	,, ,,
Almost complete hæmolysis	,, ,,	,, ,,	Almost complete hæmolysis	,, ,,	,, ,,	Half hæmolysed	,, ,,	,, ,,
Almost complete hæmolysis	,, ,,	,, ,,	Complete hæmolysis	,, ,,	,, ,,	Complete hæmolysis	,, ,,	,, ,,

normal saline solution. The third row constituted the extract control. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. The experiment was then incubated for one hour, and then there were added 0·5 c.c. of a 1 in 20 suspension of washed sheep corpuscles and a 1 in 500 dilution of hæmolytic serum.

is possible between *Bacillus typhosus*, *Bacillus paratyphosus* A, *Bacillus paratyphosus* B, and *Bacillus Aertryck*. When, however, a 1 in 100 dilution of the serum is employed the typhoid extract is completely differentiated from the other extracts.

Table III: This table illustrates the titration of a serum derived from a rabbit, which had been injected with paratyphoid A bacilli. A comparison of row A with row B shows the influence of excess of antibody in diminishing the reaction.

TABLE III.—ANTISERUM DERIVED FROM RABBIT AFTER INTRAVENOUS INJECTION OF PARATYPHOID A.

Extract prepared from the same strain of Paratyphoid A.

	Paratyphoid A extract, 0·5 c.c. of a dilution	A + 0·5 c.c. of paratyphoid A antiserum dilution 1 in 5	B + 0·5 c.c. of paratyphoid A antiserum dilution 1 in 20	C + 0·5 c.c. of 1 in 80 dilution of antiserum	D + 0·5 c.c. of 1 in 320 dilution of antiserum	E + 0·5 c.c. of 1 in 5 dilution of normal serum	F + 0·5 c.c. normal saline
1	1 in 20	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis	Complete hæmolysis	Complete hæmolysis
2	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 80	Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 160	Complete hæmolysis	Trace hæmolysis	Trace hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,
5	1 in 320	,, ,,	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,

As a further control, 2 in 5, 1 in 5, and 1 in 10 dilutions of extract were prepared. No antiserum was added to these tubes, but the bulk was made up with 0·5 c.c. normal saline solution. Dilution 2 in 5 showed marked hæmolysis, dilutions 1 in 5 and 1 in 10 showed complete hæmolysis. The serum controls of tubes containing single and double the amounts of serum used in the corresponding columns of the experiment, but without the addition of bacillary extract. All the serum controls showed complete hæmolysis.

In this experiment six identical and parallel rows were prepared so that the tubes of the same number in each row contained the same dilution of extract. For instance, tube 1 in every row contained 0·025 extract diluted to 0·5 c.c. with saline solution. Every tube of row A then received 0·5 c.c. of a 1 in 5 dilution of antiserum. To every tube of row B was added 0·5 c.c. of a 1 in 20 dilution, to every tube of row C 0·5 c.c. of a 1 in 80 dilution, and to every tube of row D 0·5 c.c. of a 1 in 320 dilution of antiserum. Each of the tubes of row E received 0·5 c.c. of a 1 in 5 dilution of normal serum, and in row F each tube received 0·5 c.c. of normal saline solution. All tubes then received 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum, and the experiment was then incubated for one hour. At the expiration of the hour there were added 0·5 c.c. of a 1 in 20 suspension of washed sheep corpuscles and 0·5 c.c. of a 1 in 1,000 dilution of hæmolytic serum.

Table IV: This table shows the group reaction obtained by the use of a large amount of serum (dilution 1 in 20) and the complete differentiation obtained by the use of a small amount of serum (dilution 1 in 200).

Table V: This table illustrates the titration of a serum obtained from a rabbit, which had been injected with *Bacillus paratyphosus* B, with the homologous extract.

Table VI: Extracts prepared from *Bacillus typhosus*, *Bacillus paratyphosus* A, *Bacillus paratyphosus* B, and *Bacillus Aertryck* are titrated with paratyphoid B serum in four separate dilutions. With a dilution of 1 in 10 no differentiation is possible; with a dilution of 1 in 50 a great difference is perceptible between the extracts of *Bacillus paratyphosus* B and *Bacillus Aertryck* as compared with the extracts of *Bacillus typhosus* and *Bacillus paratyphosus* A. When the serum is diluted 1 in 100 the reaction with *Bacillus typhosus* and *Bacillus paratyphosus* A almost disappears, but, with this dilution of serum *Bacillus paratyphosus* B cannot be differentiated from *Bacillus Aertryck*. With a dilution of 1 in 200, however, *Bacillus paratyphosus* B is satisfactorily differentiated from *Bacillus Aertryck*, as well as from *Bacillus typhosus* and *Bacillus paratyphosus* B.

Table VII: It is here shown that by the use of a 1 in 300 dilution of the paratyphoid A serum two strains of *Bacillus paratyphosus* B are differentiated from strains of *Bacillus typhosus*, *Bacillus paratyphosus* A, *Bacillus Aertryck*, and *Bacillus enteritidis*, Gaertner.

Table VIII: The extracts employed in this experiment were prepared from strains of the Paratyphoid-Aertryck group, isolated from various sources: Düsseldorf—a strain isolated from a case of food-poisoning in man. B.A.—isolated from food. Heyd. Ali.—a strain isolated from man (nature of illness unknown). M.A.—strain isolated from healthy man. P.R. 9—strain isolated from paratyphoid carrier. 840—Strain isolated from healthy pig. The sera used in this experiment (Table VIII) were the paratyphoid B serum used in the experiments of Tables V, VI, and VII, and a serum (designated O'B.) obtained from a rabbit injected with a strain derived from a guinea-pig. This strain, which was isolated from a case of epizootic disease among guinea-pigs was identical in sugar reactions and agglutination tests with *Bacillus Aertryck*. For all these strains and for the serum O'B. I am indebted to the kindness of Dr. Bainbridge. Table VIII shows that the strains M.A. and P.R. 9 are to be regarded as paratyphoid B. The other four strains, namely, Düsseldorf, B.A., Heyd. Ali., and 840, react

TABLE IV.

The serum used in this experiment was obtained from a rabbit after injection with paratyphoid A. The extracts were prepared from the same strain of paratyphoid A, from two strains of typhoid (Math. and T.), and from two strains of paratyphoid B (Schott. and MacW.).

	0·5 c.c. of extract dilutions	Typhoid extract (strain Math.) + 0·5 c.c. normal saline		Typhoid extract (strain T.) + 0·5 c.c. normal saline		Paratyphoid A extract + 0·5 c.c. normal saline		Paratyphoid B extract (strain Schott.) + 0·5 c.c. normal saline		Paratyphoid B extract (strain MacW.) + 0·5 c.c. normal saline	
1	2 in 5	Marked hæmolysis ..		Half hæmolysed ...		Half hæmolysed... ...		Marked hæmolysis ...		Half hæmolysed	
2	1 in 5	Complete hæmolysis ...		Complete hæmolysis ...		Almost complete hæmolysis		Complete hæmolysis ...		Complete hæmolysis	
3	1 in 10	,, ,, ...		,, ,, ...		Complete hæmolysis ...		,, ,, ...		,, ,,	
.		Paratyphoid A serum		Paratyphoid A serum		Paratyphoid A serum		Paratyphoid A serum		Paratyphoid A serum	
		+ 0·5 c.c. of dilution 1 in 20	+ 0·5 c.c. of dilution 1 in 200	+ 0·5 c.c. of dilution 1 in 20	+ 0·5 c.c. of dilution 1 in 200	+ 0·5 c.c. of dilution 1 in 20	+ 0·5 c.c. of dilution 1 in 200	+ 0·5 c.c. of dilution 1 in 20	+ 0·5 c.c. of dilution 1 in 200	+ 0·5 c.c. of dilution 1 in 20	+ 0·5 c.c. of dilution 1 in 200
4	1 in 5	No hæmolysis	Complete hæmolysis	No hæmolysis	Complete hæmolysis	No hæmolysis	Trace hæmolysis	No hæmolysis	Complete hæmolysis	No hæmolysis	Marked hæmolysis
5	1 in 10	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	No hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
6	1 in 20	,, ,,	,, ,,	Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
7	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	Trace hæmolysis	,, ,,	,, ,,	,, ,,
8	1 in 80	Marked hæmolysis	,, ,,	Almost complete hæmolysis	,, ,,	Half hæmolysed	Trace hæmolysis	,, ,,	,, ,,	Trace hæmolysis	Almost complete hæmolysis
9	1 in 160	Almost complete hæmolysis	,, ,,	Complete hæmolysis	,, ,,	Marked hæmolysis	Half hæmolysed	Complete hæmolysis	,, ,,	Almost complete hæmolysis	Complete hæmolysis
10	1 in 320	Complete hæmolysis	,, ,,	,, ,,	,, ,,	Complete hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,

Serum control tubes containing 0·5, 0·025 and 0·005 c.c. of serum, without extract, showed complete hæmolysis. Tubes 1, 2 and 3 in each row were extract controls. The series of dilutions 4 to 10 were made in duplicate for each extract. To each tube of the first row was added 0·5 c.c. of a 1 in 20 dilution of the serum of a rabbit which had been immunized with paratyphoid A. To each tube of the second row was added 0·5 c.c. of a 1 in 200 dilution of the same serum. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. After one hour's incubation every tube received 0·5 c.c. of a 1 in 20 suspension of sheep blood corpuscles and 0·5 c.c. of 1 in 500 dilution of hæmolytic serum.

TABLE V.—TITRATION OF SERUM FROM RABBIT INJECTED WITH PARATYPHOID B (SCHOTT.) WITH THE HOMOLOGOUS EXTRACT.

	0·5 c.c. of dilutions of paratyphoid B extract	A + 0·5 c.c. of a 1 in 5 dilution of paratyphoid B serum	B + 0·5 c.c. of a 1 in 100 dilution of paratyphoid B serum	C + 0·5 c.c. of a 1 in 200 dilution of paratyphoid B serum	D + 0·5 c.c. of a 1 in 400 dilution of paratyphoid B serum	E + 0·5 c.c. of a 1 in 800 dilution of paratyphoid B serum	F + 0·5 c.c. of a 1 in 1600 dilution of paratyphoid B serum	G + 0·5 c.c. of a 1 in 5 dilution of normal rabbit serum	H + 0·5 c.c. of normal saline solution
1	2 in 5	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis	Half hæmolysed	Half hæmolysed
2	1 in 5	,, ,,	,, ,,	,, ,,	,, ,,	Trace hæmolysis	Trace hæmolysis	,, ,,	Complete hæmolysis
3	1 in 10	,, ,,	,, ,,	,, ,,	,, ,,	No hæmolysis	,, ,,	Marked hæmolysis	—
4	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	No hæmolysis	Almost complete hæmolysis	—
5	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	Trace hæmolysis	Trace hæmolysis	Complete hæmolysis	—
6	1 in 80	,, ,,	,, ,,	,, ,,	Trace hæmolysis	Half hæmolysed	Almost complete hæmolysis	,, ,,	—
7	1 in 160	Trace hæmolysis	Trace hæmolysis	Trace hæmolysis	Half hæmolysed	Almost complete hæmolysis	Complete hæmolysis	,, ,,	—
8	1 in 320	Complete hæmolysis	Half hæmolysed	Half hæmolysed	Almost complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,	—
9	1 in 640	,, ,,	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	,, ,,	—	,, ,,	—

Serum controls, 0·2 c.c. and 0·1 c.c., of paratyphoid B serum showed complete hæmolysis. While 0·1 c.c. of normal serum showed complete hæmolysis, a little complement was absorbed in the control tube containing 0·2 c.c. normal serum. This tendency of the normal serum to absorb complement accounts for the readings of tubes 2 to 4 in row G.

In this experiment eight identical series of dilutions were made. To each row was added a different dilution of the homologous serum. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. After the completion of one hour in the incubator, 0·5 c.c. of a 1 in 20 suspension of sheep corpuscles and 0·5 c.c. of a 1 in 600 dilution of hæmolysin were added to every tube.

TABLE VI.

	0·5 c.c. of extract dilutions	Typhoid extract (strain Math.) + 0·5 c.c. of a 1 in 10 dilution of paratyphoid B serum	Paratyphoid A extract + 0·5 c.c. of a 1 in 10 dilution of paratyphoid B serum	Paratyphoid B extract (strain Schott.) + 0·5 c.c. of a 1 in 10 dilution of paratyphoid B serum	Aertryck extract + 0·5 c.c. of a 1 in 10 dilution of paratyphoid B serum
1	1 in 10	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis
2	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 80	,, ,,	,, ,,	,, ,,	,, ,,
5	1 in 160	Trace hæmolysis	,, ,,	Trace hæmolysis	Trace hæmolysis
6	1 in 320	Almost complete hæmolysis	Trace hæmolysis	Half hæmolysed	Half hæmolysed
7	1 in 640	Complete hæmolysis	Marked hæmolysis	Almost complete hæmolysis	Complete hæmolysis
8	1 in 1280	,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,

		Typhoid extract + 0·5 c.c. of a 1 in 50 dilution of paratyphoid B serum	Paratyphoid A extract + 0·5 c.c. of a 1 in 50 dilution of paratyphoid B serum	Paratyphoid B extract + 0·5 c.c. of a 1 in 50 dilution of paratyphoid B serum	Aertryck extract + 0·5 c.c. of a 1 in 50 dilution of paratyphoid B serum
1	1 in 10	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis
2	1 in 20	Trace hæmolysis	,, ,,	,, ,,	,, ,,
3	1 in 40	Half hæmolysed	Trace hæmolysis	,, ,,	,, ,,
4	1 in 80	Marked hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,
5	1 in 160	Complete hæmolysis	,, ,,	,, ,,	,, ,,
6	1 in 320	,, ,,	Complete hæmolysis	Trace hæmolysis	Trace hæmolysis
7	1 in 640	,, ,,	,, ,,	,, ,,	Almost complete hæmolysis
8	1 in 1280	,, ,,	,, ,,	Complete hæmolysis	Complete hæmolysis

TABLE VI—(*continued*).

	0·5 c.c. of extract dilutions	Typhoid extract + dilution of 1 in 100 paratyphoid B serum	Paratyphoid A extract + 0·5 c.c. of a 1 in 100 paratyphoid B serum	Paratyphoid B extract + 1 in 100 paratyphoid B serum	Aertryck extract + 0·5 c.c. of a 1 in 100 dilution of paratyphoid B serum
1	1 in 10	No hæmolysis	No hæmolysis	No hæmolysis	No hæmolysis
2	1 in 20	Almost complete hæmolysis	Half hæmolysed	,, ,,	,, ,,
3	1 in 40	Complete hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,
4	1 in 80	,, ,,	Complete hæmolysis	,, ,,	,, ,,
5	1 in 160	,, ,,	,, ,,	,, ,,	Trace hæmolysis
6	1 in 320	,, ,,	,, ,,	Trace hæmolysis	Almost complete hæmolysis
7	1 in 640	,, ,,	,, ,,	Half hæmolysed	Complete hæmolysis
8	1 in 1280	,, ,,	,, ,,	Complete hæmolysis	,, ,,

		Typhoid extract + 0·5 c.c. of a 1 in 200 dilution of paratyphoid B serum	Paratyphoid A extract + 0·5 c.c. of a 1 in 200 dilution of paratyphoid B serum	Paratyphoid B extract + 0·5 c.c. of a 1 in 200 dilution of paratyphoid B serum	Aertryck extract + 0·5 c.c. of a 1 in 200 dilution of paratyphoid B serum
1	1 in 10	Marked hæmolysis	Almost complete hæmolysis	No hæmolysis	Trace hæmolysis
2	1 in 20	Complete hæmolysis	Complete hæmolysis	,, ,,	Marked hæmolysis
3	1 in 40	,, ,,	,, ,,	,, ,,	Complete hæmolysis
4	1 in 80	,, ,,	,, ,,	,, ,,	,, ,,
5	1 in 160	,, ,,	,, ,,	Trace hæmolysis	,, ,,
6	1 in 320	,, ,,	,, ,,	Almost complete hæmolysis	,, ,,
7	1 in 640	,, ,,	,, ,,	,, ,,	,, ,,
8	1 in 1280	,, ,,	,, ,,	Complete hæmolysis	,, ,,

Extract controls, without serum, in dilutions of 1 in 5, 1 in 10, and 1 in 20, and serum controls in dilutions of 1 in 5, 1 in 10 and 1 in 20 were put up. All six control tubes showed complete hæmolysis.

With each extract four identical series of dilutions were made. From the paratyphoid B antiserum four dilutions were made—1 in 10, 1 in 50, 1 in 100, 1 in 200. All four extracts were tested in a series of dilutions with each of the four serum dilutions. 0·5 c.c. of fresh guinea-pig serum 1 in 10 was added to each tube. After one hour's incubation each tube received 0·5 c.c. of a 1 in 20 suspension of sheep corpuscles and 0·5 c.c. of a 1 in 500 dilution of hæmolysin.

with the O'B. serum, but not with the paratyphoid B serum. From a consideration of Tables VI, VII, and VIII it appears that the complement-fixation method allows of a differentiation between *Bacillus paratyphosus* B and *Bacillus Aertryck*. This conclusion is in agreement with that which has been reached by Bainbridge (1911) who employed the absorption method of Castellani.

Table VII.

To all tubes 0·5 c.c. of a 1 in 300 dilution of paratyphoid B serum obtained from a rabbit immunized with paratyphoid B (strain Schott.).

	0·5 c.c. extract dilutions	Paratyphoid A extract	Paratyphoid B extract (strain Schott.)	Paratyphoid B extract (strain MacW.)	Aertryck extract (strain AE)	Gaertner extract	Typhoid extract (strain Math.)
1	1 in 20	Complete hæmolysis	No hæmolysis	No hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis
2	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 80	,, ,,	Slight hæmolysis	Slight hæmolysis	,, ,,	,, ,,	,, ,,
4	1 in 160	,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,	,, ,,

Extract dilutions of 1 in 5, 1 in 10, and 1 in 20 were put up as extract controls for each of the six extracts employed. Serum controls, dilutions 1 in 100 and 1 in 300, were also put up. Complete hæmolysis was obtained in all the control tubes.

In this experiment each extract was put up in four dilutions. To every tube, except the controls, was added 0·5 c.c. of a 1 in 300 dilution of paratyphoid B serum. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. After one hour's incubation each tube received 0·5 c.c. of a 1 in 20 suspension of sheep's corpuscles and 0·5 c.c. of a 1 in 1,000 dilution of hæmolysin.

General Discussion of Results.

The Tables I to VIII shows very clearly that the complement-fixation method affords an extremely delicate and specific means of separating and identifying closely allied races of bacteria. Absolute differentiation can, however, only be secured by the careful determination of the dosage of antigen and antibody. The dilution of antiserum necessary for a differentiation experiment can be ascertained by the titration of the antiserum with the homologous extract. As a rule satisfactory differentiation can be obtained with the greatest dilution of antiserum

TABLE VIII.

	0·5 c.c. extract dilutions	Extract of strain "Düsseldorf"	Extract of strain B.A.	Extract of strain Heyd. Ali.	Extract of strain M.A.	Extract of strain 840	Extract of strain P.R.9
		+ 0·5 c.c. of dilution 1 in 50 of serum (strain O'B.)					
1	1 in 5	No hæmolysis	Trace hæmolysis	Trace hæmolysis	Almost complete hæmolysis	No hæmolysis	Complete hæmolysis
2	1 in 10	Trace hæmolysis	,, ,,	,, ,,	Complete hæmolysis	,, ,,	,, ,,
3	1 in 20	Half hæmolysed	Half hæmolysis	Almost complete hæmolysis	,, ,,	Trace hæmolysis	,, ,,
4	1 in 40	Almost complete hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,	Almost complete hæmolysis	,, ,,
		+ 0·5 c.c. of a 1 in 200 dilution of paratyphoid B serum					
1	1 in 5	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	No hæmolysis	Complete hæmolysis	Trace hæmolysis
2	1 in 10	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	Half hæmolysed	,, ,,	Almost complete hæmolysis
	1 in 40	,, ,,	,, ,,	,, ,,	Complete hæmolysis	,, ,,	Complete hæmolysis
		+ 0·5 c.c. normal saline solution (extract control)					
1	1 in 5	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Half hæmolysed	Complete hæmolysis	Complete hæmolysis
2	1 in 10	,, ,,	,, ,,	,, ,,	Complete hæmolysis	,, ,,	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,

In this experiment six extracts made from six separate strains were used. Each extract was put up in four dilutions. Each extract was tested with each of the two sera. Strains M.A. and P.R.9 reacted with the paratyphoid serum, but not with the serum prepared with strain O'B. The other four strains reacted with the O'B. serum. Serum control tubes containing single and double doses of serum without extract showed complete hæmolysis. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum, and after one hour in the incubator 0·5 c.c. of a 1 in 20 suspension of sheep corpuscles and 0·5 c.c. of a 1 in 500 dilution of hæmolysin.

which is found to give a thoroughly satisfactory reaction with the homologous extract.

With low dilutions of antiserum a marked group reaction is obtained. An examination of Table VI will show that the intensity of the group reaction corresponds with the closeness of the relationship between the strains employed. In this experiment the paratyphoid A and the typhoid extracts were easily differentiated from the paratyphoid B extract. It was only when the serum was still further diluted that *Bacillus Aertryck* could be differentiated from *Bacillus paratyphosus* B.

Table IX.

	0·5 c.c. of extract dilutions	Untreated antiparatyphoid (B) serum 1 in 20	Antiparatyphoid B serum 1 in 20 absorbed with typhoid emulsion	Antiparatyphoid B serum 1 in 20 absorbed with an emulsion of paratyphoid A	Antiparatyphoid B serum 1 in 20 absorbed with an emulsion of paratyphoid B	Normal saline
	Typhoid extract					
1	1 in 10	No hæmolysis	Complete hæmolysis	Almost complete hæmolysis	Almost complete hæmolysis	Complete hæmolysis
2	1 in 20	,, ,,	,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,
3	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 80	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
5	1 in 160	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
6	1 in 320	Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
7	1 in 640	Almost complete hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
8	1 in 1280	Complete hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
	Paratyphoid A extract					
1	1 in 10	No hæmolysis	Almost complete hæmolysis	Marked hæmolysis	Almost complete hæmolysis	Complete hæmolysis
2	1 in 20	,, ,,	Complete hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,
3	1 in 40	,, ,,	,, ,,	Complete hæmolysis	,, ,,	,, ,,
4	1 in 80	,, ,,	,, ,,	,, ,,	Complete hæmolysis	,, ,,
5	1 in 160	Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
6	1 in 320	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
7	1 in 640	Complete hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
8	1 in 1280	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,

Table IX (*continued*).

	0·5 c.c. of extract dilutions	Untreated antiparatyphoid (B) serum 1 in 20	Antiparatyphoid B serum 1 in 20 absorbed with typhoid emulsion	Antiparatyphoid B serum 1 in 20 absorbed with an emulsion of paratyphoid A	Antiparatyphoid B serum 1 in 20 absorbed with an emulsion of paratyphoid B	Normal saline
	Paratyphoid B extract					
1	1 in 10	No hæmolysis	No hæmolysis	No hæmolysis	Almost complete hæmolysis	Complete hæmolysis
2	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 40	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 80	,, ,,	,, ,,	,, ,,	Complete hæmolysis	,, ,,
5	1 in 160	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
6	1 in 320	Marked hæmolysis	Trace hæmolysis	Half hæmolysed	,, ,,	,, ,,
7	1 in 640	Complete hæmolysis	Half hæmolysed	Complete hæmolysis	,, ,,	,, ,,
8	1 in 1280	,, ,,	Complete hæmolysis	,, ,,	,, ,,	,, ,,
Serum controls (without extract)						
Single dose ...		0·5 c.c. Complete hæmolysis	0·5 c.c. Complete hæmolysis	0·5 c.c. Complete hæmolysis	0·5 c.c. Complete hæmolysis	—
Double dose...		1 c.c. Complete hæmolysis	1 c.c. Complete hæmolysis	1 c.c. Almost complete hæmolysis	1 c.c. Complete hæmolysis	—

Notes on Table IX.—To each tube in column 1 was added ½ c.c. of a 1 in 20 dilution of an antiparatyphoid B serum. The absorbed serum used in the next column was prepared as follows: Two Roux bottles were inoculated with *Bacillus typhosus* and incubated twenty-four hours. The growth was emulsified in saline and washed and centrifugalized three times. To the washed deposit were added 10 c.c. of saline (previously cooled to 4° C.), and then 10 c.c. of a 1 in 10 dilution of the serum (previously cooled to 4° C.). The mixture was thoroughly shaken and left for half an hour in the cold room. It was then centrifugalized until clear and pipetted off. The serums used in column 3 and column 4 were prepared in the same fashion with emulsions of paratyphoid A and paratyphoid B bacilli.

In the actual experiment each tube contained ½ c.c. of extract dilution, ½ c.c. of serum dilution and ½ c.c. of a 1 in 10 dilution of fresh guinea-pig serum. After incubation for one hour there were added ½ c.c. of hæmolytic serum diluted 1 in 500 and ½ c.c. of a 1 in 20 suspension of sheep corpuscles.

The degree of relationship between the various strains was also evidenced by the time in which hæmolysis occurred in the tubes containing serum and heterologous extract. For example, in the experiment illustrated in Table VII hæmolysis occurred first in the rows of tubes containing the Gaertner and the typhoid extract. The row containing paratyphoid A extract was next hæmolysed, and it was only after a considerable interval that hæmolysis occurred in the row of tubes containing the Aertryck extract. In the experiment figured in Table VI a similar phenomenon was observed; in the tubes containing the serum dilution 1 in 200 the order of onset of hæmolysis was (1) typhoid extract, (2) paratyphoid A extract, (3) Aertryck extract, at a considerably longer interval. The same phenomenon was observed in the experiments figured in Tables II and IV.

When an animal is immunized with a particular strain of organism it may be presumed that a large number of chemical substances capable of acting as antigens are introduced. The majority of these are specific and peculiar to the organism in question; others, it may be assumed, are common to the organism and to other members of the group. The group reaction may be attributed to the presence in the antiserum of a number of antibodies produced by the injection of these group antigens.

COMPLEMENT FIXATION AFTER PRELIMINARY ABSORPTION OF THE GROUP ANTIBODIES.

In Tables IX and X there are illustrated two experiments in which the group antigens were removed by saturating an antiserum with a thick emulsion of a bacillus closely allied to the homologous strain. It will be seen from Table IX that, when a paratyphoid B serum is absorbed with an emulsion of paratyphoid A bacilli, the serum loses its capacity for binding complement in the presence of either a paratyphoid A extract or a typhoid extract. That is to say, the group antibodies are removed. In the same way, absorption with an emulsion of typhoid bacilli removes the group reaction both for typhoid and for paratyphoid A. After absorption with a paratyphoid B emulsion the group antibodies are found to be removed, together with the antibodies specific for paratyphoid B. Table X illustrates a similar experiment with an antityphoid serum.[1]

---

[1] It is important that the bacilli used for absorbing the "group" antibody should be thoroughly washed and that the absorption process should be carried out at a low temperature. Unless these precautions are taken it will be found that the serum possesses the property of "spontaneously" fixing complement. This phenomenon is presumably due to the fact that antigen is dissolved out of the bodies of the bacilli and unites with the antibodies of the serum. The antigen antibody complex thus formed renders the serum quite unfitted for subsequent use.

TABLE X.

		+ 0·5 c.c. of 1 in 5 dilution of typhoid serum untreated	+ 0·5 c.c. of 1 in 5 dilution of typhoid serum previously absorbed with paratyphoid B	+ 0·5 c.c. of 1 in 5 dilution of typhoid serum previously absorbed with typhoid	+ 0·5 c.c. of 1 in 5 dilution of normal serum
	Dilutions of typhoid extract M				
1	1 in 5	No hæmolysis	No hæmolysis	Almost complete hæmolysis	Complete hæmolysis
2	1 in 10	,, ,,	,, ,,	Complete hæmolysis	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 40	,, ,,	Trace hæmolysis	,, ,,	,, ,,
5	1 in 80	Trace hæmolysis	Almost complete hæmolysis	,, ,,	,, ,,
6	1 in 160	Almost complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,
7	1 in 320	Complete hæmolysis	,, ,,	,, ,,	,, ,,
8	1 in 640	,, ,,	,, ,,	,, ,,	,, ,,
9	1 in 1280	,, ,,	,, ,,	,, ,,	,, ,,
	Paratyphoid A extract				
	1 in 5	No hæmolysis	Almost complete hæmolysis	Almost complete hæmolysis	Complete hæmolysis
2	1 in 10	,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 40	Trace hæmolysis	,, ,,	,, ,,	,, ,,
5	1 in 80	Almost complete hæmolysis	,, ,,	,, ,,	,, ,,
6	1 in 160	Complete hæmolysis	,, ,,	,, ,,	,, ,,
7	1 in 320	,, ,,	,, ,,	,, ,,	,, ,,
8	1 in 640	,, ,,	,, ,,	,, ,,	,, ,,
9	1 in 1280	,, ,,	,, ,,	,, ,,	,, ,,
	Paratyphoid B extract				
1	1 in 5	No hæmolysis	Almost complete hæmolysis	Almost complete hæmolysis	Complete hæmolysis
2	1 in 10	,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,
3	1 in 20	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 40	Trace hæmolysis	,, ,,	,, ,,	,, ,,
5	1 in 80	Almost complete hæmolysis	,, ,,	,, ,,	,, ,,
6	1 in 160	Complete hæmolysis	,, ,,	,, ,,	,, ,,
7	1 in 320	,, ,,	,, ,,	,, ,,	,, ,,
8	1 in 640	,, ,,	,, ,,	,, ,,	,, ,,
9	1 in 1280	,, ,,	,, ,,	,, ,,	,, ,,
	Serum alone				
	2 in 5	Complete hæmolysis	Almost complete hæmolysis	Almost complete hæmolysis	Complete hæmolysis
	1 in 5	,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,

To each tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. The experiment was incubated for an hour, and there were subsequently added 0·5 c.c. of a 1 in 20 suspension of sheep corpuscles and 0·5 c.c. of a 1 in 400 dilution of hæmolysin.

TABLE XI.—TITRATION OF ANTITYPHOID SERUM (STRAIN T.) WITH TYPHOID EXTRACT (STRAIN MATH.).

	0·5 c.c. of dilution of typhoid extract (strain Math.)	0·5 C.C. OF DILUTIONS OF TYPHOID SERUM (STRAIN T.)					F + 0·5 c.c. normal saline
		A 4 in 5	B 2 in 5	C 1 in 40	D 1 in 160	E 1 in 320	
1	4 in 5	No hæmolysis	No hæmolysis	Trace hæmolysis	Half hæmolysed	Half hæmolysed	Half hæmolysed
2	2 in 5	,, ,,	,, ,,	No hæmolysis	,, ,,	Almost complete hæmolysis	Complete hæmolysis
3	1 in 5	,, ,,	,, ,,	,, ,,	Trace hæmolysis	Half hæmolysed	,, ,,
4	1 in 10	Trace hæmolysis	,, ,,	,, ,,	No hæmolysis	Trace hæmolysis	,, ,,
5	1 in 20	Slight hæmolysis	Trace hæmolysis	,, ,,	,, ,,	,, ,,	,, ,,
6	1 in 40	Almost complete hæmolysis	Slight hæmolysis	,, ,,	Trace hæmolysis	Marked hæmolysis	,, ,,
7	1 in 80	Complete hæmolysis	Half hæmolysed	Trace hæmolysis	,, ,,	Almost complete hæmolysis	,, ,,
8	1 in 160	,, ,,	Complete hæmolysis	Slight hæmolysis	Half hæmolysed	,, ,,	,, ,,
9	1 in 320	,, ,,	,, ,,	Almost complete hæmolysis	Almost complete hæmolysis	Complete hæmolysis	,, ,,
10	1 in 640		,, ,,	Complete hæmolysis	Complete hæmolysis	,, ,,	,, ,,
		SERUM CONTROLS					
11	Normal saline, 0·5 c.c.	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	—

A, B, C, D, E and F represent six parallel rows of test-tubes. The tubes bearing the same number in each row contained identical extract dilutions made up to a bulk of 0·5 c.c. To each tube of row A was then added 0·5 c.c. of a 4 in 5 dilution of antiserum. To each tube of row B was added 0·5 c.c. of a 2 in 5 dilution of antiserum, and so on. The tubes in row F contain extract only and acted as extract controls. The tubes numbered 11 in the rows A to F were serum controls. To every tube was added 0·5 c.c. of a 1 in 10 dilution of fresh guinea-pig serum. After incubation for one hour every tube received 0·5 c.c. of a 1 in 20 suspension of washed sheep corpuscles and 0·5 c.c. of a 1 in 500 of hæmolytic serum.

TABLE XII.

Serum dilutions		Typhoid serum		Paratyphoid A serum		Paratyphoid B serum	
		A	B	A	B	A	B
1	4 in 5	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis	Complete hæmolysis
2	2 in 5	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
3	1 in 5	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,	,, ,,
4	1 in 10	,, ,,	,, ,,	,, ,,	Marked hæmolysis	,, ,,	,, ,,
5	1 in 20	,, ,,	Slight hæmolysis	,, ,,	No hæmolysis	,, ,,	Trace hæmolysis
6	1 in 40	,, ,,	No hæmolysis	,, ,,	,, ,,	,, ,,	No hæmolysis

For each serum two duplicate rows of dilutions were prepared, so that each tube contained the diluted serum in a bulk of ½ c.c. To all tubes in the rows marked A was added ½ c.c. of normal saline solution. To all tubes in rows B was added 1 c.c. of normal saline. To the tubes in rows A was added ½ c.c. of a 1 in 500 dilution of a hæmolytic serum. This represented three minimal lytic doses of the particular hæmolytic serum. All the tubes then received ½ c.c. of a 1 in 20 suspension of sheep corpuscles and ½ c.c. of a 1 in 10 dilution of fresh guinea-pig serum. The experiment was incubated for two hours, and the results were recorded as above. It will be seen by an examination of the rows marked B that the hæmolytic titre of the typhoid and of the paratyphoid B antiserum was 1 in 10, while the paratyphoid A antiserum had a hæmolytic titre of 1 in 5.

The rows marked A contained the indicated doses of the antiserum together with three lytic doses of a hæmolytic serum. Although all the tubes in all three of the A rows showed complete hæmolysis at the end of two hours, there was a marked difference in the rate of hæmolysis between the tubes in each series. In each row A tube 6 was the first to hæmolyse and tube 1 the last. The slowing of hæmolysis in the tubes containing the large amounts of antiserum was very slight, but was sufficient to allow the inference that the presence of a large amount of serum is unfavourable rather than favourable to hæmolysis.

Table XI illustrates an experiment intended to demonstrate the effect of the presence of excess of antigen and antibody. The influence of relative excess of antiserum is well shown in the first two columns A and B. The effect of excess of antigen begins to be noticed in tube 1 of row C and is more marked in the first three tubes of rows D and E. The experiment shows very clearly that if the optimal effect is to be obtained antigen and antibody must be present in certain definite proportions. The influence of relative excess of either constituent is a matter of some interest from a theoretical standpoint. The effect of antiserum excess might be attributed to the reinforcement of the hæmolytic system by the "normal" hæmolysin present in the antiserum. The hæmolytic tube of the antisera used in these experiments is given in Table XII.

It seems, however, more probable that this inhibition by excess is comparable to that seen in other serum reactions.

It will be remembered that excess of antiserum causes a very similar inhibitory effect in agglutination, precipitation and hæmolysis experiments. The phenomenon is quite analogous to that described by Neisser and Wechsberg (1901) and it might be anticipated that both results might find a common explanation. Neisser and Wechsberg suggested that the free amboceptors took up or deviated a certain portion of the complement and that not enough remained to satisfy those amboceptors which had united with bacilli.

This explanation has not been universally accepted as the cause of the interference with bacteriolysis, and it seems almost impossible to apply it to the closely parallel interference with the fixation of complement. To do so it is necessary to assume that the free amboceptors deviate complement from the bound amboceptors merely to give it up again, later on, to the combined hæmolytic amboceptor.

Further experiments are being carried out at the present time on the quantitative relations of antigen and antibody and it is proposed to return to the question in a subsequent communication.

SUMMARY AND CONCLUSIONS.

(1) The complement-fixation method affords an extremely delicate and specific means of differentiating between various members of the typhoid and paratyphoid group.

(2) Very closely allied organisms can only be differentiated after experiments sufficiently extensive to secure a knowledge of the dilutions most suitable to the serum and extracts in use.

(3) By the use of the complement-fixation method *Bacillus typhosus*, *Bacillus paratyphosus* A, and *Bacillus paratyphosus* B can be easily differentiated from one another.

(4) By the use of a suitable dilution of serum it is possible to differentiate *Bacillus paratyphosus* B from *Bacillus Aertryck*.

This series of experiments entirely confirms the classification of Bainbridge.

(5) The group reaction and the degree of relationship between members of the group can be demonstrated by the use of various dilutions of an antiserum in the presence of extracts made from homologous and heterologous strains.

(6) Under certain circumstances the degree of relationship can be seen during the course of an experiment by observing the time of onset of hæmolysis in the tubes containing the various extracts.

(7) The group antibodies can be removed from the serum by a suitably arranged absorption experiment.

(8) Excess either of antiserum or of bacterial extract has an unfavourable influence on the fixation of complement.

(9) The maximal fixation of complement is obtained when antigen and antibody are present in certain definite proportions.

BIBLIOGRAPHY.

ALTMANN. "Komplementbindung und Agglutination bei der Paratyphus, Typhus und Coligruppe," *Centralbl. f. Bakt.*, 1910, Abt I, (Orig.), liv, p. 174.

BAINBRIDGE. "The Ætiology and Epidemiology of Paratyphoid Fever and 'Food-poisoning,'" *Proc. Roy. Soc. Med.* (Epid. Sect.), February, 1911, iv, p. 51.

BALLNER and REIBMAYR. "Ueber die Verwertbarkeit der Komplement-Ablenkungsmethode für die Differenzierung von Mikroorganismen, nebst Bemerkungen über den Zusammenhang dieses Phänomens mit der Agglutinations-bzw-Präzipitationsreaktion," *Archiv f. Hyg.*, Münch. u. Berl., 1908, lxiv, p. 113.

BORDET and GENGOU. "Sur l'existence de Substances sensibilisatrices dans la plupart des Serum antimicrobiens," *Ann. de l'Inst. Pasteur*, Par., 1901, xv, p. 289.

DEAN, H. R. "Recognition of *Bacillus typhosus* by Complement Fixation," *Brit. Med. Journ.*, 1910, ii, p. 1516.

LEUCHS. "Ueber die diagnostische Zuverlassigkeit und die Spezifizität der Komplementbindungsmethode bei Typhus und Paratyphus," *Berl. klin. Wochenschr.*, 1907, xliv, pp. 68, 107.

MEIER. "Die Komplementbindung mit besonderer Berücksichtigung ihrer praktischen Anwendung," *Jahresb. ü. Ergebn. der Immunitätsforsch.* (Weichardt), Stuttg., 1909, iv, p. 58.

NEISSER and WECHSBERG. "Ueber die Wirkungsart bactericider Sera," *Münch. med. Wochenschr.*, 1901, xlviii. p. 697.

PITT. "Der Bacillus nodulifaciens Bovis Langer, ein Vertreter der Enteritis II (Gaertner) Gruppe," *Centralbl. f. Bakt.*, 1909, Abt. I (Orig.), xlix, p. 593.

POSNER. "Ueber die klinische Verwendbarkeit der Komplementablenkungsmethode bei typhoiden Erkrankungen," *Berl. klin. Wochenschr.*, 1908, xlv, p. 1677.

SACQUÉPÉE. "Sur les Salmonelloses: Les Sensibilisatrices," *Compt. rend. Soc. de Biol.*, Par., 1907, lxiii, p. 421.

SIMNITZKY (quoted by Meier, 1909). "Ueber die Natur der Gruppenimmunkörper," *Russki Wratsch*, 1908, No. 33.

SPAT. "Ueber die modernen Methoden der Typhusdiagnose," *Wien. klin. Wochenschr.*, 1908, xxi, p. 1017.

WASSERMANN und BRUCK. "Ist die Komplementbindung beim Enstehen spezifischer Niederschläge eine mit der Präzipitierung zusammenhängende Erscheinung oder Ambozeptorenwirkung?" *Med. klin.*, Berl., 1905, i, p. 1409.

WIDAL and LE SOURD. "Recherches expérimentales et cliniques sur la sensibilisatrice dans le serum des typhiques," *Compt. rend. Soc. de Biol.*, 1901, liii, p. 841.

Pathological Section.

February 21, 1911.

Lamarckism and Callosities.

By S. G. Shattock.

De callositatibus in Camelo, Camelopardali, Cynocephalo, etc., quoad doctrinam transmissionis notarum acquisitarum referunt.

Summarium.

Hypertrophiae ex usu immoderato exemplum nullum forsitan indicari possit tam simplex quam teguminis callositates quae pressu trituque acquiruntur.

Notas acquisitas, illas praecipue usu immoderato causatas, transmissas esse, atque in hoc modo, omnes varietates quae evolutionem subtendent oriri, haec est doctrina quam Lamarckismum appellare licet.

In Camelo recumbente callositates supra sternum, carpos, genua, et eminentias quasdam minores, constanter acquiruntur.

Adsuntne in foetu? In neonato callositates omnino absunt. Cutis cum quâ hae partes teguntur, pilis praedita est non minus abundanter quam tegumentum commune adjacens; nec epidermis hic callosior.

Callositates etsi in singulis animalibus ex usu acquiruntur, haud tamen ad progeniem transmittuntur.

De Camelopardalis adulti callositate supra carpum; in neonato quidem pili sic producuntur ut tegiticulam supra articulum formant.

Callositates ischiales in Cynocephalo, et Macaco, repraesentantur, ex contrario, in neonatis, areis nudis quarum scrutatio microscopica nec pilos demonstrat nec pilorum folliculos.

Areâ atrichosâ pressui postea subjectâ, epidermis in crassitudine crescit et callositas, ut recte dicam, evolvitur.

Adhuc ergo non confirmatur quod vulgo putatum est, aream nudam ischialem ex usu acquisitam esse et abhinc transmissam.

Folliculorum absentia notanda est, quum in varietatibus animalium atrichosis (Mus musculus, Mus rattus, Talpa europaea, etc.), praedita est cutis pilorum folliculis, etsi pili aut absunt, aut exigue crescunt.

In the investigation of biological problems it is a maxim that they are best approached by a study of their simplest expressions. The question whether characters which are acquired by the individual during a lifetime, are capable of being transmitted, is one that is still under discussion, in spite of Weismann's important contribution to the evolutionary problem, which directly denies it.

The Lamarckian hypothesis that the acquisition of characters, as distinguished from their spontaneous appearance, is the chief source of the variation underlying evolution, though fully adopted by Herbert Spencer, was not admitted by Darwin as a satisfactory manner of accounting for the adaptation of the various forms of life to the localities in which they are found and the media in which they live. Much hangs upon the meaning given to terms in all such discussions, and neither of the terms concerned—" acquisition " and " transmission "—is invariably used in the same sense, or with the same limitations. Let me, however, take the anatomical data indicated in the title of the paper first; and afterwards proceed to comment upon them.

The chief feature in the Lamarckian doctrine is the transmission of such characters as have been acquired by increased exercise. What, then, is a simpler instance of local overgrowth resulting from increased exercise or use than an epidermal callosity?—the local thickening that comes from an increase of the pressure and friction which it is the natural function of the epidermis to bear. Any area is capable of so thickening under such circumstances; and, as a yet more pronounced result, the corium and subcutaneous tissue may participate.

The Callosities in the Camel.

Everyone is familiar with the callosities over the sternum of the camel, and those parts of the limbs on which the animal rests. They are constant features, whether the animals are reared in a menagerie or under the natural conditions of the desert; and they are present both in the one-humped and the two-humped varieties. The callosities are hairless areas of skin over which the epidermis is notably thickened, hard, dry, and fissured. The largest is that over the sternum, upon

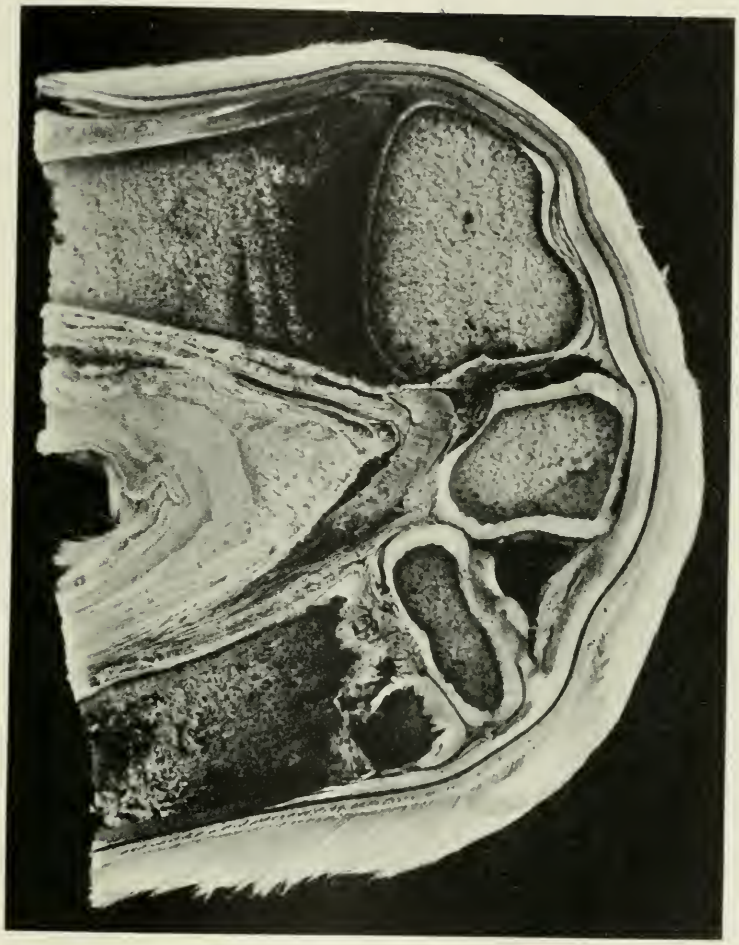


Fig. 1.

Carpi flexi sectio sagittalis ex Camelo neonato. Cutis ubique crassitudinis equalis est, atque pilis praedita. In situ callositatis supra ossis metacarpi extremitatem postea acquisitae, nec pili absunt, nec epithelium callosum.

A sagittal section of the flexed carpus ("knee") of a newborn camel, made exactly in the mid-line. The skin is of uniform thickness throughout, and is everywhere equally well provided with hairs. The site of the callosity which is subsequently acquired is over the metacarpus, immediately below the flexure of the joint. The preparation, with the others referred to in the text, is now in Museum of the Royal College of Surgeons. (The figure is slightly reduced.)

which the weight of the body chiefly rests. In the forelimb the callosity lies over the carpus (sometimes called the "knee"), and there is a second over the eminence of the olecranon. In the hind limb they are situated over the knee, and on the outer aspect of the ankle. An examination which I was able to make of the sterna of four camels shows that the bone beneath the callosity does not in any way participate in the overgrowth or sclerosis. One of these skeletons is in the Museum of the Royal College of Surgeons; the others I was able to inspect at the British Museum (South Kensington), through the kindness of Mr. Pycraft.

Such being the condition of the callosities in the adult, what is it at the time of birth? In order to study this I obtained, through the courtesy of Captain Flower, the Director of the Zoological Gardens, Cairo, the fœtus of a camel, which reached the College in excellent preservation, the animal having been dismembered and placed in formalin. The femur of this fœtus measures 25 cm. (10 in.), a length which closely corresponds with that of an animal at term. The hair of the limbs and trunk is quite white. The parts having been completely hardened, I was able to make very satisfactory sagittal sections through the sternum, the carpus, and the knee. The result shown is quite unambiguous. In not a single one of these positions is there the slightest absence of hair, or the least thickening of the epidermis, or of the corium. In short, the skin over the sites of the callosities which are afterwards acquired, is absolutely undifferentiated from the rest; nor is there any excess of subcutaneous fat over the carpus or knee; no fat, indeed, has as yet appeared in the subcutaneous tissue of the limbs at all. Beneath the skin over the sternum there is a thin layer of fat which attains a maximum of 0·5 cm. This, of course, does not constitute a callosity; at the most it could only be named a fat-cushion. And there is a fold of skin with a scanty inclusion of fat, representing the dorsal hump; this I have elsewhere fully described.[1]

In the two-humped camel born in the Zoological Gardens, April 10, 1909, the callosities were present at the several spots on which the animal rested, when I examined it three months later; they had by this time been acquired. This observation I can confirm from the inspection of a Bactrian Camel which was born in the Zoological Society's Gardens on March 12, 1911. The animal, which was very lively and active, I examined with Mr. R. I. Pocock, the Superintendent of the Gardens, twelve days after its birth.

---

[1] "Tumour-like Formations of Fat," *Proceedings*, 1909, ii (Path. Sect.), p. 207.

The hair over the sternum, carpus, and knee was as abundant as elsewhere, and there was no palpable trace of thickening or hardening of the integument in any of these positions. There were two small but well-pronounced dorsal humps, dropping somewhat to one side. The length of the femur, as approximately ascertained in the living animal, was 30 cm. (12 in.). When I again saw this animal, recumbent, on June 15, 1911, there were well-marked callosities apparent over the carpus and knee.

The Callosities of the Giraffe.

As a second illustration of the same kind let me next adduce that furnished by the giraffe. In the adult there is a hairless callosity over the front of each carpus, upon which the weight is received when the animal drops upon the joint in order to reach the ground before assuming the recumbent position. The callosity is of precisely the same nature as that of the camel; and the question whether it is present at birth can be quite as decisively answered.

About four years ago a giraffe was prematurely born, in the Zoological Society's Gardens, at the eighth instead of the sixteenth month of gestation. The fœtus, which of course did not live, was afterwards preserved in alcohol at the Natural History Museum, and I am indebted to Dr. F. S. Harmer and Mr. Pycraft for one of the fore-limbs. In this specimen, not only is there no trace of a callosity over the carpus, but the hair is here particularly thick and long. The presence of this "mat" was noticed by Mr. Pocock and by Dr. Beddard,[1] in this particular specimen; and by the former, moreover, it was observed in another animal which died in the Society's gardens when three weeks old. In the eight months' fœtus the hair over the limb in general is fine and downy; over the carpus it is long and coarse. The area so differentiated is sharply limited, and of peculiar form; superiorly it is semicircular, but below it tails off into a long, narrow process which extends over the front of the metacarpus, so as to give a racquet shape to the whole.

In a sagittal section carried exactly through the middle of the entire mat, a naked-eye inspection shows that the corium is thickened in connexion with the upper, more extensive portion, the hairs of which are readily traceable, and traverse the whole of its substance. The increased thickness of the corium is obviously associated with the greater depth to which these coarser hairs penetrate. The preparation exhibiting this is now in the College Museum.

---

[1] Beddard, *Proc. Zool. Soc.*, 1906, ii, p. 630 (fig. 109).

Of the mat I made a microscopic examination by means of longitudinal and of transverse sections, the latter being cut so as to include the adjoining unaltered skin. In the transverse sections the hairs in both situations have a similar direction, and are cut crosswise, or with slight obliquity; beyond the mat they are slender, of uniform size, and lie about four deep. Many of the follicular apertures are shown at the free surface; these give exit to single hairs only. In the mat the hairs are of two sizes: some resemble those of the surrounding skin; others of a much stouter kind are intermixed with them. Both kinds emerge separately at the surface. The thickness or depth of this cutaneous zone exceeds that of the skin around, the coarser hairs extending the deepest. In longitudinal sections of the mat both kinds of hairs are seen to be disposed lengthwise; they run parallel, and they emerge by separate apertures.

As to the glands present: these are all connected with the hair-follicles, and must be regarded, morphologically, as sebaceous. They lie at no great distance from the surface, parallel with, and close by the side of, the follicle; they are of a simple spindle form, and are occupied with the polyhedral vacuolated cells proper to sebaceous glands. There are no glands below the level of the hair-bulbs, and none other than those described.

The callosity subsequently acquired corresponds with the central part of the higher circular area of the mat, the tail of which persists. The area of overgrown hair increases in correspondence with the growth of the limb: and the relative length of the hairs is likewise maintained.

The occurrence of similar hair-mats over the carpus is a well-known feature in certain antelopes. Mr. R. I. Pocock[1] observes that it was pointed out some years ago by Dr. Murie that the carpal mat in one of these (Saiga) was glandular. Mr. Lydekker suggested that the same was true of gazelles.[2] The truth of this suggestion was proved by Mr. Pocock, who found that the skin in *Gazella dorcas* was here notably thickened, and that this was associated with an increase in size of the sebaceous glands; the hairs covering the area entangled a quantity of "scurfy secretion."

In these ruminants a well-marked circular callosity not infrequently

---

[1] "On the Specialized Cutaneous Glands of Ruminants," *Proc. Zool. Soc.*, 1910.

[2] *Field*, March 14, 1908.

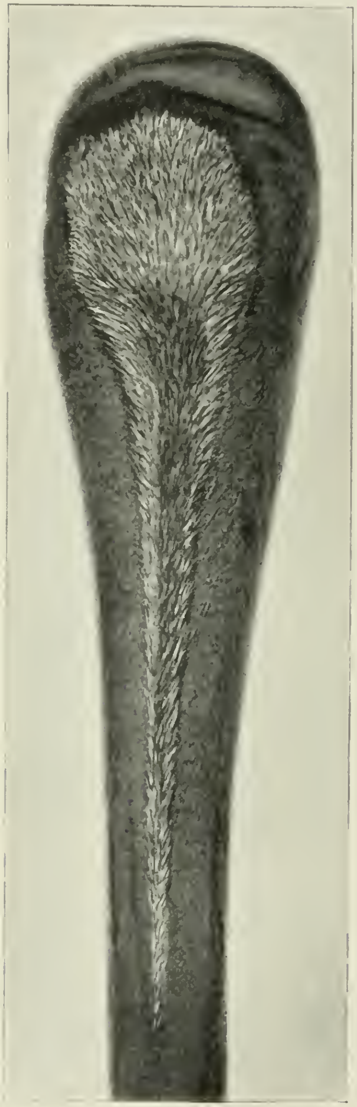


FIG. 2.

Camelopardalis praemature nati carpus plexus, a fronte aspectus, et pilorum tegeticulam supra articulum monstrans. In parte superioris hujus tegeticulae callositas postea acquiritur.

A front view of the carpus, flexed, of a fœtal giraffe born at half term, showing the mat of long hair over the joint, and which is prolonged for a considerable distance over the front of the metacarpus. Over the upper part of the mat the callosity is subsequently formed. From a photograph (natural size).

forms over the carpus from the animal's habit of dropping on to the joint in order to assume the recumbent position. It is noteworthy that the callosity in this case, however, is *above*, and does not involve the hair-mat; an observation which proves that the conjunction of the two, when it does occur (as in the giraffe), is a coincidence only. I have been able to examine in detail the carpal mat in a stillborn fœtus at term, of *Gazella subgutturosa*, with which Mr. Pocock was good enough to furnish me. The mat, as in the giraffe, is fully developed at the time of birth. In longitudinal section the corium, to the naked eye, is markedly thicker here than elsewhere; the roots of the long, pigmented, close-set parallel hairs being very obvious in its substance. A microscopic study of longitudinal sections made so as to include a portion of the adjoining skin shows that the hairs of the mat are stouter and penetrate more deeply than the rest. There is a large amount of unstriped muscle, in dense bundles, connected with the hairs (arrectores pilorum). The sebaceous glands are well developed and filled with the characteristic cells; these glands open into the follicles within a short distance of the surface and do not extend at all deeply by the side of the hairs. In addition to these there is a second series of glands more deeply situated, though not below the level of the hair-bulbs. These glands are of an extremely simple, flask-like form, lined with a single layer of cells, with wide lumen, and furnished with a long narrow duct which passes upwards, with shallow undulations, between the hair-follicles. These structures morphologically represent sweat-glands, and they occur both in the mat and in the thinner corium beyond it.

It cannot be said, therefore, that the area in question contains glands which are morphologically distinct from those of the skin around. Nevertheless, it cannot be denied that the glands may be physiologically modified so as to produce an odoriferous secretion, in the same way that the peculiar odour of the human axilla is not due to the presence of morphologically differentiated glands, but to a physiological specialization, and it is noteworthy that the hairs in this situation are correspondingly hypertrophied, as in the case of a hair-mat or hair-tuft.

Mr. R. I. Pocock (loc. cit.), in discussing the function of the cutaneous glands in the antelope, adopts the view taken by Flower and Lydekker,[1] viz., that the secretion serves to guide the individuals of a species, whether gregarious or not, to one another. The position of the pedal (or interdigital) glands, and of those on the carpus, tarsus, or metatarsus, would result in the leaving of a trail, either in moving over bare ground or through herbage, or when the animal lies down; and the same is true of the inguinal glands, when such are present. He does not think that this is the sole function of the cutaneous glands, since the odour of different glands in the same animal may vary, and what tale they may tell he confesses that he is unable to surmise. In regard to the well-known pre-orbital gland or "pit" of antelopes, Mr. Pocock cites evidence which indicates that it may play a sexual part: the gland, for instance, is more highly developed in the male, is small before puberty, and is most active during the summer season.

---

[1] "Mammalia," pp. 315, 316.

A curious case in the human subject is cited by Zippe, from Professor Most, of Rostock, where the cutaneous scent of the axillary glands of a particular man possessed a remarkably sexually excitant quality. It is noteworthy that the normal overgrowth of the axillary hair, like the pubic, is inhibited by removal of the sexual glands, or in cases of their hypoplasia—a fact suggesting that the cutaneous appendages in general, of these areas, have specialized sexual characters. Examples of the converse, where the odour of the "perspiration" of the female produced a corresponding effect upon the male, are given by Krafft-Ebing ("Psychopathia sexualis").

In the case of the glandular hair-mats of ruminants, the local thickening of the corium cannot be ascribed solely to the increased development of the glandular appendages; the local overgrowth of hair is an equal factor in its production.

The Ischial Callosities of Monkeys.

Let me next take up the subject of the ischial tuberosities so well known in the baboon and certain other monkeys of the Old World, for they are absent in those of the New; nor are they present in the chimpanzee, gorilla, or orang. These callosities are hairless cutaneous areas which are covered with a thickened—it might be called even horny—epidermis, and lie over the ischial tuberosity. They are confined to the tailed forms, and are especially evident in the large terrestrial baboons, with dog-like muzzle, or Cynocephali. They are not limited to this group, for they are well developed in the Common Macacus, an arboreal form. In the tailless and arboreal Gibbons (Hylobates) there are naked spots on the buttocks in the position of the callosities, though these may be hidden by the long hair of the skin around.

In regard to these callosities, the general view held is that they are attributable to the squatting attitude adopted by such monkeys as possess them, during the greater part of their life, especially during sleep. As will presently appear, they offer a striking histological difference from those of the camel, and one so marked that if the latter are due to friction and pressure, as they undoubtedly are, those of the monkeys cannot be attributed solely to the same cause. The fully developed callosities of the baboon and macaque are as hairless as those acquired by the camel; and to put the same question here: Are they present in the newborn animal? In both monkeys the area is quite hairless at birth. Whether it can, at that date, be strictly called a callosity will appear from its microscopic anatomy.

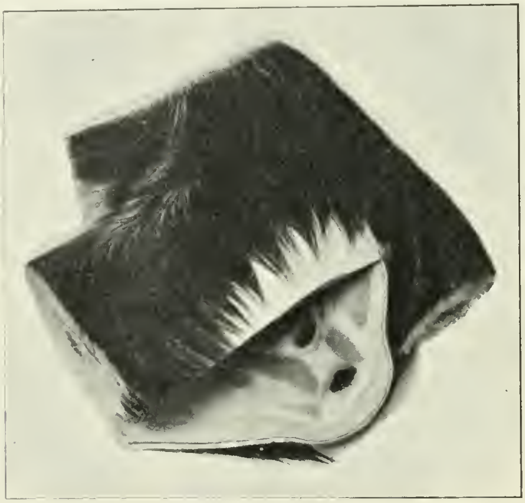


Fig. 3.

Callositatis ischialis sectio sagittalis in Cynocephalo (C. sphinx) neonato. Cutis nuda sed tenuis, in situ callositatis postea acquisitae. (Magnitudinis naturalis).

The lower part of the trunk of a newborn Guinea Baboon (*Cynocephalus sphinx*), showing the hairless "callosity" of the left side, in sagittal section. The surface over the most prominent part of the naked area, viz., that corresponding with the subjacent tuberosity of the ischium, is devoid of the fine wrinkles presented by the skin around. The subcutaneous fat is thicker here than elsewhere, but the hairless corium is thinner than the surrounding piliferous skin. Microscopic sections show no appreciable difference in thickness of the epidermis in these different situations. The tail has been cut off. From a photograph. (Natural size.)

I have been able to examine the ischial "callosity" at the time of birth in the Guinea Baboon (*Cynocephalus sphinx*) and in Macacus. Of the former a preparation is now added to the College Museum. It exhibits the lower part of the trunk of a newborn fœtus, showing the hairless areas over the tubera of the ischia, a sagittal section being made through that of the left side. The surface over the most prominent part of the hairless area, viz., that corresponding with the subjacent tuberosity of the ischium, is quite devoid of the fine wrinkles presented by the surrounding integument. The subcutaneous fat is thicker here than elsewhere, but the hairless corium is thinner than that of the piliferous skin around. The investing epidermis is no thicker on the naked area than elsewhere.

Microscopic sections made through the parts, so as to include portions of the surrounding skin and of the subjacent, and as yet cartilaginous, ischial tuberosity, show that the last-named consists of hyaline cartilage, invested with a stout perichondrium: there is a well-marked layer of subcutaneous fat, which is thicker over the summit and sides of the cartilaginous eminence than elsewhere. The skin over the tuberosity and for some way in front of it, is absolutely hairless and without papillæ, but immediately behind it bears hairs. Vertical lines

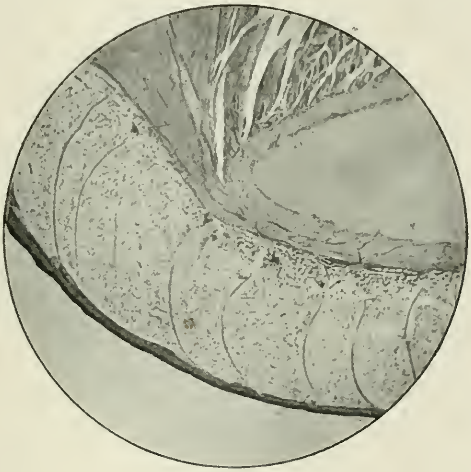


FIG. 4.

Sectio microscopica areae nudae ischialis Simiae praecedentis. Pili atque pilorum folliculi omnino absunt. Cutis epidermide incluso tenuis. Panniculus adiposus inter cutem et tuberositatem ischii insignis crassitudinis.

A vertical section through the "callosity" of the newborn fœtus of a Guinea Baboon (*Cynocephalus sphinx*), showing the septa of connective tissue passing from the corium to the perichondrium of the ischial tuberosity. The skin is smooth, thin, and quite devoid of hairs or hair-follicles: the epidermis is not thicker over the area in question than over the piliferous skin around. (2-in. objective. Hæmatoxylin and eosin.)

—i.e., sections of septa—of connective tissue pass from the corium to the perichondrium, fixing the overlying soft structures firmly in position. Not only are there no hairs, but there are no traces of hair-follicles. There are, moreover, no sebaceous glands, and no sweat (or other) glands, although these are well developed in the piliferous skin immediately adjoining.

In regard to the epidermis over the hairless area, this consists of a deep palisade series of cells, succeeded by polyhedral (about three deep), then by flat, and most superficially by a thin horny layer without stained nuclei. There is no continuous stratum granulosum, but isolated granular cells occur in the proper position of the stratum.

Between the hairs on the piliferous skin behind the naked area the epidermis has the same structure, but it is wrinkled in place of being flat. There is no appreciable difference in the thickness of the epidermis over the naked corium and that between the individual hairs of the skin behind it.

In the case of a newborn Macacus, on which the stump of the umbilical cord was present, and which I have been able to examine, the microscopic sections showed a simple corium over the naked area, without papillæ, and devoid of hairs or hair-follicles. The investing epidermis is quite thin, and the deepest cells, unlike those of the piliferous skin around, have a very imperfect palisade arrangement, and are small and polyhedral.

It will appear from these observations that at the date of birth there is no callosity in either the Baboon or Macaque—i.e., there is no hardened area, but simply a hairless patch of skin over the ischial tuberosity. The thickening of the epidermis is acquired after birth by the use, the increased exercise, to which the area in question is put. In a young Mandrill (*Cynocephalus mormon*) in which the first set of teeth had been erupted and the third molar was present in the upper and lower jaws, a sagittal section of the callosity, carried through the subjacent ischial tuberosity, shows the naked area to comprise a thickened, unpigmented epidermis investing a thin corium, beneath which there is a layer of dense fat, that is elsewhere absent. Microscopic sections show that the epidermis of the callosity presents a striking thickening of the horny layer; beneath this there is a continuous stratum granulosum, and beneath the latter a Malpighian layer. Into the last-named there project a regular series of somewhat obliquely set papillæ. Beneath the corium there is a layer of fat parted into lobes by fibrous processes or septa, which are fixed on the deep aspect to the periosteum covering the ischial tuberosity. At the margin the horny and Malpighian layers become thinner and merge into the general epidermis, the papillary construction becoming correspondingly less pronounced. The corium beyond the callosity is abundantly furnished with complex branching pigment cells. The macroscopic preparation is now in the College Museum. In the adult Macaque the callosities are particularly well developed, and assume the form of two pale, callous circular bosses. In microscopic sections the corium of the callosity is regularly papillary, the interpapillary spaces being evenly filled in with an epidermis of the regular pattern, including prickle cells, and stratum granulosum, with a stratum lucidum, succeeded by a thick horny cap. From the under side of the corium stout septa of connective tissue are prolonged, the meshes of which hold groups of fat-cells. The skin beyond is wrinkled and piliferous; the epidermis devoid of thickening, and without papillæ.

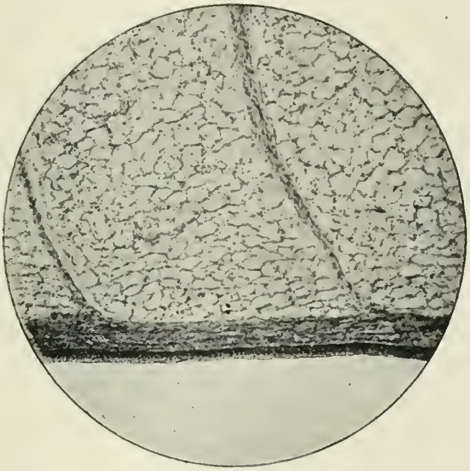


Fig. 5.

Sectionis praecedentis portio magis magnificata.

A portion of the preceding section more highly magnified, showing the thin epidermis covering the smooth corium of the "callosity"; there are no traces of hairs or of hair-follicles, or of any kind of glands. (Two-thirds objective.)

I have had the opportunity of examining one Gibbon (Hainan Gibbon—*Hylobates haïnanus*) which died in the Zoological Society's Gardens when about three-parts grown. In the Gibbons the callosities are absent, but their position is indicated by the presence of a hairless area on either buttock. The area in question is of a deep black colour in this specimen, like the rest of the skin. The corium is well developed, and provided with papillæ, but the surface is levelled and smooth, from the filling of the interpapillary spaces with epidermis. All the epidermal cells hold fine granules of black pigment; there is upon the Malpighian layer a stratum granulosum, followed by a stratum lucidum, and a somewhat thick horny layer of flat cells, which are likewise pigmented. The fat beneath presents no special features. The naked area is not absolutely devoid of hairs, but they are few in number; well-developed sweat-glands occur throughout its extent. The area, in this young adult, has acquired no such corneous thickening as it has in the young adult of the Mandrill already described.

The group next below the monkeys, in the mammalian scale, is that of the marmosets, and it is of interest to inquire whether the callosity is indicated in these. In the adult there is not only no indication of hairlessness, but microscopic sections reveal no differentiation whatever in the integument of the buttock. The skin is loose and wrinkled, regularly provided with fine hairs (as elsewhere); the epidermis not thickened; and the subcutaneous fat not tied down by septa to the subjacent ischial tuberosity.

Congenital Atrichosis.

The callosities acquired by the camel, as already pointed out, differ from those in the monkey in so important a way that whatever explanation holds for the one must fail for the other. In the camel the hair is fully developed on the areas in question at the time of birth; in in the monkey it is, even microscopically, completely absent. This complete absence is a striking fact, for my own observations show that the skin of animals born as hairless variants is abundantly furnished with hair-follicles, although the hairs are absent or exceedingly undeveloped.

Hairless Mice.—The longest known example of this variation amongst rodents is that of the mouse; and the first recorded instance is that placed by Clift, in 1820, in the College Museum, of which he was then the conservator. It was caught in the fireplace of a room in his house in London, and is a fully grown animal, which is stated to have been hairless from birth: the vibrissæ on the muzzle are normal. In 1855 Mr. J. S. Gaskoin[1] described the same condition from further observations as a zoological variety which he named *Mus nudoplicatus*. In this case hairless parents had hairless progeny; the latter, unfor-

---

[1] *Proc. Zool. Soc.*, 1855, p. 38.

tunately, did not propagate. Gaskoin states that he examined sections of the skin but that he could see no hair-follicles. This last observation is open to grave doubt, for not only do my own microscopic sections of Clift's specimen demonstrate that the apparently hairless skin is abundantly provided with hair-follicles, but the same is true of four hairless rats and of a hairless mole which I have had the opportunity of examining. In all of the animals mentioned the skin is remarkably wrinkled or rugose, as in Gaskoin's breed, whence his name "nudoplicatus." Microscopic sections of one of the cutaneous folds of Clift's specimen reveal the presence of a full number of patulous hair-follicles, provided with sebaceous glands. The supraglandular segments of the follicles, or the portions above the entrance of the ducts, are loosely filled with delicate, desquamated epithelium; the infraglandular, are markedly finer, and without lumen. In connexion with certain of the lower segments small follicular cysts have arisen; these are lined, and filled, with similar epithelial scales.

Of *hairless rats* there are no fewer than four specimens in the possession of the College. All are fully grown and well developed, but quite hairless, except that the vibrissæ on the muzzle are not wanting. The skin is white, and in each it is everywhere remarkably rugose, as in the hairless mice already referred to. Specimen (1) is exhibited in the College Collection of Teratology. Microscopic sections of one of the folds of the skin of the neck show an abundance of hair-follicles, loosely filled with desquamated squamous epithelium, and furnished with sebaceous glands. In the cross-section of one follicle there lay a delicate pigmented hair. The animal was killed in a barn near Colchester (1872), and the interesting note is preserved that it was killed with a second of the same kind, which is now in the College Stores. The history suggests that the two individuals were of the same litter, and that the variation was probably a transmissible one, as in the case of Gaskoin's *Mus nudoplicatus.* Both of these rats, I find, are female. The microscopic examination of one of the cutaneous folds of the second of these two rats shows a similar abundance of empty hair-follicles. In a very few there is a central hypoplastic hair in the midst of the desquamated epithelium. The follicles are furnished with well-developed sebaceous glands, presenting the characteristic vacuolated cells. Microcysts occur, as described in the case of the hairless mouse. In one of the largest of such cysts there is a group of fine downy hairs, in addition to the desquamated epithelium. Similar cysts containing undergrown downy hairs are met with in senile atrophy of the human skin, the hairs being shed within the follicle as their development is repeated at its fundus. The striated muscle of the panniculus carnosus is well developed. The muscle follows the course of the fold, the central part of which consists of extremely delicate open connective tissue. In order to ascertain how far the rugosity of the integument might be ascribed to an ill-development of elastic tissue, sections were stained with Unna's acid orcein for twenty-four hours. The dye disclosed a number of very fine crisp fibrils of elastic tissue, intermingled with the feltwork of coarser "white" fibres constructing the corium. On comparing the sections with those of the skin of the thorax of an adult albino rat, stained by the same method, I could detect no difference in the amount of elastic tissue in the two cases. As an histological detai I may notice that the proper fibrous walls of the patulous follicles in the hairless skin are furnished with a beautiful network of fine elastic fibrils disposed horizontally to the axis of the follicle.

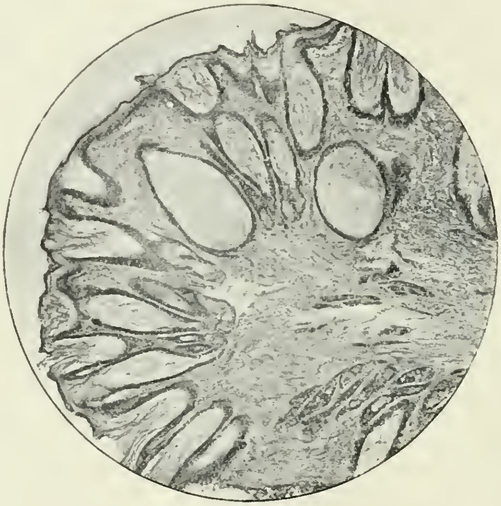


FIG. 6.

Cutis plicae sectio ex mure adulto atrichoso (Mus rattus). Cutis plurimis pilorum folliculis praedita est, folliculis cum cellulis squamosis desquamatis distentis. Folliculi quidam in cystibus dilati sunt. In plicâ mediâ monstrantur fasciculi musculares qui ad panniculum carnosum pertinent. Pili absunt.

Section of a fold of skin from the neck of a fully grown hairless female rat (Tegetmeier's specimen). The skin is provided with an abundance of follicles, which contain no hairs, but are loosely filled with desquamated epithelium. Some of the follicles are in this way distended into microcysts. In the connective tissue are shown sections of well-developed muscle-bundles—panniculus carnosus. (Low magnification.)

A third hairless rat (female) was presented to the College by Mr. W. B. Tegetmeier. Microscopic sections of a fold of skin from the neck, and of a second piece of the skin of the abdomen, showed, as in the preceding specimens, an abundance of hairless follicles filled with loose desquamated epithelium. Sebaceous glands occur in connexion with the follicles, and the bundles of the panniculus carnosus are well preserved.

Of a fourth specimen (a male), which was obtained for the College by Dr. C. G. Seligmann whilst pathologist to the Zoological Society, microscopic examination of a cutaneous fold from the neck shows precisely similar appearances, but in this case a certain number of very fine hypoplastic hairs are to be met with. The hair is in some instances closely fixed in the follicle, in others it lies in the midst of a core of desquamated follicular epithelium. To the follicles there are appended the usual sebaceous glands. It is to be further observed in regard to the hair-follicles, that whilst they are of striking diameter, this is so only above the entrance of the sebaceous glands, which, as already

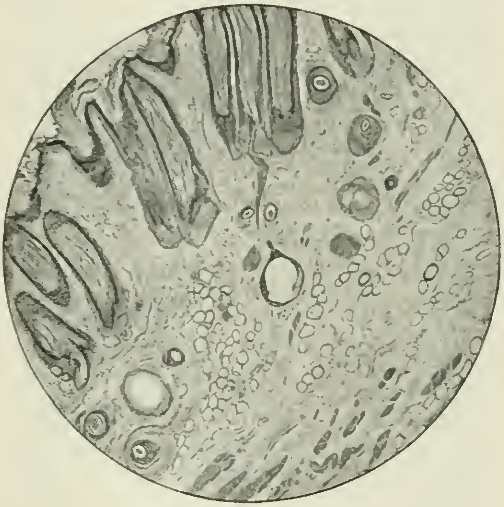


FIG. 7.

Cutis sectio ex mure adulto atrichoso (Mus rattus). Ut in exemplo praecedenti, cutis plurimis pilorum folliculis praedita est, folliculis cum cellulis desquamatis distentis. Hac atque illac trausverse sectus, pilus monstratur tenuis, aut in epithelio desquamato aut in folliculo minuto stricte infixus.

A section of the skin of another fully grown hairless rat (Dr. C. G. Seligmann's), showing a regular series of empty hair-follicles. Here and there a fine hair is to be seen in cross-section, either closely fixed in the follicle, or in the middle of a core of desquamated epithelium. The fasciculi of the panniculus carnosus are well developed. (Low magnification.)

stated, are well developed; below this level the follicle is very slender, of considerable length, and terminates in a solid bulb of epithelial cells. No papilla has been developed in connexion with the bulbous end, and the narrow portion of the follicle, being hairless, has no lumen. Some of the solid bulbs show a small central cavity, but this is in most, if not all, cases empty; in follicles slightly larger a small hair may be present. Owing to the absence of any hair to stiffen the structure, the slender infraglandular part of the follicle, though often straight, is at times bent out of line. The marked breadth of the supraglandular portion of the follicle must be attributed to the desquamation of its lining epithelium, and to the passage of sebum into it; it does not imply that the cavity was at one time filled with the shaft of a hair.

In all these hairless rats and mice the corrugation of the skin is a striking feature, which I should be disposed to attribute to a tonic reflex contraction of the cutaneous musculature, resulting from the unprotected character of the surface. The panniculus is, in fact, fully developed, and there is no lack of elastic tissue intermingled with the coarser fibre of the corium. The claws in all are dense and well grown.

Hairless Mole.—Of this variation a curious example was presented to the College Museum in 1905 by Sir Jonathan Hutchinson. The preparation is a Common Mole (*Talpa europæa*), the skin of which is quite devoid of hair; the tactile bristles of the snout and those of the forepaws, however, are of normal length. The integument is everywhere thrown into folds, which in general take a horizontal direction, and on the back form a remarkably regular pattern. The animal, when accidentally unearthed, was quite active. Microscopic sections of the skin show the presence of closely set follicles partly filled with shed epithelium, but no hairs.

Spineless Hedgehog.—In the College Museum there is also a specimen of a hedgehog (*Erinaceus europæus*), of which the skin is almost devoid of spines. The largest of the spines present measures an inch in length, and equals the normal in general dimensions. The hairs on the skin of the face and the abdomen are well represented. Of the integument of this specimen I have made no microscopic examination.

Hairless Dogs.—In the same group of atrichous varieties we must place the hairless dogs of China, Mexico, and Africa. In this country hairlessness may occur as a variation, more particularly in the black and tan terriers, which appear to be associated with the blue and tan. Slaty-blue and tan dogs are prone to have the hair short and sparse, and hairless examples may occur in the same litter. The hairless dogs of

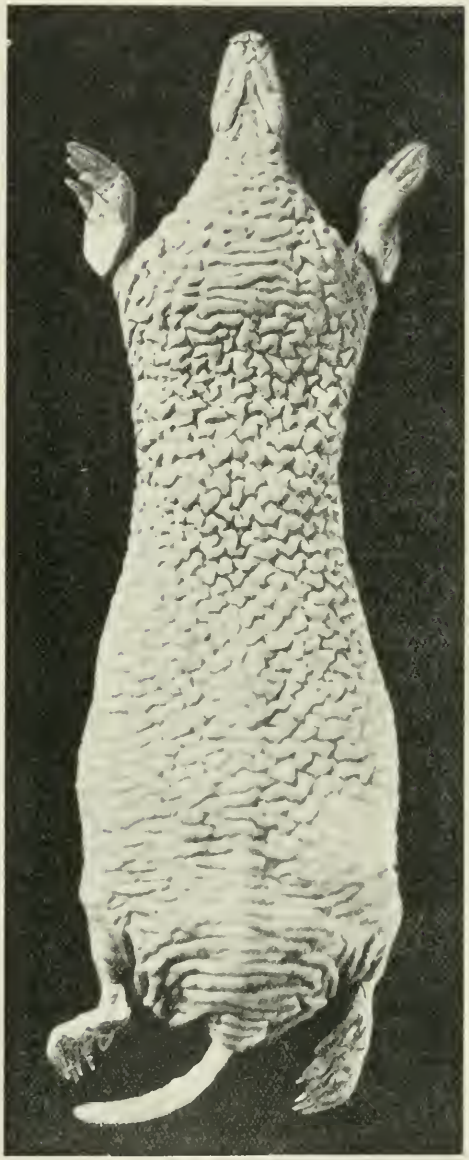


FIG. 8.

Talpa europaea atrichosa a dorso aspecta. Cutis insigniter rugosa, quum in muribus jam descriptis. Sectio microscopica cutem folliculis sine pilis praeditam esse demonstrat.

The hairless mole described in the text, showing the rugosity of the skin, common also to the hairless mice and rats referred to. The animal is viewed from the dorsum.

Peru and Mexico are usually held to have been imported, and not to be indigenous to South America. In support of this, Buffon[1] observes that whilst two varieties of American dog have native names and are certainly indigenous, the hairless dog has no such name. Humboldt, in his work on Deserts, Vegetable Physiognomy, &c.,[2] remarks that he was much surprised at Quito and in Peru by the large number of black hairless dogs (*Canis egyptius*, Linn.), and he states that in the Spanish Colonies the hairless dog was regarded as having been introduced from China, from Canton, or Manila, and was called the Chinese Dog; he puts the question whether it might not have been imported by the Portuguese from Africa, with the productions of that country. I had the opportunity, not long ago, of inspecting a famous Chinese dog from the Pekin Palace. The skin was quite hairless, except that there was some thin black, silky, fairly long hair on the scalp, which lay back over the head, and a scanty tuft of hair at the tip of the tail; some hair, furthermore, was present on the face and feet. The skin was black, mottled with pink, and acquired a more pronounced and general blackness as the weather became warmer; in September the pink mottling would reappear. There was no general rugosity of the integument, but a local wrinkling over the back. The animal did not feel the cold, and wore no coat. The pups bred by this dog (a male) were also hairless. The dog was highly intelligent; it had no goitre.

In the *Proceedings of the Zoological Society*[3] is recorded the exhibition by Mr. William Yarrell of an example of the hairless Egyptian variety of *Canis familiaris*, which had recently died in the Society's Gardens. There were no incisors and no canines in either jaw; the molars were reduced to one on each side, the large tubercular tooth being the only one remaining. Yarrell stated that he had examined the mouths of two individuals of the same variety then living in the Gardens, and in both of these the teeth were remarkably deficient. In neither case were there any false molars, one animal was entirely destitute of canines also, these teeth being, in the other, short of the proper number; the incisors were in both numerically deficient. The same author also exhibited the cranium of a hairless terrier in which the false molars were wanting.

Heterocephalus glaber; H. phillipsi.—Under the above names are described two species of a small "hairless" rodent indigenous to Africa

---

[1] "Histoire Naturelle," tome xv, chap. xiv, p. 151.

[2] "Tableaux de la Nature," sec. ed., 1826.

[3] 1883, p. 113.

—the first from Abyssinia, the second from Somaliland. *H. phillipsi* was established by Mr. Oldfield Thomas at the Natural History Museum, South Kensington, from an adult female obtained in Somaliland, and afterwards preserved in alcohol. This specimen is figured in the *Proceedings of the Zoological Society*, 1885, p. 845. The animal is of about the size of a common mouse. The muzzle is well furnished with tactile hairs, but the hair of the rest of the body is so fine and sparse that the skin at first sight appears hairless. There is a general and marked wrinkling of the skin. There is no want of claws or of teeth. The rodent is well known in Somaliland from the small craters of earth which it throws up in making its burrows, and has a native name.

Featherless Fowl.—Aplasia or hypoplasia of the epidermal appendages is not confined to mammals. One curious example is furnished by a specimen in the College Museum of a fully grown and almost featherless fowl (Teratological Series). It was hatched without feathers, and lived in this condition for seven months. A few small feathers are sparsely scattered over the sacral region and upper part of the neck. The skin is delicately wrinkled.

I may here raise the question whether these forms of hairlessness are etiologically associated with thyroid deficiency. Theoretically, hairlessness, like the shortness of the long bones in short-limbed dogs, such as the dachshund, might be attributed to a physiological deficiency on the part of the thyroid gland, short of that which results in the combination of lesions characterizing cretinism and myxœdema. Hairlessness and shortlimbness might represent graphically the occurrence of chemical aberrations in the thyroid secretion, the secretion being in different cases deficient in different of its constituents. And to these two kinds of modified external characters might be added certain forms of obesity, characterized by a particular firmness of the fat, and which are especially benefited by the administration of thyroid extract.[1] The case of the dachshund is fully discussed by Dr. C. G. Seligmann and myself in the third volume of these *Proceedings*.[2] The histological picture of the epiphysial line in the dachshund pup at the age of 3 months presents no departure from the normal; in this it differs from that furnished by the human nanomelic or short-limbed fœtus at term, whether the want of growth is an element of true cretinism, or the non-cretinous condition, named, for the sake of distinction, achondroplasia. In a fully investigated example of human achondroplasia lately added to the College Museum (95.1, General Pathology), vertical sections of the lower end of the femur cut after decalcification in picric acid, showed the following points: The cartilage cells at the epiphysial line are increased in size, but form only short muriform rows; the intervening matrix is calcified, as normally, but the trabeculæ are individually short, and produce rather a honeycombed than a tubular structure; beyond the primary areolæ the cartilage cells are enlarged and flat for a few strata, but row-formation is absent, the cells having apparently enlarged in situ without having proliferated. There was no history of the condition being hereditary, and the other children of the family presented nothing of an unusual kind. The condition in this case may be classed as achondroplasia, since the marks of cretinism are absent. The subcutaneous fat is not excessive; there are no fat-masses at the root of the neck, and there is no thickening of the integument of the lips or of the alæ nasi. The root of the nose is depressed, but this, arising as it does from diminished intracartilaginous growth of the basis cranii, is not more a mark of cretinism than of simple achondroplasia. The important point is that the thyroid gland is of full size, and microscopically I find that it is constructed of a plexus of solid epithelial columns, a fair number of which present a sharply defined lumen filled with colloid, the spaces so occupied being lined with a single layer of cubical cells; the amount of colloid equals that normally present at the time of birth. The pituitary is of natural size, and the distinction between its lobes is sharply marked; histologically the glandular lobe exhibits no discernible deviation. The adrenals are of full dimensions, and neither the medulla nor the cortex present any histological abnormality.

---

[1] A. Lorand, *Trans. Path. Soc.*, 1906, lvii, pp. 17, 18.

[2] S. G. Shattock and C. G. Seligmann, *Proceedings*, 1910, iii (Path. Sect.), p. 107.

The thyroids from the two dachshunds, whose bones were examined histologically, and who died when 3 months old of distemper, showed a perfectly normal, fully developed vesicular structure. In one case the spaces were filled with homogeneous colloid, well stained with eosin; in the other a certain number of the spaces only were so occupied, the others being empty. The crucial experiment of administering sheep thyroid to a dachshund pup until the animal was fully grown was unattended with any increase of the bones beyond what is normal in this variety. (S. G. Shattock and C. G. Seligmann, loc. cit.)

To revert to the hairless animals described; I have examined the thyroid in the rats, and also in the featherless fowl already described. Owing to the rarity and costliness of hairless dogs in this country the results of thyroid feeding have not been ascertained. Of the featherless fowl the thyroid was dissected out and microscopically examined. In the bird the gland consists of two widely separated lobes, deeply situated at the root of the neck, and lying on the common carotid artery. The left gland of the featherless bird was a compressed oval body, 1 cm. by 0·6 cm. in diameter, and 2·5 mm. in thickness. The microscopic sections exhibit a proper vesicular structure, the smaller spaces of which have a cuboidal or subcolumnar lining. The colloid occupying the vesicles is homogeneous, and stained of a pink colour with eosin; the margin of the drop is, in some, dentate; in most cases the substance is to a certain degree vacuolated. On comparing the section with one of the thyroid of a normal fowl, no difference of any significance was recognizable.

In regard to the thyroid glands in the hairless rats, the condition of the material, unfortunately, did not allow of any satisfactory study, except in the case of the fourth. The microscopic sections of this show a well-pronounced vesicular structure, the spaces of which are lined with cuboidal epithelium, but hold very little colloid, the contents being finely granular, with hyaline droplets only here and there. There is no small cell infiltration of the stroma. The parathyroid on the outer border of the lateral lobe of the gland is of normal size and structure. This observation is insufficient to allow of the establishment of any relationship between the atrichosis and a physiological thyroid deficiency. In the normal adult albino rat the vesicles hold considerably more colloid, but I find that many may be empty.

P. Ziegler[1] has, in a case of congenital atrichosis in a girl, aged 17, treated the patient with fresh thyroid gland, but after two months she disappeared, without the slightest benefit having by that time resulted. Dr. P. S. Abraham[2] has obtained some improvement in a case of complete alopecia, after ten months' treatment with thyroid gland; hair began to show itself on the eyebrows and body, as well as on the scalp.

ATRICHOSIS AND HYPOTRICHOSIS IN MAN.

This subject is so fully discussed in dermatological literature that I need only refer to it in order to make the preceding list complete.

---

[1] *Archiv. f. Derm. und Syph.*, 1879, xxxix, p. 213.

[2] *Brit. Journ. Derm.*, 1895, vii, p. 162.

One of the best accounts of such a case is that given by Paul Ziegler (loc. cit.), since it is accompanied with a full histological examination of the skin. The sebaceous and sweat-glands were well developed; the hair-follicles were without hairs, but distended with epithelial debris; the arrectores pilorum were readily recognizable. The subject was a girl, aged 17; her parents and sisters presented no similar abnormality.

In our own literature there is the case recorded by Thurnam[1] of two cousins, in whom atrichosis was associated with deficient development of teeth, and who never perspired or shed tears. The hair was represented by a lanugo-like growth on the body and head. Macnaughton-Jones and Atkins[2] describe the skin of a congenitally hairless child, as showing, on microscopic examination, sparse, abortive hair-follicles. In Sir Jonathan Hutchinson's case,[3] that of a girl aged 3½, the scalp was exceedingly thin and provided only with down; it looked semitransparent and tight. The skin was everywhere very thin; over the summit of the shoulders it was not much thicker than brown paper. The nails were remarkably thin. There were no nipples and nothing like a mammary gland could be detected. There was nothing abnormal with the teeth. The child's five sisters, all older than herself, had excellent hair, but her mother had suffered since childhood from alopecia. The connexion between these two manifestations of hairlessness the author leaves open. Dr. Radcliffe Crocker, in his "Atlas," figures (Plate X6, fig. 15) a case of congenital atrophy of the skin in a female child, aged 5, accompanied with congenital absence of the hair and nails. The hair of the scalp hardly grew at all till the child was 3 years old, but it eventually grew well. There was nothing abnormal in the condition of the teeth. The parents were healthy, as were also two other children. The skin was thin and dry, and the whole surface, especially of the limbs, was finely wrinkled. During the first years of life small blisters would appear on the fingers and toes. This production of blisters is explicable by the readiness with which mechanical displacement of the attenuated epidermis is brought about; a result which has received the name "epidermolysis bullosa."

In some of the preceding cases the hypoplasia of the cutaneous appendages was associated with a similar want of development of other glands of cutaneous origin—viz., the mammary and lachrymal. Physiologically interesting, in one case of aplasia of the sweat-glands, is the occurrence of fever, which was induced on exposure to sun, or by moderate work, the subject being an adult male; although the skin became flushed no sweating took place; the temperature would rise to 39° C. or more.[1] In some of the examples of human atrichosis more than one member of the family was affected, and the condition in some was inherited.

---

[1] *Med.-Chir. Trans.*, 1848, xxxi, p. 71.

[2] *Dublin Med. Journ. Sci.*, 1875, lx, p. 200.

[3] *Med.-Chir. Trans.*, 1886, lxix, p. 473.

Miklucho-Maclays[2] has recounted the case of a family of Australian aborigines, of whom the father was reputed to be hairless; of the mother no information was forthcoming. The children comprised one son and four daughters; of these the son and the two eldest daughters were hairless, and were born so; the two younger sisters (by the same father?) were haired. The hairless brother was devoid of hair, with the exception of scanty eyelashes and a few hairs in the nostril; the axillæ and pubes were as hairless as the scalp and rest of the body. The same description applied to the sister, a woman of about 50 years of age. The teeth were sound. One of the hairless daughters had two normally haired children.

Dr. P. S. Abraham[3] records the case of an almost hairless woman who had two children (girls), both of whom were born with scanty hair. The mother, aged 33, was born with a little "fluff" on the head, but this soon came off, and she remained quite bald and without hair anywhere, until about 18 years of age, when it appeared on the head, and on the axillæ and pubes. One child, aged 5, was born with a very little white down on the head, which fell off at the age of 3 months, and since then she has been in a condition of almost complete alopecia. The other, aged 15 months, was born with a little black hair, which fell off at the same age.

Schede[4] describes a family in which there were two congenitally hairless children, a boy aged 13 and a girl aged 6 months; two other children, like the parents, were normal. Microscopic examination of a piece of the boy's scalp showed the presence of large sebaceous glands, rudimentary follicles, and sweat-glands.

If we take a broad view of the various examples of hairlessness cited in the present communication, they may be divided into two categories.

---

[1] Züntz and Tendlau, "Physiol. des Mensches," Berl., 1901, p. 311.

[2] *Zeitschr. f. Ethnol.*, 1881, xiii, p. 143.

[3] *Brit. Journ. Derm.*, 1895, vii, p. 162.

[4] *Archiv f. klin. Chir.*, 1872. p. 158.

One set may be viewed as pathological, and as involving a hypoplasia not merely of the epidermal appendages, but of the derma as well. The thinness or absence of the nails in some cases proves that this is so, as does likewise the extreme thinness of the skin in general. The absence of hair is not to be ascribed to the absence of follicle, but to the aplasia of the papilla in its fundus, which is, of course, the true source of the hair. The absence of teeth is part of the same pathological condition, the development of the dental papilla failing like that of the hair-follicle. Into the other group fall the examples which may be viewed as morphological or zoological variations. In these the corium is normally developed, but the papilla at the fundus of the hair-follicle has failed wholly or in a marked degree to grow, though the sebaceous glands are well represented. In the four hairless rats described in the present communication, the claws are fully grown and hard, and the teeth are quite normal; and the same is true of the hairless mole. It may be added, too, that in the two females of the hairless rats the nipples are well developed. Under natural conditions, the survival of a sport wanting claws and teeth and mammæ would be impossible, but hairlessness alone does not necessarily constitute a biological unfitness, as is proved by the existence of the hairless species of African rodent, *Heterocephalus*, already referred to. And, as Gaskoin's breeding experiment shows, such a spontaneous departure is capable of being propagated. It would appear to be an abrupt, well-defined, or "discontinuous" variation—a foundation upon which a subspecies might well be raised.

The elephant is another example of a hairless mammal quite fitted, notwithstanding, to live in tropical or temperate zones, if not in Arctic regions like its hairy predecessor, the mammoth (*Elephas primigenius*).

REMARKS.

According to the theory of Lamarck (1744-1829) the inheritance of acquired characters is the basis of the variations which underlie the whole of evolution. In any such discussion it is necessary to agree at the outset as to what is to be understood by acquisition and what by inheritance. The secondary sexual characters, for example, which appear at maturity, though technically spoken of as acquired, do not properly come within the Lamarckian category. For although they are acquired in the sense that they are not congenital, they are innate characters of which the appearance is only delayed. Certain of these external sexual characters are dependent upon the equally delayed function of the sexual glands, others are not. These two classes of sexual characters should be distinguished by different names, for it is obvious that those which may be cut out by castration or oöphorectomy have a different genesis, and therefore a different significance, from those that arise at maturity independently of the function of the sexual glands. This may be illustrated by the case of a sexually precocious chick which I have already described in the Pathological Section of the third volume of these *Proceedings* (p. 55). In this bird the sexual precocity was accompanied with a marked development of the comb; but no spurs, and none of the masculine sickle feathers in the tail had at that time appeared. The latter are marks of somatic maturity in the male as distinguished from the sexual. In the testicles of this chick spermatogenesis was in full progress. In harmony with this observation is the fact that after castration of the immature bird, the comb fails to grow, but the spurs and a certain number of the sickle feathers in the tail appear regularly in the capon. Even if castration is carried out in the adult cock, a retrogression of the comb is at times to be observed. The spurs are acquired independently of the function of the sexual glands, yet as they do not grow in the normal *hen* they must be regarded as sexual. The particular characters that result from the action of the internal secretion elaborated by the sexual glands, alone or in conjunction with others, might be distinguished as "hormonic," using Starling's term "hormone" as synonymous with internal secretion.

We must, for the sake of clearness, even if by an arbitrary use of words, limit the designation "acquired" to characters of which the acquisition is under control; so that if the circumstances that call forth such acquisitions are prevented from acting, the characters themselves would not appear. Weismann's category of acquired characters, like the Lamarckian, is precisely of this kind, and comprises such as might result from injury, from functional hypertrophy or atrophy, and from environment, mainly of a climatic kind.[1] It will be apparent that the essential element of the Lamarckian doctrine is, that function determines anatomical variation, as contrasted with the other, that anatomical variation precedes function. It is necessary, moreover, for the sake of completeness in this connexion, to distinguish conditions that may be acquired *in utero* as a result of intrauterine environment, &c., from those properly called congenital, since what holds good for post-natal acquisitions will hold equally true of these. The mutilations of limbs by knots of the umbilical cord or amniotic bands, the changes in the form of the articular ends of bones from intrauterine distortion, are among such ante-natal acquirements. If we merely for a moment go beyond the grosser departures which constitute the foundation of Darwinian varieties, and which, as Huxley[1] points out, may be reduced to three headings—excess of development, suppression (complete or partial), and the coalescence of parts originally distinct—if we go beyond these, and consider the evolution of *tissues*, it will become at once apparent how inadequate the Lamarckian theory is to explain the whole body of evolutionary phenomena. I may in this connexion cite from my review[2] of the article on "Transformation and Descent" with which Virchow inaugurated the first number of the *Journal of Pathology*.[3] Virchow, it may be stated, was not at that time either a believer or a disbeliever in the Darwinian doctrine. His position simply was that the genetic proof of the descent of man from a lower animal was still wanting, and that whenever we meet with theromorphism (the apparent reversion to an animal form) we have to deal merely with phases in the development of the individual. He proceeds to say that the phenomena of transformation present themselves in two aspects; either they are purely cellular—that is, the individual cells change in such a way that the type of the tissue becomes a different one—or, on the other hand, the change may consist in a new arrangement and combination of cells and tissues into organs or larger parts of the body, whilst the structure of the cells and tissues themselves remains unchanged. To cite from the review referred to: "In his paper, however, Professor Virchow does not discuss the metaplasia (or transformation) of tissues except in the individual; yet the importance of this subject in relation to the Darwinian generalization could scarcely be exaggerated." "Without such metaplasia there could have been not only no development, but no evolution." "The coarser or organological variations are reducible to the three headings of hypertrophy, atrophy, and the coalescence of parts once distinct. . . . These changes, however, will account for only a certain class or proportion of variations. . . . The great subject of histological, and therefore of functional, differentiation would be altogether left out of consideration in such a scheme." "If we take the development of the human embryo as an epitome of the evolution of the human species, the advent of Wolffian bodies, of a liver, a thyroid and other viscera, proceeds in definite order; we are not dealing with overgrowth merely, but with differentiation accompanying growth. . . . Nay, the very definition of hypertrophy excludes differentiation, for it is by general consent used to imply that the newly formed tissue shall precisely resemble the original."

---

[1] A. Weismann, "The Germ-plasm." English translation by W. N. Parker and H. Rönnfeldt.

---

[1] *Proc. Zool. Soc.*, 1880, p. 649. In this paper Huxley points out that this threefold process obtains alike in the later stages of the embryonic development of the individual vertebrate and in the ancestral development, a circumstance which creates a strong confidence in the general validity of the latter.

[2] *Brit. Med. Journ.*, 1892, i, p. 1209.

[3] *Journ. Path. and Bact.*, Edinb. and Lond., 1893, i, pp. 1-12.

Before a liver, for instance, can vary in its grosser morphology, hepatic tissue must be produced, and no increase or diminution of function can possibly affect that which has not yet come into existence. And so of the other organs, considered histologically. The fundamental origin of such variations as bring about the genesis of species must be ascribed, as Weismann maintains, to changes in the germ-cells, to alterations arising in connexion with the "determinant" particles of the germ-plasm. A single determinant may control the character of a larger or smaller group of cells of the same class. But even so, it will be obvious that the changes to which the determinants are subject must be, at least, of two kinds—qualitative and quantitative, if the genesis of new tissues is to be explained by the hypothesis, as well as their mere numerical variation.

That somatogenic characters acquired after birth cannot be directly transmitted, will not be disputed; the question at issue is whether the germ-plasm can be indirectly modified by these in such a way as to render them transmissible through the sexual cell. Weismann is constrained to acknowledge considerable difficulties in regard to climatic influence, as illustrated by the climatic variation in certain butterflies. He would suppose, by way of explanation, that the modifying influence of climate here affects not only body cells, but coincidently the germplasm of the reproductive cells. He rejects Darwin's theory of pangenesis, not only because it supposes that gemmules are given off and circulate through the body, but mainly because it implies an addition of gemmules—i.e., of primary constituents—to the germ-plasm of the germ-cells. The biochemical possibilities entering into the problem are unnoticed in his argument; few, if any, experimental data bearing on such a possibility were at that time extant. Yet, as Dr. Mott has urged, it is by no means incredible that the germ-plasm *might* be reached from the body cells, and so influenced, through the medium of a biochemical process, in a way somewhat the converse of that in which the somatic characters of puberty result from the activity of the sexual cells, even with the full allowance that this action takes place indirectly by the formation of an internal secretion elaborated by other cells as an accompaniment of spermatogenesis or ovulation. So far as *functional* hypertrophy and *functional* atrophy are concerned, nevertheless, it seems to me that no post-natal numerical increase of somatic cells already developed, could, even in this subtle way, affect the germ-plasm, since such a numerical increase would not lead to the establishment of any new biochemical process, although in the case of climatic environment the event might be otherwise. For as regards plants, it has long been known that a change of "habit" may be brought about by a change of climatic conditions, and Klebs[1] has succeeded in producing changes which have been transmitted, although it cannot yet be said for how many generations these will hold. The sole method of determining the question at issue is, of course, that of observation and experiment, and it is as a small contribution in this direction that the anatomical investigations described in the present paper are brought forward.

As to the non-transmission of the negative results ensuing from disuse and mutilation, Paley (1743-1805), in discussing the evolutionary schemes of his time, has long ago written: "The mammæ of the male have not vanished by inusitation, nec curtorum per multa saecula Judaeorum propagini deest praeputium."[2] Even the famous case of the blind fish obtained by the *Challenger* expedition[3] is, as I have long been accustomed to point out, by no means an indisputable instance of the atrophy and disappearance of a part from such a simple process as mere disuse—from the want of penetration of light to the depths at which the fish live. For, in the first place, it is only in certain of such deep-sea fish that the eye is absent; others are furnished with particularly large eyes. And, in the next place, such blind fish are provided with phosphorescent or luminous organs, which are not produced at the site of the abortive eye, and the function of which organs seems, in some way, to replace that of the eye, in enabling the fish to obtain its food, possibly by attracting prey. The development of one organ is accompanied with the disappearance of the other, and the phenomenon may become, thus, an example of co-ordinated or correlated development, of the same class as the disappearance of the gills of the tadpole, which proceeds with the growth of the lungs. Günther (loc. cit.) observes that light-producing organs are very generally distributed in the abyssal fauna, and that those parts of the ocean in which phosphorescent animals are abundant must be sufficiently illuminated to enable such as are provided with well-developed eyes to perceive objects around.

---

[1] Croonian Lecture, *Proc. Roy. Soc. Lond.*, Ser. B, 1910, lxxxii, p. 547.

[2] "Natural Theology," chap. xxiii.

[3] *Challenger* Expedition Report. Deep-sea Fishes, by Dr. Günther.

It is, however, with the results of increased exercise that Lamarckism more particularly deals, and with this I may briefly conclude.

In the case of the camel, then, the callosities which form at the various spots on which the animal rests, are matched in kind, for example, by that produced in an untreated talipes varus, where a circular cushion with thickened epidermis forms in front, and to the outer side, of the proper heel as a result of the intermittent pressure to which the part is submitted in walking; similar callosities have at times been seen on the knees of infants who for different reasons are unable to stand, and who, in consequence, progress upon the bended knees. The callosities of the camel are strictly acquired by increased use during the lifetime of each individual. Yet all that is transmitted is the capacity to acquire them under the action of the same external conditions—not the structures themselves, as the observations recorded in the present paper demonstrate.

Precisely the same is true of the carpal callosity which is acquired in the giraffe by the animal dropping upon the flexed joint in order to gain the recumbent position. At the time of birth, the carpus is covered with hair even more abundantly than elsewhere, and there is no trace of epidermal thickening.

In this respect there is a pronounced difference between the callosities of the camel and those of the baboon and other monkeys. The areas concerned are, in the first, hairy at the time of birth; in the baboon they are, even microscopically, hairless. In neither case is there any epidermal thickening. It is clear from this that the structures fall into different categories, and that they cannot both have been similarly acquired. And seeing that the hairless callosity in the camel obviously *is* acquired as a result of pressure and friction, we may conclude that the hairless ischial area in the monkey has *not* been. The reasoning will apply equally to the hairlessness of the palm and sole of the monkey, which, of course, are as devoid of hair in the fœtus as they are throughout the rest of life. The marked differentiation in thickness of the nails in the fœtus may be adduced as a proof that epidermal thickenings *may* be ante-natal, for it answers the objection which may be urged, that all such are accompaniments of post-natal growth as distinguished from intrauterine development. And the fact still further increases the difficulties of the Lamarckian doctrine; for if the ungual thickenings have been ancestrally acquired by the increased use of the epidermis on the backs and apices of the terminal phalanges (from scratching, &c.), how is it that these are congenitally present, whilst the thickening on the ischial area is not?

The hairless gluteal integument overlying a local thickening of subjacent fat, and the latter of an unusually fibrous character, is comparable, in some ways, to that of the palm and sole. Yet it differs from these in the simplicity of its structure. It is sometimes supposed that the epidermis of the palm is thicker at the time of birth than that, say, on the dorsum of the hand. This is true only with reservation. The essential feature of the palmar and plantar areas is their papillary complexity, and associated with this, their increased tactile sensibility. The increase in thickness of the epidermis is due to the filling in and levelling of the interpapillary spaces; on the summits of the papillæ the epidermis is no thicker than it is on the dorsum of the hand. This is illustrated in the accompanying microphotographs, which show vertical sections of the skin of the palm and of the dorsum of the hand from a fœtal Macacus at full term, from the same animal, in fact, as that from which the sections of the ischial "callosity" previously described, were prepared. A proper thickening may afterwards result in correspondence with the degree of use to which the structure is put. And so in the case of the naked area over the ischial tuberosity.

What the significance of the thinly skinned hairless gluteal districts may be, can only be guessed at. It may be pointed out that the hairless area is not strictly limited to the callosity itself—i.e., to the site of the horny thickening acquired after birth. In the fine example of Anubis Baboon now in the Gardens of the Zoological Society, the hairless condition extends considerably beyond the proper callosity, and beyond the parts actually in contact with the ground when the animal is sitting. In the Mandrill, likewise (Zoological Gardens, 1911), there is a widespread, thinly piliferous area, red and blue in colour, over the whole buttock, the extent of which exceeds that of the proper callosities.

Specialized glandular areas, though not gluteal in position, are of somewhat wide occurrence amongst antelopes. In some cases the glandular area is piliferous, in others bare, and in yet a third set there

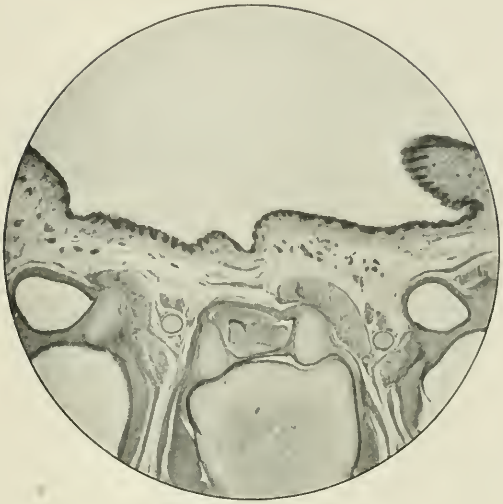


FIG. 9.

A vertical section of the palm of a newborn Macacus. The chief feature of the skin is its papillary complexity. The thickening of the epidermis results from the filling in and levelling of the interpapillary spaces. On the summits of the papillæ the epidermis is no thicker than it is on the dorsum of the hand.

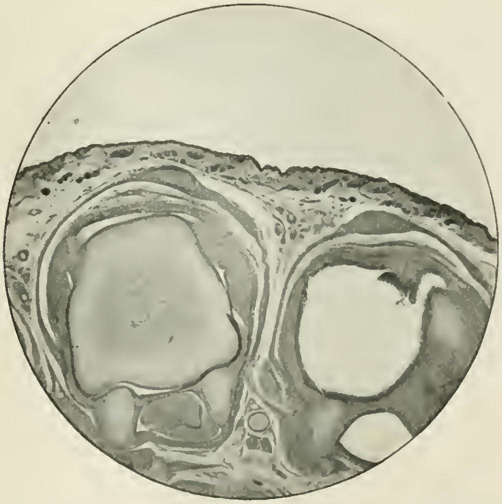


FIG. 10.

A vertical section of the dorsum of the hand of the same newborn Macacus. As contrasted with the palm, the papillary development is notably less, but the thickness of the epidermis is as great as it is over the *summits* of the palmar papillæ.

is a thinly skinned naked area around which the glandular zone lies.[1] They are found in diverse parts of the body: on the head, the trunk, or limbs. The well-known congenitally hairless foci on the inner side of the forelimbs of Equidæ fall into the same category. The naked ischial districts, themselves glandless, are possibly, then, vestiges of the third of the conformations just referred to, and perhaps sexual in their function. Teleologically there is no reason why the ischial callosity should be formed on histologically hairless cutis rather than on hairy; and as an exceptional occurrence, the callosity may be acquired in monkeys in which the naked area is normally wanting. Neither the one nor the other is present in the normal anthropoid. The chimpanzee in the sitting attitude rests with the buttocks not on the ground, but touching the upper side of the heels, like the coloured races of mankind. The exceptional case I may refer to, is that of the Orang at present in the Gardens of the Zoological Society. This particular animal presents two callosities, but they are not strictly normal in position, and the explanation of their unusual formation must be that the bones are misshapen and their relations disturbed; for Mr. Pocock tells me that the animal, when young, was extremely ricketty. Here, of course, the callosities have formed on piliferous skin, the hair of which has been worn down.

To sum up: the conclusions of the foregoing considerations would be that the microscopically hairless condition of the gluteal skin in the monkey at birth is not attributable to the transmission of a character ancestrally acquired as a result of pressure and friction, and that the proper callosity being acquired from these causes, is not present in the newborn, but has to be again acquired.

---

[1] R. I. Pocock, "On the Specialized Cutaneous Glands of Ruminants," *Proc. Zool. Soc.*, 1910.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FOURTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1910-11

SURGICAL SECTION

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1911

Surgical Section.

OFFICERS FOR THE SESSION 1910-11.

President—

Rickman J. Godlee, M.S.

Vice-Presidents—

C. T. Dent, M.C.
G. H. Makins, C.B., F.R.C.S.

Hon. Secretaries—

Jonathan Hutchinson, F.R.C.S.
L. A. Dunn, M.S.

Other Members of Council—

C. A. Ballance, M.V.O., M.S.
J. Berry, F.R.C.S.
J. Bland-Sutton, F.R.C.S.
W. H. Jessop, F.R.C.S.
Ernest Lane, F.R.C.S.
B. G. A. Moynihan, M.S.
Stephen Paget, F.R.C.S.
W. G. Spencer, M.S.
Robert Jones, F.R.C.S.Ed.
Herbert F. Waterhouse, C.M.

Representative on Library Committee—

J. Berry, F.R.C.S.

Representative on Editorial Committee—

Stephen Paget, F.R.C.S.

SURGICAL SECTION.

CONTENTS.

November 8, 1910.

PAGE

On Fractures in the Neighbourhood of Joints. A Discussion, opened by Robert Jones, Ch.M. 1

Mr. Rushton Parker (p. 23)—Mr. Whitelocke (p. 26)—Mr. Richard Warren (p. 27)—Mr. Robert Jones (p. 30).

November 29, 1910.

Discussion on the Treatment of Fractures in the Neighbourhood of Joints (*continued*)—

Mr. Herbert J. Paterson (p. 31)—Mr. C. H. Fagge (p. 34)—Mr. Clayton-Greene (p. 36)—Mr. H. P. Symonds (p. 38)—Mr. A. J. Walton (p. 39)—Mr. A. E. Johnson (p. 42)—Mr. Rickman J. Godlee (p. 43)—Mr. Robert Jones (reply) (p. 44).

December 13, 1910.

Septicæmia Hæmorrhagica. Severe Capillary and Parenchymatous Hæmorrhages of Septic Origin. By W. G. Spencer, M.S. 47

Gastric and Intestinal Hæmorrhages (probably of Septic Origin) occurring after Abdominal Operations. By Jonathan Hutchinson, F.R.C.S. 58

Transverse Fractures of the Patella, without Separation of the Fragments. By A. E. Johnson, F.R.C.S. 69

January 10, 1911.

The Delimitation of the Rectum and its Subdivisions. By F. Wood Jones, B.S. 85

The Operative Treatment of Cancer of the Rectum. A Discussion, opened by F. Swinford Edwards, F.R.C.S. 99

Mr. Harrison Cripps (p. 105)—Sir Frederic Eve (p. 110)—Mr. H. F. Waterhouse (p. 112).

January 18, 1911.

PAGE

A Discussion on the Operative Treatment of Cancer of the Rectum (*continued*)
Mr. A. Boyce Barrow (p. 115)—Mr. Sampson Handley (p. 118)—Mr. F. C. Wallis (p. 122)—Mr. P. Lockhart Mummery (p. 125)—Mr. C. H. Fagge (p. 128)—Mr. Gordon Watson (p. 129)—Mr. Rickman J. Godlee (p. 131)—Mr. Swinford Edwards (reply) (p. 131).

March 14, 1911.

Traumatic Myositis Ossificans. By G. H. Makins, C.B., F.R.C.S.... 133

The Formation of Bone in Periosteum separated by Injury. By Anthony A. Bowlby, C.M.G., F.R.C.S. 142

Myositis Ossificans Traumatica. By Rickman John Godlee, M.S. 144

Large Congenital Diverticulum of the Bladder treated by Resection. By James Berry, F.R.C.S. 158

The Inguinal Method of Radical Cure for Femoral Hernia. By C. H. Fagge, M.S. 165

May 9, 1911.

Demonstration of Cases of Cleft Palate... 169

The Treatment of Cleft Palate. An Address Introductory to a Discussion on the Subject. By W. Arbuthnot Lane, M.S. 169

Fifty-six Cases of Cleft Palate treated by Operation, with Special Reference to the various Methods of Operation, and the After-results. By Johan Ulrich, M.D. (Copenhagen) 173

Discussion: Mr. Douglas Drew (p. 180)—Mr. C. H. Fagge (p. 181)—Mr. James Berry (p. 182)—Mr. G. E. Waugh (p. 186)—Mr. F. F. Burghard (p. 188)—Mr. T. H. Kellock (p. 189)—Mr. L. E. Barrington Ward (p. 191)—Mr. W. G. Spencer (p. 197)—Mr. Lane (reply) (p. 198).

June 13, 1911.

On the Division of the Posterior Spinal Nerve-roots: (I) for Pain, (II) for Visceral Crises, (III) for Spasm. A Discussion, opened by Ernest W. Hey Groves, M.S. 199

Resection of the Posterior Spinal Nerve-roots in the Treatment of Gastric Crises and Spastic Paralysis. By Professor Foerster (Breslau) 226

Discussion:—Mr. Rickman J. Godlee (p. 247)—Dr. Otto May (p. 247)—Mr. C. A. Ballance, M.V.O. (p. 248)—Dr. Purves Stewart (p. 251)—Mr. D. M. Aitken (p. 252)—Dr. Hinds Howell (p. 253)—Mr. Rickman J. Godlee (p. 254), Professor Foerster (reply) (p. 254).

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

John Bale, Sons & Danielsson, Ltd., 83-91, Great Titchfield Street, London, W.

Surgical Section.

November 8, 1910.

Mr. Warrington Haward in the Chair.

On Fractures in the Neighbourhood of Joints.

An Address Introductory to a Discussion on the Subject.

By Robert Jones, Ch.M.

The subject selected for this discussion relates to the management of certain fractures in the immediate neighbourhood of joints. In opening the discussion, my remarks will be of a strictly clinical nature, and will be based very largely on personal experience and practice. It is a common observation that, until quite recently, the subject of fractures has remained obscured by reason of the rapid progress made in other departments of our art and the attraction they prove to our most able colleagues. Indeed, it is scarcely an exaggeration to say that in many of our hospitals the treatment of a broken bone is left from first to last in the hands of a junior houseman. Such delegation of responsibility is often doubtless justified, but I am afraid the house surgeon himself sometimes feels that he is dealing with a comparatively unimportant branch of his art—a feeling engendered by the seeming indifference of his chief.

It is due largely to the brilliant and earnest work of Arbuthnot Lane that our attention has been re-awakened in this subject, and, whether we are in agreement with him or not, we must feel that his strenuous advocacy of his methods has been productive of much that is good.

Every general practitioner is supposed to be able to treat a fracture, but there is now a tendency for our students to be taught to rely too much upon a radiograph for diagnosis; the result of this must mean disaster, and will in some measure account for many stiff and useless joints which so often confront us. The correct interpretation of an

X-ray photograph amply repays study, but it involves a long and patient education. The radiograph should be used as a court of appeal only after a diagnosis has been attempted, and should not be made to deprive the student of his power of clinical observation. I would prefer to have a bone skilfully "plated" in any part of my body by a truly aseptic surgeon rather than that it should be left in the hands of a surgeon, however able, who would treat it in direct violation of mechanical and physiological principles. There really should not exist two schools—one operative and the other mechanical. Each surgeon should decide for himself whether in a particular case an operation should be performed or not; and the way a successful result will be achieved will depend largely upon his special ability. The word "unjustifiable" should be rigorously excluded from our vocabulary when we deal with practices which differ from our own.

During the last twenty-five years I have treated an unusually large number of fractures involving the function of joints, a goodly proportion of which were not recent and involved disability or deformity, or both. The mere appearance of a joint does not seriously distress a patient; disability invariably does. From the patient's point of view, impairment of function is a much more serious deformity than any mere outward change of shape.

I regard every case of fracture as the potential cause of disability, and am still learning from my own mistakes (and sometimes from those of others) certain fundamental rules for clinical guidance.

It is necessary, for instance, that we should recognize the importance of Wolff's law, "that the internal structure of bone, and then its outward appearance, are adapted to the strains to which it is subjected and to the function it performs, and that the structure varies to meet alterations both in strain and function." Normal bone, in fact, is structurally fortified at that point where most work is required of it. By deflecting strain to another part, the structure is correspondingly strengthened there and weakened where it is no longer wanted. The "pathogenesis of deformities is therefore functional." It is therefore necessary in all fractures about joints clearly to appreciate the mechanical function of the joint. What is required is a knowledge not so much of its mere anatomy as of its movements and the directions of the normal lines of strain during muscular exercise.

Again, a study should be made of the common disabilities which accompany so-called neglected fractures about joints. In fractures about the shoulder, they are deficient abduction and rotation; in the hip, diminished internal rotation, abduction, and often extension; in the elbow, fixation at an angle usually of about 130°; in the knee, genu valgum and sometimes flexion. The special deformities or disabilities peculiar to each injury should be studied, anticipated, and avoided.

In case some impairment of function should be feared, we should know what particular movement and position of the joint it is most important to preserve, and we should direct the treatment accordingly. As an example, I will take the case of a fractured scaphoid. This accident, as we know, occurs very much in the same way as a Colles's fracture. The neck of the scaphoid is usually fractured at its narrow part by a cross-breaking strain due to a fall on the hand which is held more pronated than when a Colles's fracture is produced. The diagnosis even in an early stage is not difficult. Added to the general signs of a sprained wrist there is marked tenderness to slight pressure in the anatomical snuff-box. If such a case is seen early and the wrist be hyper-extended, and be kept in that position for about fourteen days, an excellent functional result may be assured. If, on the contrary, it be sent to a masseur with a bias towards strenuous passive movement, a thickened wrist with marked limitation in movement will probably ensue. To those of us who have seen old cases of fracture of the scaphoid the explanation is simple. The main disability consists in a loss of the power of extension. We therefore at once secure our right of way by hyper-extending and fixing the wrist in hyper-extension. The displacement usually occurs during some movement of the hand, and the vigorous masseur often produces displacement of the fragment and traumatic arthritis by exercising his art injudiciously. Again, in the old neglected case of fractured scaphoid or other carpal bone, where the patient appears with a stiff and somewhat flexed wrist, I often apply a club-foot wrench and fix the hand in hyper-extension. This restores to the patient his firm power of grip which we all lose when our wrist is flexed. I shall have a word or two to say later on the operative treatment of fracture of the scaphoid, but for the moment I am illustrating the value of obviating disability by the anticipation of subsequent events.

Again, as the result of long experience, I have formed very definite opinions as to the relative value of passive and active movements, and as these are not quite in accord with prevailing views, I will explain them later.

Finally, mere æsthetic considerations of appearance should always be subordinate to questions of restoration of function. In recognition of this principle I always endeavour to preserve a clear right of way, free from obstruction by fragments of bone and callus, so that movements of the joint are not impeded, and, furthermore, to preserve the alignment of bones so that the lines of stress shall fall normally across the joint.

In teaching I constantly emphasize the important rôle of a correct alignment, particularly in the case of the lower limbs. At the ankle-joint it is essential to a good recovery. In fractures about the condyle of the femur it is almost of equal concern. Any deviation from correct alignment means alteration in structure in accordance with Wolff's law —an effort made by Nature to adapt the parts to the deviated lines of muscular stress, or to the deviated lines of the force of gravity. These deviations may produce changes identical with those of traumatic osteo-arthritis. A faulty alignment, whether there be end-to-end apposition or not, means erroneous deflection of body weight, a cross straining of the fracture, and a stretching of certain structures in the joints which lie above and below the fracture.

I shall scarcely refer to the question of splints. The splint is only a small practical detail compared with the method in which it is used. Inflexible fixation by wire or plate, fixation by splint and bandage, or no fixation, may each be simply good or bad. A surgeon may succeed or fail in any or all of the three methods, according as he has, or lacks, the necessary knowledge and skill. If he have not a clear idea of the precise mechanical effect he wishes to obtain, neither splints, plates, nor massage will avail him.

Coming to definite injuries, I will briefly allude to the so-called fracture of the anatomical neck of the humerus, which most often occurs in old people. It is not always a genuine fracture through the anatomical neck, but includes both tuberosities in the upper fragment. It is very often impacted and never requires more by way of treatment than is afforded by a sling and a modest axillary pad. Gentle rubbing may be continued from the time of the accident, but no passive movements should be permitted within less than from two to three weeks. At the end of six weeks the patient usually recovers with an excellent range of movement—a range which allows him to button the back of his collar and reach his brace from behind.

Fracture of the surgical neck requires more care. The head is usually rotated upwards and outwards by muscular action, and if there be much displacement a pull in the abducted direction will elicit crepitus. The ends should be manipulated under extension until a sufficient interlocking occurs so that the head responds to every movement at the elbow. When this is secured the elbow should be pushed towards the shoulder so as to compress the fractured ends, and the arm should be brought down to the side and there fixed for a few days by pad and bandage. At the end of a week it will be found that the spasm of the scapular muscles has passed off and treatment can be continued, as in the higher fracture, by pad, sling, and bandage.

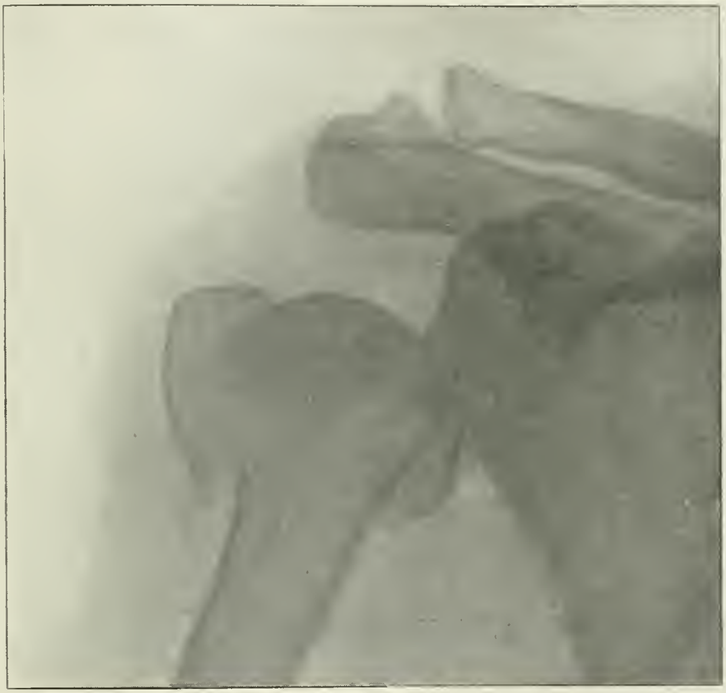


FIG. 1.

Subglenoid dislocation of the humerus, with separation of greater tuberosity to outer and lesser to inner side.

One of the most common complications of fractures of the upper humerus is dislocation of the head. I have no fewer than forty X-ray photographs of this condition (fig. 1). I have had the opportunity of seeing the case immediately at the time of accident on several occasions, but more frequently I have not seen them till a later stage. Manipulation should be tried first while the structures are relaxed, and if this fail the arm should be extended perpendicularly while the surgeon manipulates the head. This manœuvre I have found the most successful, and I will show you on the screen the X-ray photographs of the range of movement which followed a reduction by this method, where I was assisted by my colleague (Mr. Newbolt). The patient was an old gentleman, aged 74, a huge, muscular man, and he now possesses an extremely useful shoulder. In a recent case failure to reduce by manipulation should be followed by open operation to replace loose ends. An excellent device to secure this object is that of McBurney, who introduces a hook through a drill-hole in the lower end of the dislocated head, whereby, with but little handling of the parts, reduction is effected. The removal of the fragment is always indicated in old cases where nerve or artery is involved. I have been most interested to note the free range of movement which often follows these cases even when neglected. Good rotation inwards and fair abduction have been present in spite of the great deformity.

Fractures about the elbow-joint in children are the source of much trouble to the practitioner, and we have all seen cases of fractures through or above the condyles in which stiffness and limitation of movement have resulted in spite of the most assiduous passive movements begun early and continued late. Medical men have often consulted me with almost the identical story. They say: "For the first week I treated this supracondylar fracture in exactly the position you advise; the movement seemed quite good, and we have regularly applied passive movements once or twice every day and kept the arm at right angles. Instead of becoming more slack, this stiff arm is the result." They are astonished when they hear that the stiffness is probably the direct result of passive movement, and that if the arm be slung up and left alone movement will return again. A patient some years ago brought her boy to me with a stiff elbow following a fracture of the epicondyle. Her story was that six weeks previously Dr. A. had placed the child's arm in a sling, and a week later went for his holiday. The child was then transferred to Dr. B., who immediately began a vigorous campaign of passive movement. Several times a day the arm was flexed, pronated, supinated, and extended, and the ever-growing stiffness was a further stimulus to action. When I saw the child he had a stiff, painful elbow, and the mother, full of indignation, threatened Dr. A. with penalties. Dr. B., who was really the unintentional offender, received nothing but praise for his energetic care. I explained to her that both men had done their best and that a third stage of treatment was still needed. The arm was slung up at an acute angle for a fortnight and gradually dropped to a right angle, when active, not passive, movements were allowed, and in less than a month a very considerable range was obtained.

I will now for a moment explain my views as to the erroneous teaching in regard to passive movements. While nothing but good results from early and gentle massage in recent fractures, provided it be applied without moving the parts, great harm often results from combining passive movements with massage at a very early stage of treatment. I will illustrate this by a case of supracondylar fracture. Such a case, treated either with an angular splint or in a straight position, very often results in limited movement confined in range from the right angle to midway to extension. This is due to faulty alignment, to the obstructive effect of excessive callus exudation in front of the elbow, or to a projection there of the upper fragment of the humerus. The object of the surgeon being to prevent this, he corrects the faulty alignment by acutely flexing the elbow and at the same time secures a right of way before he is opposed by callus exudation. To do this he supinates, then acutely flexes, the elbow. For some days it is left in this position and then transferred to a sling, the wrist being kept immediately below the chin. At the end of a fortnight the wrist is dropped 2 in. or 3 in., and the patient is allowed to move the wrist from its resting place back to the chin. If this can be done, it may be lowered still further; if a few days later, however, it has stiffened in its new position, it requires again to be slung in the more acutely flexed position. At the end of three weeks passive movements may be begun. They should consist of only one movement in each direction (pronation, supination, flexion and partial extension), and then the arm should be replaced in a sling. This passive movement should be slowly and gently done, the fracture being protected from strain by the surgeon's hand. During the whole of this period massage can be harmlessly and even beneficially practised. Early passive movement, such as is so generally practised, is positively harmful; it disturbs the uniting fragments of bones and leads to fresh plastic effusion, excess of callus and increased formation of connective tissue about the joint, all of which are conducive to increased stiffness. The oft-repeated to-and-fro movements only do so much daily damage, and no time is given for recovery before a fresh injury is inflicted. I never move such an elbow until I am satisfied that the union is not likely to be disturbed. This is true of all joints. They should be put through each of their movements once and then left to recover from the damage. In one, two, or three days each movement is again repeated, but only once. Passive movements thus applied are comparatively painless and are not followed by rebellious reaction, which is always an indication for continued rest.

Since I have employed the acutely flexed position elbows have ceased to be troublesome. It is very rare to have anything but an excellent result. In the old-standing cases with disability which come to me even now the story usually relates to rectangular splints, pads, bandages, and early movement. The deformities—whether the result of supracondylar fractures, separation and rotation of condyle, or backward dislocation with fracture of the coronoid—are almost always the same. In supracondylar fracture, the prominent lower end of the shaft, assisted by new bone, blocks the way to flexion. In separation of the condyle the same kind of obstruction occurs. Callus exudates, bony displacements, and organized effusions oppose flexion, whether the arms are treated by splints in the straight or in the rectangular position.

For over twenty years I have instructed my house surgeons in all cases of fracture and dislocation about the elbow (with the exception of fracture of the olecranon, always easily diagnosed) first to supinate and then acutely flex the elbow and to fix it in this position. If this advice were generally followed disability would be a rare instead of, as it is, a frequent sequel.

An X-ray screen will demonstrate the effective reduction of the backward dislodgment of the lower fragment in supracondylar fracture (fig. 2). It has been suggested by certain writers who follow this method in separation of the condyles and supracondyloid fractures that the acutely flexed position is liable to produce a forward displacement of the epiphysis. Even if this be so, no harm can come of it, for callus exudation cannot prove obstructive beneath the triceps, and there will be no sharp prominence of the lower end of the shaft to limit flexion.

If the forearm be dislocated backwards we must extend and supinate and acutely flex, for in this way only can we reduce the dislocation; and should we be unable to flex the arm, we may rest assured by such failure that the displacement still remains. If the radius is dislocated forward and inward, a similar routine is effective, the supination and extension will reduce the luxation, while acute flexion will maintain the parts in position. If the fracture be intercondyloid, the position of acute flexion has this advantage—that it secures for the forearm at once a right of way before exudation of callus and displaced condyles can prove obstructive. It is true that in this particular injury some widening of the condyles is apt to occur when the arm is flexed. The parents should be warned of this, and a very effective way is to ask them which they prefer, an artistic-looking or a mobile elbow. This saves all future complication. Should the injury be separation of the internal epicondyle, there can be no better way to keep it from the common downward displacement than by acute flexion, which relaxes the muscles arising from it.

An interesting and common fracture is that of the head of the radius. It is usually vertical, and occurs within the orbicular ligament. As in all fractures of the upper part of the radius, there is muscular spasm on attempts at supination. There is usually no crepitus, but always distinct pain on direct pressure. In such a case, if supination is obstructed, immediate excision of the fractured piece is the best treatment. The rough edges should be trimmed, and, in order to lessen callus formation, the bare cancellous tissue should be well compressed by means of a smooth steel implement. If the fracture be below the orbicular ligament, crepitus is usually obtained, and full supination is effectively secured by the close approximation of the forearm upon the arm.

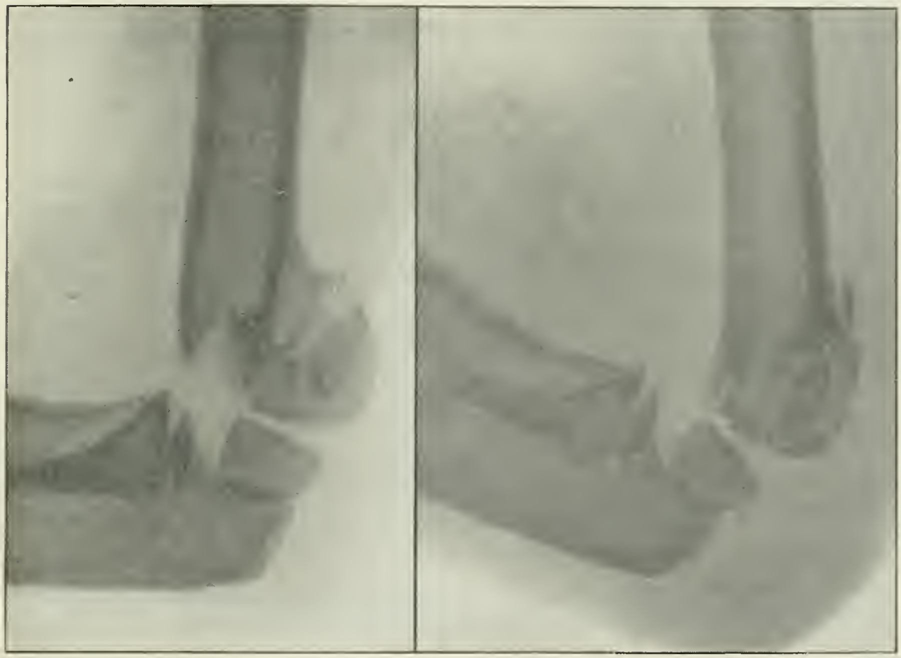


Fig. 2.

Supracondylar fracture before and after reduction by flexion of the elbow.

Fracture of the olecranon I treat by maintaining the arm in the straight position, and, if the line of fracture is transverse and the fragments well apart, I fix the fragments with screw or wire. In oblique or stellate fracture I do not operate. In fractures below the olecranon in the upper third of the ulna I have sometimes employed Lane's plates.

I have met with fractures of the coronoid on several occasions, although recent writers doubt its occurrence. Traumatic myositis ossificans followed this injury in one instance, and this ossific complication is much more frequent than reports would suggest. I have between forty and fifty radiographs of the condition, and will show you a series of four skiagrams of the same case marking the progress of the disease from the beginning to the end. I have been unsuccessful in my attack upon this disease, as in several instances after operation fresh accumulations of bone have occurred.

Before leaving the subject of injuries to the elbow, I would allude to the treatment of old cases of many weeks' or months' duration, which come with deformity and disability. The familiar gunstock deformity, if extreme, can be remedied by supracondylar osteotomy. As a rule, however, no disability accompanies this deformity, and operation can only be justified on æsthetic grounds. A far more important class is that where flexion is blocked, and both in old condylar and supracondylar fractures, and in old dislocations, very useful joints can often be secured. The patient is placed under an anæsthetic, and the arm is forcibly flexed until, in spite of all opposition, acute flexion is attained.

Mr. H. O. Thomas, twenty years ago, suggested this in the case of old dislocations of the elbow. It is equally successful in fracture dislocation. By way of illustration we will take the case of a fracture dislocation of some months' standing. We find the elbow at an obtuse angle with about twenty degrees of movement. An anæsthetic is administered, and the arm is slowly but forcibly flexed. If this can be done safely at one sitting all the better. Often, however, it is only possible to flex it about twenty or thirty degrees without risk of fracture. The arm is securely slung at the acutest angle obtainable, and in two or three days another effort is made. It is interesting to note that by that time one can quite easily flex it several more degrees. In the most obstinate cases it is not necessary to reduce in more than three stages. It is then slung up for a fortnight and gradually lowered, as I have explained before. In most unlikely cases a good range of movement often results, and it is well worthy of trial before excision of the joint is decided upon.

I have no new light to throw upon the much-discussed Colles's fracture, and can only very confidently assert my belief in the efficacy of complete reduction. Often no serious effort is made to obliterate deformity, and in no region can one conceive of a greater necessity for it; for exuberant callus must terribly handicap the tendons, both dorsal and palmar.

The text-books still describe the handshaking method of reduction with extension, flexion, and ulnar flexion. This indirect method of applying corrective force through a chain of small bones will not appeal to those of us who are in the habit of using the tubercle of his own scaphoid as a fulcrum in forcible manipulations, such as osteoclasis and manual wrenchings. I reduce a Colles's fracture in the following manner: The surgeon with his left hand, palm upwards, grasps the patient's left arm, placing the tubercle of his own scaphoid against the projecting lower end of the upper fragment; he then places his right hand, palm downwards, with the tuberosity of the scaphoid on the upper edge of the lower fragment, when, by a forcible pressure, the fragments are powerfully replaced in position and crepitus is often felt. If the reduction be complete the patient can at once move his fingers with less difficulty than before. In old cases of many weeks' standing, when deformity, marked disability, and sometimes pain exist, I place a Thomas's wrench on the lower end of the radius and forcibly reduce the deformity—usually without difficulty. Daily massage, splints for fourteen days, and passive movements later, of the kind already described, are the indications in these old neglected cases. In the recent case, early massage, fixation for three weeks, then passive movements, will always result in a mobile hand and wrist. I now experience no difficulties with my cases of Colles's fracture when I treat them in the first instance. The chief cause of stiffness of fingers and wrist is the unreduced backward displacement of the lower fragment, which impedes the action of the extensor tendons, while the same displacement interferes with full supination.

I have already referred to fractures of the scaphoid. The method of reduction I adopt is to flex the wrist acutely, and place firm pressure on the dorsal fragment, then completely hyper-extend the joint. If this effect cannot be attained, the prominent piece, or even the whole bone, should be excised. The excision is much more successful if performed immediately rather than later. In the old neglected case, where the power of grasp is diminished because extension at the wrist is limited, the wrist should be fixed in hyper-extension, even, if necessary, by the aid of the wrench.

LOWER EXTREMITIES.

The Hip.

I will only deal with two types of fracture connected with the hip, the one associated with the young and the other with the old. The adolescent fracture is much more common than is generally appreciated, and is productive of the deformity known as traumatic coxa vara. It consists of a partial or complete separation at the epiphysis of the head, a fracture through the neck, or a fracture involving both neck and epiphysis. I will describe for clinical purposes a typical case of separation, or partial separation, due to a comparatively slight strain. It is of all injuries to the hip the most often overlooked even by extremely good surgeons; it is furthermore often a serious matter for both the patient and surgeon if an early diagnosis be not made. A boy of perhaps 12 or 14, while playing football, attempts to turn suddenly, and feels a sharp pain and a disability in his hip. He hobbles off the field, and, after resting a short time, walks back to school and rests. The surgeon is sent for, and makes his examination. He finds the movements fairly free in the direction of flexion, extension, and adduction; the rotations are distinctly painful, and abduction beyond a short range is not tolerated. The measurements of the limb are normal. Ten days or a fortnight's enforced idleness, and the little patient is back to the playground, when simple games are proceeded with. In days or weeks another strain occurs and further disability, with symptoms no more diagnostic than before. Games are prohibited for the rest of the term, and one day something gives in the hip, and he has to be helped to his bed. There may be now a half or three-quarter inch of shortening, and on examination a separation of the epiphysis, partial or complete, will be found. The case I have given is one I am very familiar with, and it is generally diagnosed as a sprain or as a synovitis. The X-ray photograph (fig. 3) shows the femoral head in position and the femoral neck travelling upwards.

In another type the injury is much more severe, the disability more pronounced and lasting, and we find we are dealing either with a fracture of the neck or with a complete separation of the head. There is a third type which completes the clinical group. A youth slightly injures his hip, and is incommoded, hardly disabled, for a day or two. As the weeks pass he complains of an increasing lameness and stiffness at the hip. His symptoms are very much those of a starting hip affection. There is limitation of movement in all directions, shortening of the limb, elevation of the trochanter above Nélaton's line. A skiagram shows a coxa vara of the epiphysial type. The slight injury has partly torn the periosteum and separated the epiphysis, and under the strain of body weight the characteristic deformity occurs.

(*a*) From the clinical side, I would suggest that a single strain or a succession of strains of the hip-joint in adolescents, if followed by disability and pain on abduction, denotes an epiphysial separation.

(*b*) If strain completely disables the patient, and is accompanied by elevation of the trochanter, a fracture of the neck is to be diagnosed.

(*c*) If a slight injury be followed by a limp, rigidity and shortening of the limb, we are probably dealing with a coxa vara following a partial epiphysial separation, but exhibiting signs of traumatic epiphysitis.

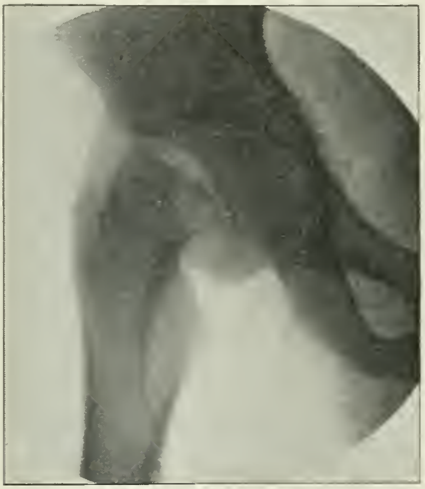


Fig. 3.
Separation of epiphysis.

We must remember that a neglected traumatic coxa vara results in a stiff, adducted, and slightly flexed hip. The treatment should consist of immediate fixation, with extension and extreme abduction. By this means the sliding head or fractured neck is placed in its proper relation with the shaft. Under an anæsthetic the pelvis is fixed while the limb is abducted, so that the upper border of the acetabulum acts as a fulcrum. The patient is then fixed in plaster or, as I prefer, in an abduction frame until consolidation has occurred—probably for about six weeks.

Lest we have to deal with a softened femoral neck, a simple calliper splint, which I will show later, should be worn for some time, so devised and fitted that no strain upon the yielding femur is allowed while the patient walks.

In fracture of the neck of the femur in the old, several problems confront us, more especially in regard to nursing and in regard to the dangers of pulmonary complications. I am convinced, however, that quite a large proportion of cases, under appropriate mechanical control, make excellent recoveries with but little shortening and comparatively negligible disability.

An interesting question arises in cases of impacted fracture of the neck. Owing to the fact that there may be no shortening, practitioners sometimes hesitate to diagnose a fracture. The presence of external rotation and tenderness about the neck of the femur is sufficient, to my mind, to determine a diagnosis.

Opinion varies as to the propriety of disimpacting the fracture in these cases. I make a practice of disimpacting in every case where there is any shortening or external rotation of the limb, leaving only those cases in which there is no rotation and little or no shortening. It must be remembered that a patient with an impacted fracture can often lift the limb from the bed, though with more or less pain, and sometimes a correct diagnosis is only arrived at when spontaneous disimpaction occurs two or three weeks after the injury, and a consultation is called for. It is necessary to emphasize this statement, as most authors insist that in no case of fracture of the neck can the limb be lifted from the bed. Cotton, in the latest work published, states: "Uniformly there is inability to raise the leg from the bed," but in a footnote he adds, "I have seen one exception to this rule."

Provided a case is one suited for complete mechanical treatment—i.e., that there exists no condition which renders the recumbent position inadvisable—I endeavour to secure immobilization and apposition. This can only be done by fixation of the limb in the position of abduction, with fixed rather than elastic traction. With elastic traction, such as the weight and pulley, there must be a sliding of fragments one upon the other when the muscles relax in sleep and on awakening. Liston's long splint and perineal bandage was an attempt to keep the joint at rest. Extension by weight and pulley sought the same end. Fixation by plaster is an advance on either of these plans, but has obvious disadvantages. The splint we use in Liverpool is a modification of the double Thomas's frame, so devised that abduction can be maintained at any angle. The illustration shows the splint in use (fig. 4). By its use starting pains never occur, for the pelvis, lower extremity, and splint are all firmly fixed together, and movements of the patient do not alter the tension in relation to the fracture. The patient and the frame are lifted together without in any way disturbing the fracture, and both the frame and patient can be carried together without divorcing the one from the other. This rigidity at the seat of fracture favours repair with a minimum of friction, and thus frees the joint from embarrassing obstacles. In six weeks a Thomas's knee bed-splint is applied with extensions, and passive movements on the lines already indicated are adopted, safeguarded by the splint against a slipping upwards of the femur. A fortnight later and the patient walks in a calliper, so that walking is permitted with no weight-bearing strain on the fracture. During the whole of this time massage is assiduously practiced. In those cases where hypostatic congestion is threatened the patient is placed in a Thomas's knee-splint with the limb abducted and is allowed to be propped up, if necessary even in the sitting position. In old cases of fracture with very little movement and no signs of traumatic arthritis adhesions may be safely broken down under

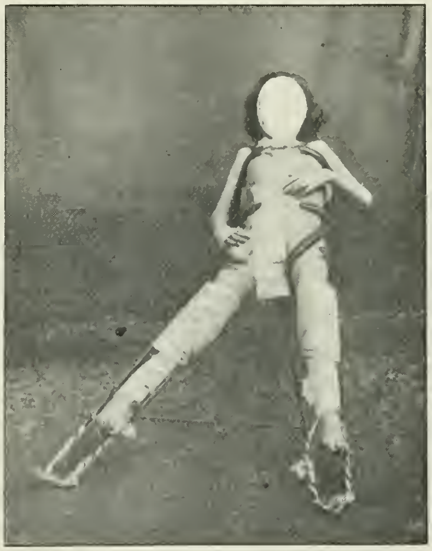


Fig. 4.

Thomas's abduction frame with extension and counter-extension by groin-strap.

anæsthesia and mobility restored by massage. The greatest care should be exercised in the manipulations. By these methods patients are enabled to walk early without in any way interfering harmfully with the fractured neck. Split fractures of the shaft of the femur involving the trochanter may need a couple of screws, but nailing the trochanter to the neck in fractures of the aged is undesirable, and many surgeons who have enthusiastically advocated this method are now abandoning it. Good abduction makes for correct alignment, and the trabeculæ of bone laid down in the callus as it becomes hard will follow the normal lines in response to the normal strains placed on the fracture.

The Knee-joint.

I treat fractures of the lower end of the femur by means of traction in the line of the limb. This, in my opinion, is best secured by means of a Thomas's knee-splint. The backward and downward rotation of the lower fragment, due to contraction of the gastrocnemius, is of slight importance compared with the effect of upward traction of the quadriceps and hamstrings. This is a doctrine I have taught for many years, and I am pleased to find my opinion confirmed by Cotton in his work just published. The overlapping which we often find depicted by the Röntgen screen is a sliding of the lower fragment in front of the upper fragment. This can always be rectified by powerful traction. From the classical description we would infer that the upper fragment invariably lies in front of the rotated lower fragment, but this is not true in every case. The use of a double-inclined plane to neutralize the action of the gastrocnemius is based on a theory which is not found to be true in practice. Our procedure in femoral osteotomy is suggestive in this connexion. In the many thousands of these operations performed, how few surgeons have ever resorted to the double-inclined plane! Energetic traction to overcome shortening is the secret of a successful result. The spasm of muscle in any part of the body is very transient and readily yields to pressure and extension. When functional disability results, it is usually due to the interference with movements of the patella by the *forward* displacement of the upper fragment.

If there exists as a complication a condyloid separation, the displacement may be manually rectified by extension, aided by lateral pressure. I have never found it necessary to fix the fragments by nail or screw, although there can of course be no objection to this practice. A surgeon obviously must attain his best results by methods he trusts the most. Two conditions, however, are essential to success: (1) freedom should be secured for movements of the knee, and (2) a correct alignment of hip, knee, and ankle.

Fracture of the internal tuberosity of the tibia is very apt to result in a genu valgum, unless a perfect reduction is made. The sartorius, semitendinosus, gracilis, and the internal lateral ligament of the knee run across and support the fragment. If the knee be kept straight and these structures be kept tense, they form a most excellent internal splint. The displacement is usually slightly backwards. I have treated a large number of these cases, and the results are generally quite good, although sometimes there remains a weakness of the internal lateral ligament. The fracture may be caused by direct or indirect trauma; indeed, it is not unusual to meet with cases where the injury has occurred solely from lateral strain. If within the first few days one is unable to secure a level intra-articular fracture line, open replacement with nail fixation should be made. I have seen much disability in connexion with these fractures when they have been inefficiently treated.

I have had an exceptional experience in having seen three cases of long-standing fracture of the spine of the tibia, each accompanied by marked interference with function. In the one case, a man, aged about 40, was thrown off a bicycle six months previously. He was laid up with severe effusion, and was not able fully to extend his knee. When he consulted me, three months later, walking was difficult, and recurring effusion took place, although attempts had been made under anæsthesia to straighten the limb by means of manipulations, splint and bandage, but with no success. He remained unable to extend his knee by about 15°. An X-ray showed a mass of bone in the centre of the joint. On opening the knee by the incision which I adopt for semilunars, I came down upon the tibial spine, which was displaced backwards, carrying with it the anterior crucial ligament, but surrounded by a mass of fibrous tissue. I removed the bone, leaving enough adherent fibrous tissue to enable me to fix the anterior crucial. The result is a much stronger and more useful knee, which even yet cannot be fully extended. I cannot remember ever seeing a reference to this injury in literature. A similar case shows the fractured spine lying in front of the joint preventing extension by purely mechanical means; full restoration of function followed its removal.

Avulsion of the tubercle of the tibia is by no means a rare accident, and I have found it most common between the ages of 12 and 16. It may occur, as in the case of the femoral head, by a single strain or by a series of strains, and it is very apt to be overlooked. The symptoms consist of pain on acute flexion and on pressure; but there is never a complete loss of voluntary extension. When we remember that the tubercle is sometimes part of the main epiphysis, from which a long protuberance descends, that sometimes there is a separate epiphysis, and that only a part of the ligament is inserted into it, one can understand the rarity of a complete displacement. It is never necessary to pin this fracture, for in the recent case a pad and splint will completely rectify the trouble. Both in the recent and the older case a hinged knee-cage limiting flexion is an admirable contrivance. Later, the quadriceps must be vigorously massaged. In a few instances I have met with a good deal of thickening and tenderness following these injuries. They occur in those cases where no diagnosis has been made, and where the joints have usually been subjected to movements and massage. A linear incision with a chisel into the tubercle in its longitudinal axis acts as a charm, and in a few weeks a disability of some years' standing may be rectified.

A volume could be written upon injuries about the ankle-joint, but time will only allow me to refer (and that briefly) to a few of the more interesting fractures that call for special care.

An injury which I have several times met with is a split fracture of the outer portion of the lower and anterior articular surface of the tibia. A triangular piece in juxtaposition to the fibula, which may involve only the anterior and external surface of the bone, is displaced slightly forwards (fig. 5). This fracture occurs as a separate entity, but is often found in conjunction with a Pott's fracture, and sometimes in connexion with a fracture of the astragalus, and *then* this injury is associated with considerable violence. It is generally produced by a fall upon the foot such as occurs in the act of jumping. Unless the foot is X-rayed the injury is likely to be overlooked, and the consequences are anything but pleasant. In the neglected case the patient is unable to flex his foot even to a right angle. He complains of constant pain over the flexure of the ankle, increased on pressure, and the foot has the appearance of being swollen above the instep. There is a pain which catches him across the front of the ankle in walking just when he is about to change his weight on to the other foot; this is the main ground for complaint. There is a widening of the lower end of the tibia, and a forward projection; indeed, the deformity is suggestive of a displacement backwards of the foot, but a comparison of the heels will at once correct this mistake. The displacement of the fragment is always forwards.

If the case be recent, sharp pain is experienced over the slightly prominent lower end of the tibia, and the foot cannot be properly flexed. To reduce the fracture, the foot should be fully extended, and, while firm pressure is made upon the forward fragment, the foot should be acutely flexed. If this position be maintained for a few days the deformity will not recur. If the foot cannot be acutely flexed, the tendo Achillis should be divided, or, if the case falls into sufficiently reliable hands, a plate or screw may be applied. In old-standing cases removal of the obstructive bone is required. When operation is refused, an addition should be made to the heel of the boot, so that the prominent tibia is not subjected to pressure. Even experienced radiographers fail to recognize this condition in old cases, for all that is seen sometimes is a thickening and blurring from osteitis of the lower margin of the front of the tibia. I have described this interesting fracture at length for the benefit of those remote from X-ray installations.

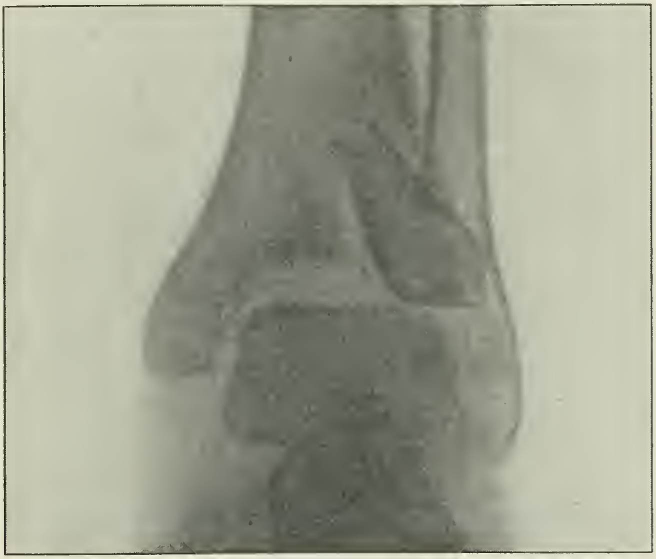


Fig. 5.
Split fracture of the lower end of the tibia.

The same type of fall upon the foot produces a transverse fracture of the astragalus. The posterior half is displaced backwards and usually lies just in front of the Achilles tendon (fig. 6). The anterior portion may be displaced forwards, but more generally remains *in situ*. The surgeon may at once decide on the wisdom of immediate excision of the displaced half. If one portion of the astragalus remains in position it may be safely left there, and good movement, with no shortening, may be predicted. After a large experience of the horizontal fractures of the astragalus, both recent and remote, I am biased in favour of immediate removal of the bone. More especially is this the case where it is driven into the os calcis. I regard the mechanical treatment of fracture of the astragalus and os calcis in combination as very unsatisfactory.

I shall only say a few words about Pott's fracture. Although this injury has been a constant source of irritation to both surgeon and patient, I believe it to be very tractable to reasonable treatment. The material deformities in Pott's fracture are two: first, the well-known outward dislocation of the foot, which must be fully corrected; secondly, a less generally recognized slight backward displacement of the foot. Failure to correct this fully is the most frequent cause of the stiffness and limitation of flexion of the ankle which too often follows the injury. All a Pott's fracture requires is really complete reduction, and, secondly, protection from erroneous deflection of body weight when walking is begun. This concerns the practitioner who first sees the case rather than the consultant. The reduction should be immediate, in spite of the swelling and general effusion. Delay adds seriously to the difficulties and prospects. Some surgeons are afraid to exercise enough force. To reduce this fracture I first of all increase the deformity to disentangle the ends, and with the foot somewhat flexed, pull the heel forwards and push the tibia backwards until I feel that the dislocation of the astragalus is amended. I then direct all the force that is needed to slightly overcorrect the valgus. Unless there is a split fracture of the posterior portion of the tibia, there is but little tendency to subsequent displacement, and for three weeks this overcorrected position is maintained. During that period I shall have carefully examined the position of the foot every few days. At the end of three weeks passive movements will be performed in the manner I have already suggested, but no weight-bearing should be done earlier than the fifth week. If the patient, although the position of the foot is perfect, is now allowed to walk without protection, he will return to the out-patient department with a valgoid deformity; and herein lies the application of the second principle to which I referred—namely, the correct deflection of body weight. For six weeks or less the patient wears a boot with the heel crooked so that the body weight is deflected from the inside to the outside of the tarsus. If these simple methods are applied, Pott's fractures will not prove in the least difficult. Unsatisfactory results are chiefly due to inefficient reduction and indiscriminately applied plaster. If, when the case is not seen for a day or two after the injury, difficulty is experienced, division of the tendo Achillis will materially help the reduction of the displaced astragalus. In lateral deviation of several weeks' standing I have reduced the lateral displacement with a foot-wrench, and, where that is not available, by a simple device which I learnt from H. O. Thomas. It consists in placing a roller towel round the patient's heel and the surgeon's shoulders, and another round the lower end of the tibia and the surgeon's foot. When the surgeon endeavours to straighten himself, his thigh muscles pull the patient's tibia down, and his shoulders pull the ankle up. After reduction and fixation walking is aided by the foot being kept in inversion by an altered heel and outside iron.

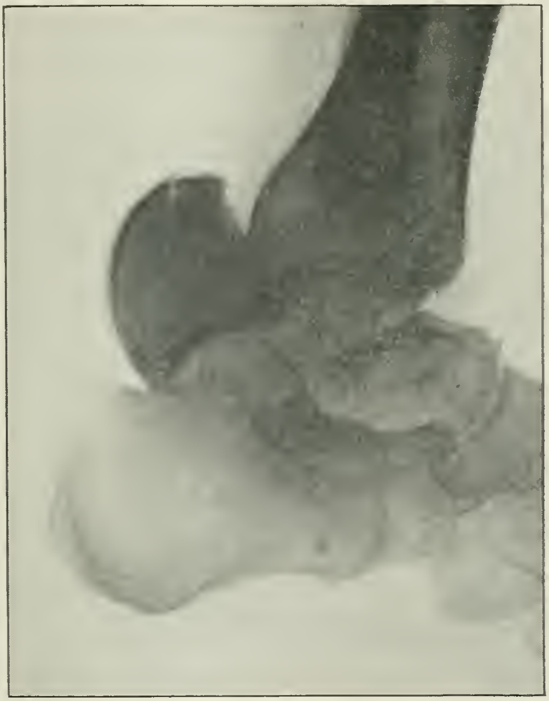


Fig. 6.

Fracture of the astragalus with displacement backwards of the posterior fragment.

In old mal-united cases I operate. If the main deformity is eversion, I remove a wedge from the tibia three-fourths through the bone. The fibula is subjected to open osteotomy. The wounds are closed and the stitches removed in a fortnight, when the limb is fractured for purposes of correct alignment. I find it advantageous in this and other fractures and deformities to operate in two stages, so that in my subsequent manipulation I have only a simple fracture to deal with. In old cases of bad eversion and unreduced displacement of the ankle I remove the astragalus and correct the lateral deviation by osteotomy and wedge.

Conclusion.

I have given a brief outline of the methods and principles of treatment I employ. They are the result of long practice in a manufacturing and mining district, where I have had exceptional opportunities of seeing a very large number of injuries both immediately after the accident and at a later stage.

Briefly, I pay more attention to function than to appearance, consider correct alignment of more importance than meticulous care in reposition of fragments, believe that pain is always an indication for physiological rest, and therefore deprecate early forced passive movements.

If I prefer methods of manipulation and fixation in some cases where others of my colleagues would prefer an open operation, it is perhaps because for many years an increasing proportion of my work has been the treatment of deformities and disabilities, whether of traumatic origin or not, and I have thereby gained confidence in using powerful but carefully graduated force in cases where another would hesitate to do so.

My object has been not so much to discuss various methods of treatment as to expose my own for discussion, and hope that those who follow may do the same for our mutual profit.

Mr. Rushton Parker expressed a general agreement with the views stated by Mr. Robert Jones in regard to matters that had interested him greatly for over thirty-five years, dating from his first acquaintance with the late Mr. H. O. Thomas, from whom he learnt most important improvements in clinical practice, especially in the management of injuries to the bones and joints; and whom he persuaded to publish an account of his splints, since celebrated throughout the world.

He would go a step further than Mr. Jones, and would make not even a late concession in the matter of passive movement, which he ventured to think we could do very well without, and maintained that, when not injurious, passive movement is useless. Massage, however, he would welcome, on account of the aid given thereby to the functional nutrition of injured parts in certain stages of recovery, as in no circumstances could it do harm. The time at the disposal of the meeting would not permit him any attempt at a general review, and, in order not to encroach on the privileges of other Fellows, he would confine his remarks to the upper limb. These remarks are an amplification, though to some extent a repetition, of some made by him, somewhat hurriedly, at the end of a meeting of the Section on June 16, 1908, when he related a case of severe compound fracture of the arm,[1] from the report of which he now made use of illustrations by means of the epidiascope.

The first point is the great practical advantage derived from slinging the wrist to the neck in the acutely flexed position of the elbow (loc. cit., figs. 5 and 6). This is of great assistance in the first examination of the upper limb when injured anywhere between the collar-bone and the elbow, both inclusive. It will be seen that the upper limb is thus divided into halves, one of which, the hand and forearm, is hung to the neck, and its weight removed from the arm. The other, the upper arm alone, hangs from the shoulder, and falls by its own weight, straight and undeformed, along the side of the chest; and does not suffer the painful distortion otherwise caused in fractured humerus, by the irregular dragging and ineffectual efforts of the arm muscles, which in this slung position are not called upon, and therefore remain at rest. The position thus facilitates diagnosis, and is a convenience to both patient and surgeon. In the figures referred to, the fractured bone was as loose as a flail at the time of the photograph, and yet hung straight, as proved by the X-ray illustration in fig. 8. On putting the patient to lie down, the displacement shown in fig. 7 immediately occurred.

---

[1] *Proceedings*, 1908, i, (Surg. Sect.), p. 221.

This position is furthermore the best in which to treat fractures of the humerus, whether near the shoulder or elbow, or elsewhere. The swelling of the hand and forearm that so easily occurs in the rectangular position of the elbow does not now happen, and the clumsy and tiresome separate bandaging of the fingers is done away with. In fracture of any part of the shaft, including that of the surgical neck, an assortment of hollowed, padded splints of various sizes surrounds the bone, tied together by strips of bandage, as partly shown in fig. 3; a final shoulder spica, in fracture of the neck, giving further support. In some cases hollowed splints of wood or of Gooch material do quite well, and have the advantage of permitting X-ray representation of the state of the bone, while they are still in place. But, as shall be shown presently, splints of sheet metal are sometimes indispensable, and are generally more convenient. In simple fractures it is convenient to have the padded splints lined with antiseptic gauze to prevent itching and other discomfort. In compound fracture the sheet-metal splints may be laid bare and unpadded on the enveloping dressings.

In fracture of the anatomical neck no splint is required. The limb is slung as above described and kept to the side, while, if thought desirable, strips of sticking-plaster may be laid round the shoulder before and behind, some vertically and others horizontally. These are just as effectual as the conventional shoulder-cap of gutta-percha, pasteboard, or plastic felt; are applied in a few moments, and, by fixing the skin, afford any additional protection that may be required.

Oblique and vertical fractures through the humeral condyles, head of radius, and some fractures of the neighbouring end of the ulna, are best treated by immediate slinging in the same attitude, without splint, and kept that way till well. On gradual resumption of use any want of suppleness gets less and less under slight voluntary movements, but without any attempt at passive movement being necessary, provided the patient has regained power to voluntarily bend the elbow to less than a right angle. Till that power returns the original attitude must be continued.

In fractured humerus, right through the shaft anywhere in the lower third, surgeons are aware of the tendency of the lower end of the lower fragment to be adducted or rolled inwards by the weight of the elbow in the position indicated. This was the case in the subject of these illustrations, and could be counteracted by the careful use of the conventional rectangular splint. But we are here dealing with the vastly superior attitude of acutely-flexed elbow, in which the lower fragment is kept in line much more neatly, easily, and effectually by putting a twist into one of the splints, as shown in fig. 4. It is needless to remark that for such use only a splint of sheet metal can serve. The twist is made or unmade in a moment by holding one end of the splint in each hand, pronating one forearm and supinating the other in the same act, and thus imparting any degree of twist required. If any of the surrounding splints overlap, their utility and convenience are not impaired.

This is one of the numerous devices of H. O. Thomas, and was further applied by him in a remarkable way to the treatment of Colles's fracture. A word first, however, on the "reduction" of that fracture, which was advised by Colles himself without explaining how. Many a Colles's fracture is loose and ready for splints from the first. Others are distorted in the well-known manner, very painful, and in a condition that has been called "impacted." The late Professor Gordon, of Belfast,[1] showed how and why real impaction never does or can occur. Museum specimens thought to prove the fact of impaction are merely instances of union and callus around unreduced cases. But the condition that requires "reduction" is the rotation backwards of the lower fragment, which thus becomes locked by the tendons running in its grooves and now twisted at a painful angle. To reduce the deformity and remove the constant pain caused by this locking, Mr. Parker demonstrated the method adopted by Mr. Thomas, which is both simple and effectual. Mr. Dunn kindly permitted the use of his own limb for the purpose, which consists in over extending the wrist-joint by holding in one hand the hand of the patient, and in the other the forearm above the seat of fracture. In this position the extensor tendons lodged in the radial grooves are kept quite slack, while the lower fragment, attached to the wrist, is thrust into position by forcibly pushing down and forwards the hand and carpus. Though the movement is itself momentarily painful, relief follows immediately, the part being then ready for the application of splints.

These consist, in the method described, of two hollow splints of sheet metal, suitably padded, each twisted in the direction of pronation, according to the side affected. Besides the twist a lip is forcibly imparted to one end of each, in exactly the same manner and direction, and corresponding to the radial edge of one. The lip catches the carpus and radial fragment of one and the lower end of the ulnar in the other. The effect on holding them together in the forearm is to prevent abduction of the carpus with its attached lower fragment of radius.

---

[1] "Treatise on the Fractures of the Lower End of the Radius, &c.," 1875.

Slack bandaging is enough to keep the splints in place, but, to prevent them from sliding off, it is convenient to attach to each a strip of sticking-plaster doubled with its sticky sides out. One sticks to the splint and the other to the skin of the patient. Moreover, it is not necessary to use a sling, as if the wrist swells it will favour union. Mr. H. O. Thomas was very particular about this slack bandaging in this and, in fact, all fractures. Otherwise undue pressure and consequent anæmia may lead to delayed union, especially in elderly persons, as once actually happened to a patient of Mr. Parker's, for, on removing the splints on the supposition that union had occurred after some five weeks, and while the fragments were in perfect apposition, the abduction deformity afterwards occurred. He attributes the fault thus occurring to an insufficiently slack application of the bandage.[1]

Mr. WHITELOCKE (Oxford) said he was afraid that at such a late stage in the meeting he could not attempt anything like a criticism of the paper, but he thanked Mr. Robert Jones for having given such an excellent opening. The ground had been so well covered that very little loophole had been left for anything like definite criticism. But there were one or two points which struck him forcibly. The author did not mention fractures of the patella. He (the speaker) had hoped to hear something of that, and of Mr. Jones's methods of treating that disability. Mr. Jones had just said that he operated for that condition, and that statement disarmed the criticism he had been about to offer. He also thought that in all cases of fracture of patella in which there had been much separation the open method was the only satisfactory one, at all events if one had to consider the question of the Workmen's Compensation Act and the Employers' Liability Act, because he believed the lay public would not be satisfied with anybody who allowed them to walk about, even comparatively comfortably, with a separation of 2 in. or 3 in. He had been particularly struck with the beautiful illustrations of Mr. Jones, especially those illustrating the traumatic coxa vara, which was so liable to occur in adolescence. It was an injury which he had noticed, and which he had formerly missed because of the slight inconvenience caused at first. Mr. Jones pointed out clearly where mistakes were liable to be made, and for that he thanked him. In following

---

[1] *Note after the Meeting.*—Mr. Parker agrees completely with Mr. Jones's reference to the treatment of fractured femur in Thomas's hip- and knee-splints. He brought the matter before the International Medical Congress in London, 1881, reported, with illustrations, in the *Transactions*, ii, p. 452.

Mr. Jones's descriptions of his various methods of treatment, he could not help feeling how very liberal were his statements; and he believed Mr. Jones struck the principal note when he said that almost any method would succeed if the principles of correct alignment, as well as well as complete functioning of the joint, were consistently aimed at. He thought all surgeons would feel that, with proper care, most of the fractures could be treated satisfactorily. He was pleased to hear the opener of the discussion admit that there were certain fractures in which the open operation was the quickest and most satisfactory.

He would wish to support very strongly the statements both of Mr. Robert Jones and Mr. Rushton Parker as to the practice of early passive movements in cases of fracture. He believed that in most cases early passive movements were not only unnecessary, but positively harmful.

Mr. RICHARD WARREN: From the point of view of treatment, fractures in the vicinity of joints may be roughly divided into two classes: (1) Those which actually involve the bearing surfaces of the joint—i.e., in which the continuity of the cartilage-covered surface of either bone at any point where it comes in contact with the other bone is interrupted by the fracture; e.g., fracture of the patella. (2) Those in which the line of fracture is close to the bearing surfaces of the joint but not through the latter—e.g., Colles's fracture of the wrist.

It will therefore happen that, while all the fractures in the first group are inside the capsule of the joint, in the second group some fractures will be found partly inside the joint, most will be outside the capsule. It is rather a nice point as to how near the fracture should be to a joint to be admitted to the group under consideration to-day.

All fractures treated by fixation implicate joints, especially in patients who are past the first bloom of youth. For instance, some of the worst cases of stiffness of the knee and hip I have seen have followed a fracture of the middle of the shaft of the femur in which immobilization of the limb has been prolonged. Too much attention is concentrated on the position of the fractured bone and not enough on the equally important points—ligaments, muscles, nerves, and vessels—which go to make up a limb, and which deserve every bit as much consideration as the bone. In the treatment of fractures one of the most important points to be settled in a given case is the necessity for fixing the fragments by open operation, and in fractures about joints this question will pretty frequently arise. For this reason I have made the above-mentioned division of fractures in the neighbourhood of joints, because one must consider the degree of accuracy in the reposition of fragments, which is essential to the proper working of the limb. That counsel of perfection, absolute accuracy in reposition of fragments, is seldom possible without open operation, and may be exceedingly difficult if operation be practised. Fortunately this is not essential in cases where the fracture does not involve the bearing surface of a joint. Minor degrees of malposition may co-exist with perfect junction of the limb, provided that good alignment is obtained. It is otherwise when the fracture passes through the bearing surface of a joint; here, unless the fragments are brought into perfect apposition, the cartilage may be ground off by protruding rough edges of bone and a condition of traumatic osteo-arthritis will supervene with great impairment of the joint. This is particularly the case in adults, for in children fractures involving joint surfaces do well with much less accuracy of replacement of the fragments, partly because, in these young subjects, the epiphysis is largely formed of cartilage, and there is consequently less chance of a sharp, bony point obtruding itself between the smooth working surface of the joint, and partly because, in children, the power of repair in joints is vastly superior to that found in adults. Put in more definite terms, I should say that fractures involving the bearing surfaces of joints in adults should be replaced to within $\frac{1}{32}$ in. or thereabouts; whereas fractures not actually involving this important surface may be $\frac{1}{4}$ in. to $\frac{1}{2}$ in. out of touch without causing any trouble.

In general the fracture involving joint surfaces will, in adults, frequently—perhaps one may say usually—in children less frequently, require open operation even when the displacement is small. Fractures close to but not through joints seldom require open operation unless gross displacement, which cannot be corrected by external manœuvres, is present. A great factor in the rapid and successful restoration of a fractured limb to position is early movement and massage. This is particularly the case in fractures near joints, for even if the fracture does not involve the bearing surface of the joint, yet the organization of the hæmorrhage and exudate about the ligaments and tendon will produce great, possibly permanent, stiffness unless this be combated by appropriate treatment.

It might be thought that the importance of early massage and movement is a strong argument in favour of open operations, because by this method one can produce absolute parity of fragments and yet move the joints easily without risk of displacement. This is true to some extent. Some fractures will always slip out of position when the joint is moved; but they are not very common, and, when found, obviously demand open operation and wiring, screwing, plating, or what not. The greater number of fractures about joints, however, can be reduced by manipulation and kept in position by appropriate arrangement of the limb, nor does early movement in, say, a week or so, as a rule, cause any displacement; movement is, of course, made very gently at first. A fracture which has been reduced without open operation can be managed earlier and moved nearly as early as one which has been wired.

Turning now to special regions:—

The shoulder-joint: Fractures commonly occurring here are of the surgical, less frequently of the anatomical, neck, and in a variety of fractures between these points reduction can practically always be maintained by a pad in the axilla and allowing the elbow to hang, the hand being slung at the wrist. There is a tendency to keep these fractures fixed too long; as a rule, movement can be made in a week to a fortnight, and massage instituted on the third day. The chief source of bad results here is an associated injury of the brachial plexus.

The elbow: By far the commonest injury here is separation of one or more of the epiphyses of the lower end of the humerus. Acute flexion seems the best way of correcting displacement and the tendency for the lower fragment to become hyper-extended, leading to diminished range of flexion of the joint. Note that acute flexion cannot always be obtained at first owing to the swelling and danger of pressure on the vessels, but this position can always be got in a very few days by increasing the flexion; cases also do well in a posterior gutter splint with the elbow at a right angle. In young subjects malposition can be corrected by active movements in an appropriate direction, the new bone being bent gradually—e.g., when the lower fragment is in a position of hyper-extension and the elbow cannot be fully flexed, one makes the patient practise flexion of the fore-arm against a resistance with excellent results, very much in the same way that after excision of the knee the hamstring muscles produce an angular deformity although firm bony union has taken place.

The wrist: Colles's fracture is by far the commonest trouble. Good results are got by early massage and very light splintering. Many cases do not need splints at all. Slight malposition is of no consequence as regards practice; gross deformity is usually associated with disability. The most deleterious factors in the prognosis of Colles's fracture are:

(1) old age; (2) severity of the fracture and consequent coronation of ligaments, &c.; (3) length of time the fracture is kept fixed before movement and massage are begun. Of ninety cases of this fracture traced, two-thirds were good working results; one case in twelve was absolutely bad. Early massage is the all-important point of treatment here.

An interesting group of cases are those of epiphysial injury to a part of a limb containing two bones—i.e., the forearm or legs—since at times there are impairment of growth of the injured epiphysis and deviation of the axis of the limb. I have notes of two such cases, in both of which the lower labial epiphysis had been injured. The result was a retardation of growth, not a complete cessation, of the inner part of the lower end of the tibia; the outer part of the lower end of the tibia and the lower end of the fibula went on growing normally, producing a condition of talipes varus.

Case I, aged 8; injury to the ankle six months before, varus position, the foot marked but not grave; kept in walking instrument, double irons to knee and varus T-strap. At the end of eighteen months the condition was improved.

Case II, aged 12; injury two years before to ankle, bad varus. Position of foot: Sole vertical, too bad to go into walking instrument. Operation: Osteotomy of lower end of tibia in an oblique direction, and foot bent out so that the sole was horizontal, and put in plaster; three months later walking well on sole of foot, with bayonet-shaped tibia; given a walking instrument.

Seeing the years in front of these cases before the epiphyses unite, it will be interesting to note what is their ultimate fate, and whether or not they can be kept in position by instruments. Judging from the first case, I am inclined to hope that they can.

Mr. Robert Jones, in answer to Mr. Warren, said that he had met with the difficulty to which that gentleman alluded, and treated it, as he said, by an oblique osteotomy of the tibia. Fracture of the patella he had wired in young and athletic folk, whereas to older folk he mentioned the risks, and if they wished for operation, he did it. But in the cases of people past the prime of life he preferred mechanical methods, for they invariably resulted in a good walking limb, and the patient could walk in a Thomas's caliper splint about five days after the fracture, so that he could attend to his business practically right away.

(The discussion was adjourned until November 29.)

Surgical Section.

November 29, 1910.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

Discussion on The Treatment of Fractures in the Neighbourhood of Joints.[1]

Mr. HERBERT J. PATERSON said he thought Mr. Robert Jones had treated the subject so exhaustively that but little remained to be said; but the debate illustrated the value of meeting together to discuss disputed points. As the author said, there were two schools: those who relied mainly on operative measures, and those who depended on mechanical means. The discussion indicated that there was less difference in principle between the two schools than was generally believed. Mr. Jones's practice was that if he could not place the bones in perfect alignment he operated. That was the same position which had been taken up for years by Mr. Arbuthnot Lane. This debate showed that Mr. Lane and Mr. Jones were agreed as to first principles. Mr. Jones referred to one very important point—namely, what might be termed the abuse of X-rays. He (Mr. Paterson) thought that it was only too true that nowadays house surgeons, and perhaps even others, relied too much on the X-rays as a means of diagnosis, and so failed to educate fully their powers of clinical examination. Thus they could not diagnose fractures so well as they did before the days of X-rays. As Mr. Jones said, the X-rays should be used only as a court of appeal, after a diagnosis had been attempted by other means. The author said he attached more importance to good function than to mere changes in form. But, whether the surgeon liked it or not, he had to recognize that since the introduction of X-rays the public had become increasingly critical as to the results attained in the treatment of fractures; and that nowadays courts of law were not always satisfied that everything had been done, if an X-ray picture showed anatomical imperfection, however good the functional results might be. He did not argue that the attitude was right, but, as it existed, it must either be accepted or an attempt made to alter it. He was much interested to learn from Mr. Jones that the treatment of fractures above the femoral condyles by a double-inclined plane was based upon a superstition. He was afraid it was a superstition which still survived, and he (Mr. Paterson) was brought up on it. He did not think Mr. Jones's analogy of a fractured femur above the condyles with an osteotomy was a true one. After a traumatic fracture there was much more laceration and separation of muscles than in section of the femur with the chisel; and he did not think one would expect to have to treat an osteotomy with a double-inclined plane any more than one would find it necessary to use either a screw or a plate to keep the bones in position after osteotomy. Recently he had had the advantage of seeing Mr. Jones at work, and had observed the wonderful resource and skill he showed in dealing with all kinds of fractures. He realized that often Mr. Jones was able to succeed with manipulation when others less skilled would be almost certain to fail. His own experience of fractures immediately above the knee-joint, treated by extension and splints, had been very unsatisfactory; and, although he had designed a special splint for treating them, he had now come to the conclusion that in most cases an open operation was the best method of treatment. It was just in such fractures as these that one often found torn pieces of muscle interposed between the fragments which rendered apposition difficult or impossible without an open operation. He asked Mr. Jones whether in a fracture above the condyles he would consider inability to elicit crepitus on extension as an indication for open operation. He cordially agreed with all Mr. Jones said as to early passive movement, and believed that many of the bad results following those fractures about joints were due to the advocacy of the present fashion of early passive movement. It would be good if that Section could agree that early passive movement was not so desirable as many would have them believe. He supposed this advocacy was due to the widely prevalent belief that if a joint were kept at rest for a long time it would become permanently stiff; and he was glad to hear that Mr. Jones classed this amongst the surgical superstitions. Notwithstanding the advances in surgery, some superstitions still existed which had been handed down from generation to generation. It would be well if this debate were instrumental in exploding them. Possibly many surgeons kept their fractures at rest for too short a time, and he thought the periods given in most text-books were too short. Some of the most troublesome fractures were those about the ankle-joint. Mr. Clinton Dent had said that he had hardly known a member of the police force who was able to return to duty after having suffered from a Pott's fracture. In these fractures accuracy of alignment and careful reposition of the parts were so important that it was wiser to operate; and he gathered that Mr. Jones's practice tended that way. Possibly some of the bad results were due to the circumstance that many surgeons did not keep their fractures under observation long enough, and bad valgus was likely to result. The patient, thinking he had been badly treated, sought advice elsewhere; and consequently surgeons saw the bad results from the practice of others, but not their own. He fully agreed with Mr. Jones that an open operation was rarely, if ever, required in the case of Colles's fracture. The results obtained by reduction and treatment by splints were almost invariably excellent. It always struck him as curious that even the most vigorous opponents of the open method of treating fractures were often enthusiastic advocates of operation in cases of fracture of the patella. That attitude did not seem to be readily explicable. Firm fibrous union of a fractured patella, when treated without operation, was the rule. There was no question of correct alignment, and it was not of great importance whether or not the fragments were brought into close apposition, and the fracture did not result in any shortening or deformity. No one could assert that there was absolutely no risk of sepsis in opening a knee-joint. And yet many of those who regularly operated in fracture of the patella, maintained that it was unjustifiable to operate on a fracture of the femoral shaft. Personally he (Mr. Paterson) considered that operation was more certainly indicated in the case of a fractured femur than in the case of a fractured patella. In the case of the femur correct alignment was of the utmost importance, and the risk of operation was *nil*. For his own part he did not advise routine operation for fractured patellæ, because he believed that as good a result could be obtained without as with operation. He quite realized that in making this remark he was laying himself open to the risk of being deemed old-fashioned, but he was trained under a surgeon who obtained excellent results without operation, and he had endeavoured to follow in his teacher's footsteps. Indeed, he went further, and maintained that in this instance fibrous was better and stronger than bony union. He had seen four cases of a second fracture of the same patellæ, and in each case it had not been the fibrous union but the bone itself which had given away.

---

[1] Adjourned from November 8, 1910.

As he said at the beginning, he had recently had the privilege of seeing Mr. Jones at work, and so realized that he had good reason for the faith that was in him. He could assure them that a visit to Liverpool was a very humbling experience, and made one feel very diffident about disagreeing with the views and opinions of the distinguished author of the paper, to whom they had listened with such pleasure and profit.

Mr. C. H. Fagge said he thought it would be agreed that the great obstacle in restoring the normal alignment of such fractures as were now under discussion was the small size of the articular fragment and the consequent inability to exert any leverage upon it, so as to bring its broken end into apposition with the broken end of the larger fragment. For this reason, because deformity near a joint had worse consequences than in fractures near the middle of a long bone, he thought this class of fractures was peculiarly suitable to treatment by open operation as introduced by Mr. Arbuthnot Lane. The necessity for accurate replacement of the fragments varied in different parts of the body and in different persons according to their occupation and position in life, but he thought there could be no difference of opinion as to the suitability of fractures which were commonly called "Pott's fractures"—whether of the adduction or abduction type—for open operation. The common experience of all surgeons now, as twenty years ago, was that if the parts were not restored to an alignment which was at least but little short of normal, flat-foot, pain in the ankle-joint, and even genu valgum usually occurred afterwards. Similar injuries in boys generally resulted in partial separation of the lower tibial epiphysis. He used the term "partial" advisedly, for injury to this epiphysial line seemed, as in other epiphyses all over the body, to produce a bone lesion which was a complete separation at the epiphysial line, but was partially a separated epiphysis and partially a fracture. This was strongly brought to his mind recently by the examination of a large number of X-ray photographs which had been taken at Guy's Hospital during the last ten years, from which it was apparent that one of the common injuries to the lower end of the humerus in a child was very rarely a completely separated epiphysis, but a partial separation of the epiphysis, with which was a fracture passing upwards into the shaft. It was equally true in the injuries he had seen in adolescence to the lower end of the tibia. Recently he operated in Mr. Lane's absence upon a boy who had such marked deformity from partial separation of the lower epiphysis of the tibia that it was diagnosed in another hospital as a dislocated ankle, and efforts at reduction had already been made. There the lower end of the diaphysis was 1 in. in front of the epiphysis, and there was on the outer and posterior aspect a strong, spike-like portion of bone attached to the epiphysis, which had already formed an early union with the shaft in a bad position.

He would also hold that many cases of supra-condylar fracture of the humerus in a child could not be accurately replaced by manipulation. In another place one speaker said he had never known this method fail; but he (Mr. Fagge) had failed several times, and showed an X-ray photograph of an injury to the lower end of the humerus in a child in which, even under an anæsthetic, efforts to replace the two fragments in alignment failed, in whatever position the arm was put. The lower fragment was seen to be lying behind the upper fragment, with the arm in full flexion and the hand carefully supinated. In dealing with the growing ends of bone, such as the lower end of the humerus in a child, he had avoided the necessity of putting in a plate, but had brought the lower extremity into apposition with the upper, and it had remained there. He agreed with Mr. Drew that that was not always possible, for in the case to which he (Mr. Fagge) referred, in which the lower end of the tibia was broken from the shaft, it was necessary to put in two strong plates to keep the parts in good apposition.

Of all injuries in the upper extremity calling for operation, Colles's fracture in the wrist, or separation of the lower radial epiphysis in the child, was the one, in his opinion, which most demanded open operation, for he had never seen a result which was in any way anatomical with the ordinary splint treatment of Colles's fracture. He admitted that in many cases fair function was acquired in spite of the deformity; but if the primary requirement in the treatment of fractures here as elsewhere was that the parts should be brought into alignment, that could only be attained in many cases by an open operation. He had also dealt with, by operation, five or six cases of fractures of the scaphoid, in which bony union was often absent. Here, he thought, the line of treatment by operation was that one or both fragments of the scaphoid should be excised; he had found both methods to give a good functional result. Fractures near the shoulder-joint did not usually demand operation, because the movements of the scapula vicariously took on the movements lost at the shoulder-joint. He agreed with what Mr. Paterson said concerning the overdoing of X-rays in these matters. He was willing to agree that the X-ray picture which he had himself handed round *proved* nothing and *showed* comparatively little, because it did not indicate how much movement the child had. He showed another picture of a T-shaped fracture of the lower end of the humerus in a man, which again was fallacious, as all X-rays must be; this had been treated by open operation and a Y-shaped Lane's plate applied. The X-ray suggested a perfect result and showed no anatomical cause for limitation of movement to some forty-five degrees. It would be granted that after such an injury even so limited a range was not to be expected after splint treatment, and the patient evidently found little inconvenience from it, as he could not be persuaded to take further steps to increase it, saying he could carry out all movements he wished. But, on the other hand, X-rays were of the greatest value to the surgeon in telling him whether the parts were in strict alignment, and to obtain this must be one of the first aims of treatment; and again were of help in warning him of the cases in which he should not operate, as there was a class of cases in which degenerative changes had already taken place in the bone, so that operative treatment was, under ordinary conditions, not advisable. In these senile, fragile bones the X-ray showed an unusual translucency of the bony parts and pencilling of the cancellous tissue. Having once tried to put a plate on to the shaft of the femur in such a case, he was now wary about neglecting the contra-indications afforded him by X-rays.

Mr. W. H. Clayton-Greene said the paper gave him a great deal of valuable information, by which he hoped he would profit. He cordially agreed with what Mr. Robert Jones said concerning passive movement. He was brought up to practise movement in connexion with fractures, and he was convinced that it was often practised to the detriment of many patients. He had frequently seen bad results follow passive movement, notwithstanding the care which had been used, especially in one case where traumatic arthritis supervened, and in which passive movements were persisted in, but failed to restore movement. In another recent case he determined to stop all movements, and did so for a month, and, to his great surprise and gratification, he found the joint had become movable. It was curious that surgeons should so depart from the ordinary principles of treatment in connexion with fractures as to make such free use of passive movements. The first object in treatment of a wound or lesion was to give the part rest, yet they were told that in such a serious injury as fracture movement should be commenced as soon as the third day. In the case of separation of the lower epiphysis of the humerus, if he could get the fragments into good apposition, he did not attempt movement before the fourteenth day, and he was satisfied with the result. He had had only a limited experience of fracture and dislocation of the upper end of the humerus—three recent cases, one old. The old case was that of an engineer who had fallen upon his shoulder and received an injury, which was treated as a dislocation. It was apparently reduced, and he saw him seventeen weeks afterwards with a completely stiff joint. The head of the bone was not in its normal place, and a skiagram showed a dislocated head. He could not agree with Mr. Jones that the functional result was good. He (Mr. Clayton-Greene) excised the head of the bone, and the man improved, but he was not very pleased with the result. In the other three cases he operated immediately, and excised the dislocated head, and made no attempt to wire the bones. In the cases he had had there was much comminution, and an attempt to fix the mosaic of bone would, he thought, result in a stiff joint. The result of complete excision of the dislocated head was good. He asked whether Mr. Jones's experience was similar in such cases. With regard to fractures about the elbow-joint, he agreed with previous speakers who had advocated operation in certain cases. He had had a proportion of fractures above the condyles in which he had been unable to manipulate the fragments into position. They were cases with so much swelling that one could not consider bringing the arm to an acute angle ; it was often impossible to get it to a right angle ; the skin was tense, and often had blebs upon it, and there was much blood between the muscles and beneath the periosteum. If operation were delayed in such cases, there was so much fixation of the parts that the deformity could not be rectified. He had rarely to put in foreign material, because in most cases it was easy to get the fragments to interlock by leverage, and put the arm up into an acute position after having let out the hæmorrhage which had been preventing flexion. He had an X-ray picture taken to show whether there was extreme deformity, and if he could not get the arm into that position he operated. With regard to the hip, Mr. Jones was opposed to operative treatment in intracapsular fracture, but he would ask him if he would be opposed to intracapsular fracture being so treated in a young adult. Recently he saw a man, aged 36, who sustained an injury to his hip three weeks previously. He thought it probably was extracapsular fracture, but there was not sufficient thickening about the great trochanter. He was X-rayed, and that showed a typical intracapsular fracture. There was adduction and commencing coxa vara, so he was put up with the limb abducted, and extension was applied. Three weeks later he tunnelled through the base of the trochanter and fixed the head of the femur to the neck with a screw. The result was perfect. Another speaker had referred to the separation of the lower epiphysis of the femur. It was a rare accident, and he had only had one case of the kind. It was usually caused by rotatory violence. There was much damage, and in his own case the biceps was ruptured and all attempts to bring about reduction failed. So did a further attempt by an open operation until the tension of the quadriceps was relieved by fairly free section of the muscle. That muscle seemed to form a firm strap, fixing the separated epiphysis to the front of the femur. He saw the report of a case in which a good result was obtained by sawing through the tubercle of the tibia, and so freeing the tension of the quadriceps by mobilizing the ligamentum patellæ. It was impossible in such cases to effect reduction until the tension of the quadriceps had been relieved. He had the same difficulty which others had: he could not keep the fragments in position until he had screwed them, because the condyle had a tendency to slip forward. So in certain cases of separation of the lower epiphysis of the femur and of the lower end of the humerus he strongly advocated operative treatment.

Mr. H. P. Symonds said he had seen many fractures of the humerus in young people imitate dislocation, and he did the open operation to remove the upper end of the lower fragment, using no splint but leaving it in a sling. He had done the same with the lower end of the humerus. In this case the lower end of the upper fragment acted like a wedge, and the removal of that piece of bone did no harm. No passive movements were needed, but gentle massage and a bandage gently applied. That had been his practice in all such cases lately.

Mr. A. J. Walton said that these varieties of fractures could be divided into three groups: firstly, those involving the articular surfaces; secondly, those near to but not involving the joint surfaces; thirdly, those where the fracture was associated with a dislocation.

In the first group the conditions giving rise to difficulty in treatment were: The small size of the fragments, which were therefore difficult to control; the fact that subsequent irregularities, even if small, led to much disability; the tendency for portions of synovial membrane, ligament, or periosteum to be interposed between the fragments; the presence of much blood within the joint which was likely to be followed by adhesions. Therefore there would be a greater tendency for open operation, for by this means alone could all these difficulties be overcome. If, however, all these conditions were not present, then one could obtain good results without operative treatment.

The first question which would arise in each case was whether operative or non-operative measures should be undertaken. He would express his full appreciation of those holding what he might describe as extremist views in favour of passive movements and massage, or of open operation, for by their efforts these methods have been given full prominence; but to his mind no one method could be regarded as a panacea for all varieties of fracture, but methods of diagnosis had now become so accurate that there should be no difficulty in selecting the treatment for the individual case. In every case the fracture should be examined by general clinical methods, and then radiographed in two planes. Then under an anæsthetic an attempt should be made to reduce the deformity; afterwards a second radiograph should be taken, and, if the deformity persisted, open operation would be advisable, not more than seven to ten days being allowed to elapse before the operation was undertaken, lest secondary changes in the surrounding tissues be sufficient to cause difficulty in reduction even with open operation. If, however, it were generally taught that all these cases should be treated by open operation, even those unskilled in this special line of work might have to undertake it or be liable to action for maltreatment. In such cases the dangers of open operation might become very great, and the results of treatment much worse than could be obtained by non-operative measures.

With regard to non-operative measures, he would certainly in these cases give preference to early massage and passive movements, but only as adjuncts to retentive apparatus; there was always blood effused into the joint, and the only way to prevent adhesions was by massage and passive movements. In some cases such treatment might cause displacement of the fragments, and thereby increase the amount of callus. Such displacement would, however, be shown by a radiograph; if present, the case must be regarded as one suitable for open operation; it was one in which the fragments cannot be adequately controlled. Passive movements should, as Lucas-Championnière teaches, always be first applied to the peripheral joints. The radiograph he showed was one of a case of Mr. Kidd's, of a fracture of the surgical neck of the humerus. The instructions as to passive movements were misunderstood, so that they were given to the shoulder, but not to the hand or forearm. Consequently eighteen months afterwards, although there was perfect movement in the shoulder, and the anatomical result was extremely good, yet there was some stiffness of the fingers. Owing to the more general use of massage and passive movements, Volkmann's ischæmic contraction was becoming quite rare, and most of the cases now seen were of old standing, having been treated before the general introduction of this method. At first sight there seem to be exceptions to this general method of treatment, but it is simply that long experience has conclusively shown that they never react to manipulative treatment, and hence an operation could be proceeded with at once.

In a case of fractured patella, with wide separation of the fragments, all the difficulties were present, and bony union with complete restoration of function was almost impossible without operation. Rarely even with open operation relapses might occur from the wire cutting its way out. This could be avoided by the routine use of a mattress suture, which passed transversely across the fibres of the aponeurosis. In cases of softening of the bone it was the only suture which would satisfactorily hold. In one case where a fracture occurred in a patient the subject of tabes the fragments were extremely friable and almost as soft as a piece of cheese; but apposition was firmly secured by such a suture, the aponeurosis bearing the greater part of the strain. Rarely bony union would occur without operation. He showed radiographs of such a case belonging to Mr. Dean. This was a very extraordinary condition. Fourteen years ago the right patella was fractured and treated by palliative means. Bony union resulted, the gap between the fragments being bridged by new bone. Nine years ago the left patella was fractured, treated by palliative means, and bony union again resulted. The patient was admitted this time for another fracture of the lower end of his right patella, as shown in the radiograph, a condition said not to be uncommon in these rare cases owing to the increased length of the bone. If the patient's general condition absolutely forbade operation, he believed in the use of the much-abused Malgaigne's hooks. By their use the fragments could be more or less fixed in apposition, so that passive movements could be commenced from the first, and thus when fibrous union of the fragments had taken place the joint would be free from adhesions.

The same points would apply to a case of fracture of the olecranon. The other indication for operation was complete separation of a small fragment within the joint—for instance, some fractures of the head of the radius, complete separation of the head of the humerus, or separation of the tibial spine. If, however, the small fragment was joined at any part by the capsule or ligaments, it might be possible to bring the parts into apposition and obtain almost perfect results without operation. As an instance of this a fracture of the base of the first metacarpal bone (Bennett's fracture) might be given.

In all other cases palliative treatment should first be tried in the routine way. If this failed, an operation should be done. As an example, a radiograph of a case of Mr. Sherren's of fracture of the surgical neck of the humerus was shown. Manipulation under an anæsthetic was tried and failed. This was followed by extension with the arm fully abducted; this also failed, as the first plate showed. Operation was then undertaken and the ends plated in perfect position, as shown in the second plate. After six weeks there was perfect movement in the shoulder-joint. Thus two cases of fracture of the surgical neck of the humerus had been shown, both of which were followed by perfect results as regards the shoulder, but one had been treated by palliative means, whilst the other had required open operation.

In the case of fractures of the lower end of the humerus a good result could generally be obtained by putting up the elbow in a position of full flexion. In many cases the swelling was so great that the arm could not be fully flexed immediately; it should then be flexed as gently as possible and massage commenced at once; as the œdema decreased the flexion should be increased until the arm was in the fully-flexed position. During this gradual flexion the arm would have to be carefully watched to see that there was no injurious pressure. He did not think operation was necessary with fractures of the carpus and metacarpus involving a joint, for extension over a palmar pad usually gave very good results, especially if combined with early passive movements of the fingers. In fractures of the neck of the femur good results usually followed the application of extension with the leg in the fully-abducted position, if the amount of separation was great and could not be otherwise controlled, or, if the case was seen late, operation, although very difficult, might be attempted. With regard to the lower end of the femur, especially in cases of separation of the epiphysis, the best results followed the method first advocated by Mr. Jonathan Hutchinson of applying extension to the lower fragment whilst the knee was fully flexed, and then fixing with the knee in the flexed position. In all the cases he had seen this method had been successful and operation had never been required.

The point upon which he chiefly wished to lay stress was that there was no one method applicable to all forms of fracture.

Mr. A. E. Johnson said that he was in almost complete agreement with Mr. Jones, believing that many fractures in the neighbourhood of joints could be treated in the most satisfactory manner without operative interference. Whether the cases were dealt with by operation or not, however, sooner or later they had to be treated by massage and exercises. In the case of the lower limb there was some difficulty in carrying out this treatment in hospital patients. As soon as they were able to get about on crutches they had to leave the wards in order to make room for other patients. Then, owing to difficulties and sometimes laziness, their attendances at the out-patient department were not frequent enough to insure good results being obtained. The solution of the difficulty was to arrange for the after-treatment to be carried out at branch hospitals or convalescent homes. This entailed the attendance of a medical man and the employment of nurses skilled in massage. At the Middlesex Hospital this plan had been tried, with gratifying results.

Though the subject of the treatment of the fractures of the patella was too large to be dealt with fully in a discussion of this character, Mr. Paterson, in advocating non-operative treatment, had made a statement which could not be allowed to pass. Mr. Paterson had argued that because the bone and not the union sometimes gave way in a fibrously-united patella, therefore fibrous union was at least as good as bony; but refracture of the patella was more common after fibrous than after bony union, and whether the fibrous union or one of the fragments gave way was a minor point. The real point was that owing to the fibrous union the patella was lengthened, the whole extensor arc weakened, and the liability to refracture increased.

The PRESIDENT (Mr. Rickman J. Godlee) thanked Mr. Robert Jones for his very interesting paper. He dwelt on the importance of the two general points upon which the author laid emphasis. One of those was the question of active and passive movements, and the differences between them, and the other was his view that perfect alignment was of more importance than very accurate apposition of the ends of the bones. It was interesting to hear passive movements spoken of as one of the traditions of the elders. Some of them could go back to the time when passive movement was not dreamed of for cases of Colles's and Pott's fractures, which used to be kept immobile for six or eight weeks. But now the swing of the pendulum was in another direction, and passive movements were again going under a cloud for a season. He agreed with Mr. Jones as to the danger of using passive movements too soon. He would like to mention a case apropos of treatment by operation for fractures of the neck of the femur, one which occurred before almost anyone except Lord Lister had commenced to treat fractures by operation. The case was that of a girl aged 19 at University College Hospital, who, after a slight slip, had separated the head of the femur. It was an intracapsular fracture, and, with some heroism, he opened the hip-joint, put his finger in, and ascertained that the head was loose, and did the operation which had been described that evening, except that he drove some ivory pins through the great trochanter and the neck of the bone and into the head, and glorified himself on the result of his operation. Years afterwards she had what Lister called a "lymph abscess" over her great trochanter, and in that abscess he found the ends of the ivory pegs, which had been set free by absorption, but the head of bone had again separated. He asked Mr. Jones what took place in the slight accidents which caused coxa vara. Was the epiphysis dragged off by the ligamentum teres, or was there slight separation and a subsequent yielding? One was struck, on examining stereoscopic skiagrams of cases of coxa vara which were supposed to be due to separation of epiphysis, and looked as if they were so, to see that, besides separation of the epiphysis, it appeared as if it had slid over the end of the diaphysis, and there was a bend upwards and forwards of the neck. Was it, then, a true separation of the head, or a gradual process which occurred in connexion with rickety changes? There was a description of fracture of the spine of the tibia in the last edition of "Erichsen's Surgery," and there were two specimens of this injury in University College. In one case a boy fell down on his flexed knees and tore off the spine of the tibia on one side and a piece of cartilage of the femur on the other side by means of the posterior crucial ligament. Another case was one in which a boy was brought in with a long flesh wound of the thigh. Mr. Erichsen amputated, because, he said, he would otherwise get erysipelas and gangrene, and die of blood-poisoning. The lower end of the anterior crucial ligament had torn off the spine of the tibia, and the joint was full of blood. It is figured and described in the *Illustrated Medical News*.[1] He would like to hear more about injury to the tubercle of the tibia; he had seen a number of schoolboys who complained of tenderness of that tubercle. He had not supposed they were due to fracture, but to periostitis. Most of them occurred before the introduction of X-rays. In the last case he saw he had a careful skiagram taken, and compared it with those of normal bones; it showed how many varieties there are in the manner of ossification of the little piece of bone which came down from the main epiphysis towards the tubercle. He wondered if Mr. Jones regarded the swelling as periostitis resulting from strain, or partial separation of the tubercle of the tibia from the shaft. This year he met with a curious injury of the elbow-joint, and he wondered whether Mr. Jones had seen a similar case. It seemed to be a dislocation forwards of the radius, and he had to operate so as to put it back. But the radius had torn off the whole of the orbicular ligament from the ulna. The only thing to be done was to put a piece of silk through the ligament and fix it down to the lesser sigmoid cavity. He did not see how one could apply a peg or wire in such a case. The result was good, but not perfect.

The paper was of great interest, and he wished there had been time to include a discussion of fracture of the acetabulum, especially where the head of the bone was driven through into the pelvis, and of fractures about the neck of the mandible.

Mr. Robert Jones, in reply, thanked Fellows for their kindly criticism, which showed that there was not any wide disagreement. He thought that if speakers gave their experiences it would teach extremists that there was more than one way of achieving the same result. In the hands of a clean surgeon he would not mind having almost any bone in his body wired. Again, if he could be assured of a good functional result without operation he would be willing to forego a more artistic one. He had not known of a description of fractures of the spine of the tibia, and he thanked the President for the description of his case. He agreed one often found a rounded condition of the neck in traumatic coxa vara, which assured one there was antecedent softening of bone. He had skiagrams showing separation of the epiphysis, starting from below as a little Λ-shaped space which gradually extended, resulting finally in a complete separation, the neck ultimately becoming rounded and resulting in a typical coxa vara. With regard to pegging or wiring where there was separation of the head of the bone in a young adult, he thought it unnecessary; if it were well abducted and extended there would be a good result. If it were decided to nail through the trochanter it should not be done without first exposing the joint. With regard to avulsion of the tibial tubercle, he had several photographs showing separation in various stages; the later photographs in some cases showing the separation more marked. Unless treated by complete rest there was likely to be much thickening over the tubercle and tenderness on pressure. That was best treated by making a linear incision into it with a chisel, just as in the case of sclerosed bone to relieve tension. He had seen this year two cases of Volkmann's ischæmic paralysis, so it did not yet belong to the past entirely. It took place as often from pressure within the arm as from pressure from without it. Cutting down on each tendon and lengthening it was not so effective as a simple procedure which he (Mr. Jones) demonstrated. Mr. Walton suggested that Bennett's fracture was never troublesome, but he (Mr. Jones) regarded it as one of the most difficult to treat mechanically, and he would be glad to hear afterwards what Mr. Walton did in that condition. Incomplete mobility so often occurred that he sometimes had to cut down to remove the fragment. Mr. Fagge said that Colles's fracture was very suitable for plating, but Mr. Jones thought Colles's fracture was one of the most amenable to simple treatment. [Mr. Jones demonstrated his method of reduction.] Colles's fractures which resulted badly he did not think could redound to the credit of either the surgeon or the patient. With reasonable co-operation there should be no bad result in cases of the kind. One or two photographs, which Mr. Fagge handed round, made one think it was almost better to take a photograph of a fracture long after the recovery and not when there were masses of callus exudation. In the fracture of the elbow which had been wired there was still a marked prominence above, showing that there was nothing like complete reduction there. In dealing with acute flexion it was not sufficient to put the arm into an angle of forty-five degrees, but the flexion and supination should be complete, the hand almost resting on the shoulder.

---

[1] Godlee, R. J., "Fracture of the Spine of the Tibia," *Illustr. Med. News*, 1888, i, p. 12.

If surgeons understood each other better, and saw more often what each did, it would be realized that the differences between them were not so very great. For instance, he did not regard passive movements as a superstition, as had been suggested. What he urged was that, if one were dealing with a case in which one did not want adhesions to form, it was better to put the joint through its complete movements once only in each direction and then keep it quietly in place for twenty-four hours until it recovered from the effects, instead of handing it over to the energetic masseur, who worked it vigorously with harmful effect.

Mr. Jones was much gratified by the discussion, from which he hoped some benefit had been derived.

Surgical Section.

December 13, 1910.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

Septicæmia Hæmorrhagica. Severe Capillary and Parenchymatous Hæmorrhages of Septic Origin.

By W. G. SPENCER, M.S.

THERE is a class of grave and fatal hæmorrhages which are not included under the subjects of primary or secondary hæmorrhages, nor can they be ascribed to any congenital lesion such as hæmophilia, nor to degenerative lesions, such as occur in Bright's disease or are induced by the toxic influence of alcohol. Indeed these causes of hæmorrhage are excluded from consideration in this paper, and none of the cases referred to were predisposed to hæmorrhage from these causes.

The peculiarity of the hæmorrhage I propose to discuss is that it may occur from a mucous or serous membrane without any naked-eye breach of surface; or it may happen from a raw surface without any special blood-vessel being opened; or if into an organ, there is parenchymatous hæmorrhage without any preliminary traumatism or degenerative process in that organ.

Such hæmorrhages are well-known complications of the specific and tropical fevers, as well as of scurvy. One cannot attribute such hæmorrhages to an excess of pulse tension, for indeed the hæmorrhages, whether from the nose or gastro-intestinal canal, continue to recur when the patient has become very weak.

In bringing forward some cases which have come under my observation, I am well aware that I can deal only inadequately with the essential part of the subject—the bacteriology. For that is the clue to the treatment, whether by an antitoxin given immediately or by autovaccination as soon as possible. Yet, however imperfect the bacteriological details, the cases I have seen have been so grave as to merit consideration, especially as there has been no satisfactory treatment hitherto.

My attention was first drawn to the subject by Dr. Klein's experiments on a variety of streptococcus causing hæmorrhage. In 1897 Dr. Klein [14] received from Dr. J. E. Colby, of Malton, fluid taken from vesicles on the skin of a man who, two or three days before, had been engaged in skinning sheep dead of puerperal septic œdema after lambing, the so-called "Gargel." Dr. Klein cultivated a streptococcus from the material, which, when injected subcutaneously into guinea-pigs, caused subcutaneous and intermuscular connective-tissue hæmorrhages around the site of inoculation, along with blood effusions into the pleural and peritoneal cavities. The same results followed the injection of cultures into rabbits and sheep.

SEPTICÆMIA HÆMORRHAGICA IN ANIMALS.

The term "septicæmia hæmorrhagica" is one adopted in veterinary pathology for an important class of epizootic fevers. It has been applied to the disease called in Italy "Barbone bufali," or buffalo disease, concerning which much has been written in that country [10] [16].

In 1878 Bollinger described the disease as causing a great fatality among deer, wild boars, cattle and horses, in or near the Royal Park at Munich, and in Bavaria generally. Lignière discovered in the so-called hæmorrhagic influenza of horses a coccobacillus; in the blood he found and cultivated streptococci only, but the streptococci could not be cultivated in a virulent form. In France a disease of cattle distinct from rinderpest has been termed Pasteurellosis bovis; in it there occur hæmorrhagic pneumonia, pleurisy, and peritonitis.

In India and Egypt hæmorrhages occur with malignant sore throat, or strangles, in horses and cattle, and even in hardier animals, such as mules and elephants [23] [7]. Lingard [15] has made extensive researches in respect to this disease in cattle and deer, terming it septicæmia hæmorrhagica pleuriformis. He produced a cultivation of the organism so virulent as to kill both hill and plain cattle with certainty in nineteen to forty-two hours. If the cultivation became weak it could be restored to virulence by passing through rabbits. The organisms described in these animal and tropical infections are variously referred to as bacilli, coccobacilli and streptococci ; but the resulting pathological changes have a general similarity.

Returning to human pathology, there occur cases of *hæmorrhagic empyema and ascites.* Oliver [17] has described cases of thoracic empyema followed by fatal hæmorrhagic oozing from the wall of the cavity ; the organisms found were the diplococci of Friedländer, not the pneumococcus. (In these cases injury to an intercostal artery or pulmonary vessel was excluded.) Hæmorrhagic ascites [9] has proved very fatal, doubtless due to infection by organisms, but I am not aware of any definite report as to the organisms present.

Under the term of *purpura* a collection of hæmorrhagic diseases are discussed in works on medicine. Some of these cases are traceable to streptococcal infection, especially when a complication of puerperal fever. *Melæna neonatorum* forms another class of fatal hæmorrhagic disease. The subject has been recently summarized by Thomson [21] in Allbutt and Rolleston's " System of Medicine." Among the organisms identified are not only streptococci, but also *Bacillus pyocyaneus*, staphylococci, short bacilli and Friedländer's pneumobacillus. Klebs [13] in 1875 found a micrococcus in nine new-born children who had died of melæna, the cultivation of which produced in young rabbits fatal hæmorrhagic lesions. In three cases which Bar [5] examined he found streptococci. On the other hand, Tizzoni and Giovanni [22] reported three children of the same family who died one after the other of a contagious pustular impetigo accompanied by hæmorrhage. *Staphylococcus pyogenes aureus* was the organism found in both primary and secondary lesions. The hæmorrhages were traced by microscopical examination to a thrombosis of the smaller vessels by the staphylococci.

The acute hæmorrhages which complicate *diphtheria of the fauces* are very fatal : fifty-eight cases were reported by Austen and Coghill [3], representing 6·59 per cent. of admissions ; these all died, the organisms found besides diphtheria bacilli being streptococci and staphylococci.

Acute hæmorrhages into the stomach as shown by black vomit constitute a grave complication. In medical practice they follow the specific and tropical fevers ; in surgical practice they occur late in such cases as intestinal obstruction, gall-bladder suppuration, and gangrenous appendicitis [6], and may follow operations when sepsis cannot be prevented. I have seen this fatal termination in the various forms stated, but unfortunately I have no bacteriological information to give.

Hæmorrhagic pancreatitis is a well-known form of parenchymatous hæmorrhage, in which the exact type of organism causing the affection cannot yet be stated with certainty. There does not appear to be sufficient reason to ascribe the hæmorrhages to *Bacillus coli communis*, which is the organism generally found in such abdominal complications.

Dr. F. W. Andrewes [1] has published the post-mortem examination of a case of *muscular hæmorrhage following typhoid fever*.

A man, aged 18, the subject of double chronic otitis media, was admitted to St. Bartholomew's Hospital during the third week of an attack of typhoid fever. Before the temperature become normal he had a relapse, and died on the fifty-second day. On the day before he died subcutaneous hæmorrhages appeared on the feet and elsewhere; in addition, there formed a considerable swelling in the left thigh with œdema of the limb below. Post mortem, in addition to the lesions characteristic of typhoid fever, there was found a diffuse extravasation of blood into the muscles. From the muscles of the thigh and from the blood-clot a pure cultivation of streptococci was obtained, but no typhoid bacilli. The streptococci were not evenly distributed in the muscles, but had especially collected about the arteries and veins. Of leucocytic aggregation there was little or no evidence.

Hæmorrhages of a similar character may be produced by poisons such as phosphorus [12] and benzine [19] both in human beings and, experimentally, in animals. It would seem likely that the actual influence on the blood vessels is through the agency of toxins produced by such poisons, and therefore the actual production of the hæmorrhages may essentially not differ from those originated by organisms.

HÆMORRHAGIC CELLULITIS.

I will in the first place refer to cases of acute hæmorrhagic cellulitis in which on incision no distinguishable amount of pus escaped, but blood poured out most freely.

Case I: Hæmorrhagic Cellulitis of the Arm.—A previously healthy man, aged 25, was admitted under my colleague, Mr. Stonham, with an extensive swelling of the arm and forearm, which had followed upon a slight abrasion of the hand. At first sight there appeared nothing uncommon in the case, the subcutaneous tissue of the arm and forearm seemed to be distended by pus. But no sooner was an incision made than blood-clot escaped, then fluid blood poured out, and the patient showed alarming signs of acute anæmia. Not a single vessel could be seen to bleed, although incisions were extended along the line of the blood vessels. It was only by very firm packing of every part of the involved area, followed by similar bandaging, that the bleeding could be controlled. There was no further hæmorrhage, the exposed tissue soon became covered by granulations, and healing followed.

Case II: Acute Hæmorrhagic Cellulitis of the Left Buttock and Thigh.—A labourer, aged 47, was admitted under me in a very weak state, but what attracted my attention was his lemon-coloured pallor. He had been ill for six weeks without evidence as to cause. He presented a deep-seated elastic resistance, filling the left iliac fossa, also the left buttock, and upper third of the thigh. In the thigh the swelling was more superficial, and the skin over it showed a dusky brown brawny œdema. The swelling did not pulsate nor yield a bruit, and the circulation in the left leg and foot was as good as on the opposite side. He had a very rapid pulse, 152 per minute, the respiration 40 per minute, the temperature 100·4° F. Impressed by his pallor, which I took to indicate concealed hæmorrhage, I made a narrow puncture with a knife in four places into the swollen thigh, keeping well away from any vessel. From each puncture bright blood poured out and quickly clotted. No pus was reached. Each puncture was plugged firmly with gauze. He would soon have bled to death, had any freer incision been made or a drainage tube been inserted. Dr. Lazarus-Barlow cultivated a diplococcus from the blood which had flowed out. At each dressing clots of blood with turbid serum escaped, but no pus. Locally the swelling got less, but the man became weaker, his temperature rose to 104° F., his pulse to 172, and he died three weeks after the incision. No post-mortem was allowed, but the nature of the case was unfortunately confirmed by the infection of two patients in neighbouring beds with erysipelas.

A young man was in the next bed with whitlow and palmar suppuration, and he had a severe attack of general erysipelas from which he recovered.

An old man in the opposite bed across the ward, who had tuberculous disease of the shoulder, was likewise attacked with general erysipelas, and he died. This happened in 1900, and among my cases in that ward there has not been an outbreak of erysipelas since.

Case III: Hæmorrhagic Cellulitis of the Thigh.—My colleague, Mr. Tubby, had a very similar case of hæmorrhagic cellulitis of the thigh; the patient was admitted in an even weaker state; blood flowed freely from the punctures made, and he died the same night.

Case IV: Hæmorrhagic Cellulitis round the Knee.—A rather less severe case than the foregoing occurred in a carpenter, aged 27, who, a month before, had felt on returning home at night great pain in his right knee. He therefore rubbed horse oils and turpentine into his knee; then the knee was poulticed for a fortnight; a week before admission a discharge commenced, and the swelling began to spread down the leg and up the thigh. On admission he was very pale, weak, and half delirious. On making an incision a little pus, but chiefly broken-down blood-clot, escaped, so that it was impossible to irrigate the cavities. As compared with the previous cases, blood did not pour out, but only re-collected in the cavity as clot. There was no sign of an arterial lesion or of disease of bone. A week later, the man in the meantime having improved a little, I amputated through the thigh, a little above the knee, and the patient recovered good health. The lower end of the thigh and upper part of the leg removed was extensively infiltrated by pus and broken-down blood-clot, but the bone was unaffected, and there was no lesion of the large blood vessels. Here there is no doubt that the cellulitis arose from the pustules raised on the skin by the horse oils followed by the poulticing.

Case V: Acute Subcutaneous Hæmorrhage following a Viper's Bite.—A man engaged in stealing pheasant's eggs was bitten by a viper, the two punctures being evident on the dorsum of the proximal interphalangeal joint of the middle finger. I saw him about two hours after, very pale and collapsed. His arm and forearm were very much swollen, bright red, and there was marked pulsation when one grasped the forearm. The swelling was due to extensive extravasation of blood into the subcutaneous tissue, and in the course of the next six weeks the colouration of the limb passed through the stages of red, blue, and yellow, as the blood-clot was slowly absorbed. Suppuration was limited to the area around the actual punctures made by the viper's fangs.

I pass on to quote the obscure cases of :—

Case VI : Hæmorrhagic Enteritis.—I saw a patient at the end of a long and obscure illness which had been carefully watched by Dr. Seymour Taylor and Dr. Michael. A girl, aged 24, had diarrhœa and at first occasional hæmorhages from the bowel ; vomiting and foul stools. Then she began to pass large quantities of blood by the stools, and lapsed into a state of acute anæmia, with a pulse-rate of 120 ; but at no time did the temperature vary much from normal. There was an absence of all signs of typhoid fever, and also of any sign in favour of the second hypothesis, tuberculous enteritis. On the day preceding death there was noted for the first time a tenderness in the right iliac fossa, followed by clear signs of commencing infective peritonitis. I could detect an enlarged appendix, yet the general condition of the patient was quite different from the common type of acute gangrenous appendicitis. She was very pale, with sunken cheeks ; but had no earthy look. The tongue was pale, moist, tooth-indented but not furred. The pulse was very rapid and weak, 130 or 140 per minute, but the temperature continued normal. The abdomen was a little tumid, but nowhere persistent nor tender, except over the appendix. The appendix was removed as quickly as possible. No gangrene nor pus, nor sign of typhoid fever or tubercle, was met with ; there was congestion of the peritoneum around and blood-stained fluid was sponged out of Douglas's pouch. The patient failed to react to saline infusion ; she died four and a half hours after the operation ; there was no post-mortem. The appendix removed was distended to the size of the end joint of the little finger by blood and mucus, but there was no evidence in the interior of gangrene or pus. The microscopical examination showed acute hæmorrhagic inflammation of the mucous and submucous coats, in which were numerous cocci and rods.

Several grave cases of hæmorrhage from the kidney and bladder have come under my notice which cannot be classed with common causes of hæmaturia.

Case VII: Renal Hæmorrhage.—An unmarried female, aged 20, had had for three years pain in the left side of a dull character, especially when standing and walking, with slight and irregular albuminuria, especially in the evening. In addition, she had had several attacks of severe pain with sweating and nausea. She had not passed pus or blood, and an X-ray examination was negative. The kidney could not be felt to be swollen, nor was it markedly movable. The pain became so severe as to prevent her from exercise and games; although when lying in bed the pain was less, yet she still felt it in the lumbar region radiating down over the hip, and a tender spot could be felt corresponding to the position of the pelvis of the kidney. An operation was decided upon on account of the persistence of the pain; the kidney when exposed did not seem very movable, so the interior was explored through an incision on the lower border just sufficient to admit a finger. Finding no sign of stone, the kidney was fixed by sutures and the wound sewn up except for a drain. The case took a normal course until a week after the operation, when menstruation set in, and with this there occurred free bleeding from the wound. To check this the drainage tube was removed and a plug of gauze inserted. Three days after the hæmorrhage the temperature rose, owing to decomposition of the clot, so the clot which had collected around the kidney was wiped out. Twenty days after the operation the patient commenced to have severe rigors, and as all the clot had been cleared out, and no tracking of pus could be discovered, the kidney was removed, twenty-four days after its fixation. The removed kidney was found to be infected by numerous small abscesses in the cortex due to staphylococci, which had supervened upon old-standing fibrotic changes, presumably caused by the intermittent kinking of the ureter. The renal veins were not thrombosed, nor was there any evidence that the hæmorrhage had come from a large branch of the renal artery. Auto-vaccination with a culture of the staphylococci failed to prevent the rigors from recurring. Repeated examinations failed to detect any pyæmic metastases, but the patient died thirty-two days after the operation. In this case the hæmorrhage seems to have been concomitant with the menstruation, which set in earlier than was expected but otherwise appeared normal. The infection of the blood-clot appears to have been by staphylococci lying latent in a kidney which had been affected by intermittent kinking of the ureter.

Case VIII: Renal Hæmorrhage.—For more than ten years a man, aged about 45, had had what was supposed to be intermittent albuminuria and attacks of pain in the *right* side which were thought to be colic. He perhaps occasionally had slight hæmaturia. He was next seized with acute pain on the *left* side with obstinate constipation for a week. After a second attack of acute pain on the *left* side, hæmaturia and albuminuria were noted. He was therefore examined by the X-rays. Then it was discovered that the position of the *right* kidney was occupied by a large branched calculus. On the *left* side in the region of the kidney pelvis was a stone the size of a large orange-pip. It then became evident that the *right* kidney had been slowly destroyed by a calculus; that the so-called intermittent albuminuria was traceable to the discharge of a small amount of pus; and that the left-sided attacks were due to a calculus temporarily blocking the *left* ureter and threatening life. Therefore I operated first upon the *left* kidney, which was enlarged and vascular, making a small opening with blunt forceps and extracting the stone from the pelvis. There was no special hæmorrhage at the time, but three days later the same thing happened as in the previous case: free bleeding set in, the wound had to be plugged, the clot which collected in the wound and around the kidney decomposed and had to be wiped away. The infection in this case was easy to understand. Fortunately, after the wiping and syringing out of the broken-down clot, no further hæmorrhage occurred. But the left ureter remained for a time blocked by clot. The patient improved, so that a month after the first operation I was able to remove the *right* kidney. This had been reduced to a friable abscess cavity enclosing a large branched calculus. The second operation wound healed in about six weeks. The first wound continued to discharge urine and very little passed into the bladder until three months from the operation, when the sinus had become very narrow. Then one day he was seized with pain down the course of the left ureter, the obstructing clot moved on, and urine passed into the bladder, upon which the sinus closed. The patient now, two and a half years after the operation, is following his occupation in fair health, but there are traces of pus in the urine.

Case IX: Renal Hæmorrhage.—A footman, aged 42, was admitted to hospital suffering from severe hæmaturia. He was in such acute pain that at first sight some abdominal perforation seemed probable. On further observation the pain seemed situated in the right lumbar region, extending down the right ureter. The urine contained a large amount of dark blood. Eighteen years before he had been attacked with severe pain in the right side and hæmaturia. Thirteen years before the pain recurred and he went into a hospital in London, where he was told that the pains were "functional." A year later he went into another London hospital, but received no active treatment. All that could be made out before operation was that he was markedly anæmic, and had a resistance in the right lumbar region. There was no fever, and nothing was discovered by X-ray examination. On incising the right lumbar region the transversalis fascia was found deeply infiltrated by blood. After cutting through the fascia a large amount of blood-clot was scooped out; some of the clot was recent, some of old standing. The roof of the cavity was formed by the under surface of the diaphragm and liver. The upper half of the kidney seemed to have disappeared in the mass of blood-clot. With much difficulty the pedicle was incompletely isolated and ligatured and cut across, leaving a little kidney substance within the grasp of the ligature. There was no sign of pus, nor was there a urinous or foul odour.

The ureter was patent and not thickened. The wound healed well, except for a sinus, which was persistent for two months; he has been seen lately in good health. One explanation of the case is that a mild infection caused parenchymatous hæmorrhage which destroyed most of the kidney; the other, a more mechanical hypothesis, that a kinking of the ureter was followed by hydronephrosis, or cystic formation, upon which partial rupture supervened.

The two following cases have been previously described in the Clinical Society's *Transactions* [20]:—

Case X: Renal Hæmorrhage Recurring.—A butler, aged 42, was sent into hospital by Dr. Bunn on account of recurring hæmaturia sufficient to cause anæmia. About eighteen months before he had awoke at night feeling "full of wind" and "unable to get his breath." Not at the time, but next morning, he passed blood-stained urine. Three months later he had another attack, commencing with pain in the hypogastrium. He had several more attacks, complicated in some by retention. He went into a hospital, where he was examined under an anæsthetic and by the X-rays; after which he was told that no operation was indicated. The examination did not provoke hæmaturia, but an attack recurred three days after leaving the hospital. He was then anæmic; had normal micturition four times a day. The urine was the colour of port wine, and specific gravity 1030, without excess of albumin, and it contained nothing abnormal but blood. Whilst lying in bed the patient continued to pass large clots, which sometimes obstructed micturition until a catheter was passed. It was impossible to examine with a cystoscope, so suprapubic cystotomy was performed. After washing away all the bloody urine and some sediment, the wall of the bladder looked quite normal; it showed no tendency to bleed as a result of sponging. Clear urine was collected from both ureters, and no clot or blood came from the urethra when a catheter was passed. The suprapubic wound was drained for three weeks, and he then passed urine by the urethra. For a year the patient had no return of hæmaturia. Afterwards by letter I was informed that he had had some slight recurrence.

Hammersley [11] has described a patient who had attacks of pain in the right kidney with severe hæmaturia which was relieved by removing an inflamed appendix, and he attributed the renal affection to infection spreading from the appendix.

Eve [8] recorded a case of fatal hæmaturia due to psorosperms.

Posner [18] found amœbæ in a man subject to attacks of hæmaturia.

Case XI: Hæmaturia, Fatal without Obvious Lesion.—A man, aged 44, first noticed blood in his urine a fortnight before death. He had previously been in good health: he had not been abroad; with the hæmaturia there was neither pain nor frequency of micturition. The hæmorrhage increased to an alarming extent and I saw the patient immediately after his admission to the hospital. He was then very anæmic, almost pulseless, and was breathing rapidly. The bladder was tensely distended but not tender. The temperature was below normal: there was no skin eruption. He was infused subcutaneously and *per rectum* with saline fluid: he passed urine containing a large quantity of blood; he gradually became pulseless, and died within twelve hours of admission. Dr. Hebb made a complete post-mortem examination, without finding anything to throw light upon the cause or source of the hæmorrhage. The bladder contained a small quantity of bloody urine, which was not found in either kidney pelvis. This is the only evidence in favour of the source of hæmorrhage being the bladder rather than the kidneys. No organisms were identified.

Perhaps this case is like those of which Gull spoke and named "renal epistaxis," and Klemperer called "angioneurosis."

CONCLUSION.

It is obvious that I have included in this paper cases which differ widely from one another. But they were all grave and obscure. Some cases of septicæmia hæmorrhagica have been certainly caused by streptococci. If the classification of Andrewes and Horder [2] be adopted, one may include under streptococci the pneumococci, coccobacilli, and other varieties which have been found.

The occurrence of the hæmorrhage may be explained by supposing the organisms to collect, as described by Andrewes, especially in contact with the walls of the blood-vessels. Under the effect of the toxins the walls of the vessels become weakened and give way, so as to allow the escape of blood. This occurs without intravascular thrombosis, which in ordinary acute inflammation must be the chief factor in preventing hæmorrhage. Neither is there any massive collection of leucocytes at the inflammatory focus, so that very little pus forms.

The identification of streptococci as the commonest organism suggests an immediate recourse to streptococcal antitoxin, whilst the identification of the particular organism is being made.

The first case of success by the use of streptococcal antitoxin was reported by Ballance and Abbott [4]. A finger was pricked at a post-mortem on a case of suppurative peritonitis, and acute septicæmia set in, complicated by cutaneous hæmorrhages. Immediate relief followed the use of streptococcal antitoxin.

When the infection is not due to streptococci, or is a mixed infection with staphylococci or *Bacillus pyocyaneus*, the preparation and injection of an auto-vaccine should be carried out as quickly as possible.

It has been noted above that the extravasated blood clots readily, and this explains the fact that the administration of calcium chloride has had no influence on the hæmorrhage.

REFERENCES.

[1] Andrewes. *Trans. Path. Soc. Lond.*, 1902, liii, p. 42.
[2] Andrewes and Horder. *Lancet*, 1906, ii, pp. 708 *et seq.*
[3] Austen and Coghill. *Brit. Med. Journ.*, 1895, i, p. 694.
[4] Ballance and Abbott. *Brit. Med. Journ.*, 1896, ii, p. 2.
[5] Bar. *Brit. Med. Journ.*, 1893, i, Suppl. 79.
[6] Dieulafoy and Duvergey. *Lancet*, 1908, i, p. 1225.
[7] Evans. J. W. *Journ. Trop. Vet. Sci.*, Calcutta, 1906, i, p. 263.
[8] Eve. *Trans. Path. Soc. Lond.*, 1889, xl, p. 444.
[9] Fischer. *Lancet*, 1900, i, p. 17.
[10] Friedberger and Fröhner. "Veterinary Pathology," 6th ed., 1908, ii, p. 274.
[11] Hammersley. *Lancet*, 1909, ii, p. 1366.
[12] Hann and Veale. *Lancet*, 1910, i, p. 163.
[13] Klebs. *Archiv f. exp. Path. und Pharm.*, Leipz., 1875, iv, p. 473.
[14] Klein, E. *Centralbl. f. Bakt.*, 1 Abt., Jena, 1897, xxii, p. 81.
[15] Lingard, A. "Annual Report of the Imperial Bacteriologist for 1902-3, Calcutta."
[16] Moore. "Pathology and Differential Diagnosis of the Infectious Diseases of Animals 1908.
[17] Oliver. *Lancet*, 1906, i, p. 901.
[18] Posner. *Berl. klin. Wochenschr.*, 1893, 675.
[19] Selling. *Johns Hopkins Hosp. Bull.*, Balt., 1910, xxi, p. 33.
[20] Spencer, W. G. *Trans. Clin. Soc. Lond.*, 1904, xxxvii, p. 156.
[21] Thomson, J. Allbutt and Rolleston, "System of Medicine," 2nd ed., 1909, v, p. 873.
[22] Tizzoni and Giovanni. *Beitr. z. path. Anat. u. allg. Path. v. Ziegler*, 1889, vi, p. 299.
[23] Webb. *Journ. Comp. Path. and Therap.*, 1907, xx, p. 97.

Gastric and Intestinal Hæmorrhages (probably of Septic Origin) occurring after Abdominal Operations.

By JONATHAN HUTCHINSON, F.R.C.S.

A. B., A WOMAN aged 48, was sent to me by Dr. M. W. Loy, of Gillingham, Kent, with symptoms of gall-stones complicated by inflammation in the region of the gall-bladder. For three years she had suffered from attacks of pain which started at the lower right costal margin and passed up to the right shoulder and between the scapulæ. These attacks were not accompanied by vomiting, nor were they followed by jaundice, but they were very severe and lasted several days. They were brought on by heavy work or by indiscretion in diet. In addition she had a constant sense of throbbing in the region of the liver, increased to actual pain when she sneezed or took a deep breath. As is usual with the patients with gall-stones, she was the subject of habitual constipation.

Examination of the abdomen revealed two lumps below the ninth right costal cartilage: one superficial to the muscles and adherent to the skin; the other beneath the muscles, movable from side to side, continuous with the liver, and evidently a distended gall-bladder. She was not jaundiced and the urine was normal. The liver dullness was normal in extent.

Operation (November 18, 1909): Four-inch incision made vertically at outer border of right rectus and below costal margin. In the muscle layer a lump was found which, when cut, had somewhat the appearance of a gumma; it proved to be the thick wall of an abscess situated between diaphragm and liver. The peritoneal cavity was opened and the gall-bladder found to be very adherent to under-surface of liver; gall-stones were felt in its interior, and one especially close to the cystic duct; the peritoneal cavity was carefully closed off by means of flat sponges and gauze. The subphrenic abscess was then opened up by the channel it had made for itself to the abdominal wall. The patient was turned on her right side and the contents were allowed to escape; they consisted chiefly of thin, watery pus and a little blood. The cavity was explored with the finger and proved to be median—i.e., in the falciform ligament. Subsequent bacteriological examination of the fluid proved it to be sterile. The abscess surface was then shut off with sponges held back by retractors reaching into peritoneal cavity; the edge of the liver was held up by the fingers of an assistant. It was decided to perform cholecystectomy. The laborious work of paring the gall-bladder of its dense adhesions began. The liver was right under the costal margin, and in consequence the work had to be done at a great depth from the surface. The gall-bladder was incised and three large stones and several smaller ones were extracted. Very little bile escaped. The neck of the cystic duct was then tied, and in cutting it away an artery in the neighbourhood of the portal vein was cut. This vessel was the cystic artery at its origin from the right hepatic. The hæmorrhage was very free, and much difficulty was experienced in securing the vessel, which was ultimately tied with silk. The gall-bladder was finally removed, the two cavities left above and below the liver were isolated and separately drained, rubber tubes and gauze packing being employed for both. Silkworm gut was used to unite the wound.

I wish to emphasize the difficult nature of the operation. Cholecystectomy is not infrequently a trying performance for the surgeon, and this particular case (owing greatly to the depth and the obliquity of the gall-bladder and also to the many firm adhesions around it) was the most difficult one I have as yet met with. It was a case ill-suited for cholecystotomy alone, but possibly this would have been the wiser course to pursue in view of the presence of an abscess in the falciform ligament. It was, however, natural to attempt the removal of the cause of this abscess, just as one prefers in dealing with appendix abscess to remove that organ if it can be done safely.

The patient went on fairly well for six days, her temperature being normal, and the vomiting, which is so frequent an occurrence after difficult operations in the epigastric region where drainage-tubes have to be used, was the only trouble during this time. The gauze and tubes were removed and replaced by smaller tubes; on the seventh day it was found that the lower tube was blocked with blood-clot, and the next day she vomited 2 oz. or 3 oz. of bright blood. The wound looked somewhat red and angry, but there was no suppuration. Her temperature, which had been normal, rose slightly (to 100° F.), the hæmatemesis recurred, and some hæmorrhage also took place through the tube. Various drugs were tried without avail, and, as the patient was rapidly losing ground, I decided to explore the stomach. This was done, on the eleventh day after the first operation, through a median incision. No peritonitis was found; on opening the stomach and examining the mucous membrane through a speculum, I found three spots from which arterial blood was oozing. Two of these were cauterized, the third (a definite erosion on the posterior wall) was encircled with a fine purse-string suture. The opening in the stomach was sewn up with a double row of continuous sutures. Vomiting recurred, and after a day or two melæna showed that fresh bleeding was taking place. Continuous saline infusion was tried, but the patient became steadily weaker, and died six days after the second operation, her temperature having risen to from 102° F. to 103° F.

Post-mortem: Slight amount of blood in peritoneal cavity; a hæmatoma in the site of the removed gall-bladder. A fine opening existed in the right hepatic artery where the cystic branch had come off. Blood-clot in falciform ligament (site of abscess). Local fibrous peritonitis. Great fatty degeneration of the liver with cholangitis (?) Slight atheroma of aorta. Small aneurysm on right middle cerebral artery. Broncho-pneumonia and purulent bronchitis. Spleen large and soft; perisplenitis. Great anæmia of kidneys. Dermoid cyst in right ovary.

The foregoing case may be summed up thus: Gall-stones and chronic cholecystitis, complicated by an abscess in the falciform ligament (i.e., subphrenic). No clinical signs of pyæmia. Operation: The abscess cleared out and drained; pus thin and watery, found to be sterile on examination. Cholecystectomy, a prolonged and difficult procedure in this case. Subsequent vomiting on several occasions, and on tenth day after the operation sharp hæmatemesis. Arterial blood vomited at intervals. On twelfth day after operation the stomach was opened and bleeding-point (erosion) secured by ligature. Melæna and vomiting continued until death on sixteenth day.

This was the first case under my care in which unexplained hæmorrhage into stomach or intestine had followed an operation, and the problems presented by it, both as to diagnosis and treatment, caused me grave anxiety. It may be noted at once that the decision to perform gastrotomy and the discovery of a bleeding erosion in the stomach wall seem to have had no effect upon the progress of the case, either good or bad. It can be justified in such cases, though hardly on the ground of actual curative results. Of the twenty-four cases to be summarized later, this was the only one in which operation was resorted to.

The chief problem with regard to this grave complication after operations is as to its cause. I commenced to collect records of such other cases that had occurred in the London Hospital, and found to my relief that Mr. A. J. Walton, our Surgical Registrar, had already gone over the ground during the years 1907, 1908, and 1909. A valuable and surprisingly large collection of cases had thus been noted, and I am indebted to my colleague, Mr. Sherren, for whom it had been prepared, for the opportunity of using it.

During the three years mentioned no fewer than twenty-four patients died after operation in the London Hospital with the complication of recent acute ulcers or hæmorrhagic erosions in the stomach or duodenum. Three of these cases were examples of acute ulcers of considerable size. The following are brief particulars :—

Case I.—W. M., aged 32, admitted with diffuse peritonitis due to appendicitis. The abscess was drained and the appendix removed. Two days later he began to vomit blood in large quantities, and died of acute anæmia. Post mortem : A large recent ulcer was found on the lesser curvature. Much altered blood in stomach and intestines.

Case II.—R. B., aged 37, admitted for severe lacerated wound of leg. This was cleansed and sutured, but spreading gangrene supervened ; he died five days later. There had been melæna, but no hæmatemesis. Post mortem : An acute ulcer was found on the front wall of the first part of the duodenum.

Case III.—J. S., aged 21, admitted with appendix abscess. The appendix was removed and the abscess drained, but thoracic complications—empyema and subphrenic abscess—supervened. Both abscesses were drained, but the patient succumbed at the end of a month. Post mortem : Two recent gastric ulcers were found on the anterior wall of the stomach near the pylorus.

Far more numerous are the cases of multiple erosions of the mucous membrane, some causing such persistent loss of blood as to bring about the patient's death, others merely assisting in this end. Out of twenty-one cases the majority come into a very distinct group—appendicitis with septic complications, either localized abscess or diffuse peritonitis. This group includes eleven (i.e., 55 per cent.) ; in most the operation had consisted in drainage of the abscess with removal of the appendix, in a few only drainage had been employed, and in one the abscess had already opened and the "operation" consisted merely in dilating the aperture. The virulent nature of the septic process in this group of cases was shown by such complications as empyema, subphrenic abscess, purulent broncho-pneumonia, &c. The only case that requires special mention is the following :—

Case IV.—F. G., aged 50, admitted for right lumbar abscess of about three weeks' duration. This was drained, and the pus was found to contain the *Bacillus coli*. The appendix was not seen. On the tenth day after operation severe hæmatemesis came on ; this recurred (with melæna) repeatedly during the next five days, and gradually ceased. No operation was performed besides the original abscess incision. It must remain uncertain whether the cause of the abscess lay in the duodenum, the vermiform appendix, or elsewhere.

This case is noteworthy, as it proves the possibility of recovery. During the three years I believe there was only one other instance of this, though of course it is probable that others may have been overlooked, those in which slight melæna only was present. Whether this is so or not, we must conclude that the prognosis of well-marked hæmatemesis and melæna after an operation is exceedingly grave. Probably not more than 5 per cent. to 10 per cent. of the patients recover. The reason will have been already inferred, that the gastric acute erosions or ulcers are due, not to any peculiar influence exerted by the operation itself, but to the septic intoxication or infection which already existed. In the fourteen cases considered so far, the septic condition was due to appendicitis in thirteen, to gangrene of the leg in one.

An analysis of the remaining ten cases bears out the importance of sepsis as a cause, though there are a few striking exceptions:—

	Cases
Cholecystectomy (for inflamed gall-bladder with calculi), in one case with subphrenic abscess, in the other with cancer of duodenum	2
Cholecyst-enterostomy (for cancer of the pancreas, &c.)	1
Supra-pubic cystotomy for papilloma	1
External urethrotomy, peri-vesical suppuration	1
Cancer of colon, colotomy	1
Strangulated umbilical hernia, colotomy, abscess	1
Chronic peritonitis, intestinal obstruction, colotomy	1
Pan-hysterectomy for fibroids, followed by diffuse peritonitis	1
Abdominal section for recurrent intestinal obstruction—? ileus, nothing found	1
Total	10

It will be seen that in only five out of a total of twenty-four cases—i.e., 20 per cent.—was a condition of sepsis absent at the time the operation was performed. In two or three of these five it rapidly followed the operation.

Two cases remain to be noted of special interest in which the existence of a chronic ulcer of stomach or duodenum was revealed after an operation for a totally different disease.

Case V.—J. N., aged 66, underwent four operations for epithelioma of the tongue. Seventeen days after the last operation, at a time when the operation wound had healed, he was attacked with severe hæmatemesis and melæna, from the effects of which he died. Post mortem: A chronic ulcer was found on the lesser curvature of the stomach, which had eroded the coronary artery. There had been no symptoms pointing to the existence of the ulcer, and there was no septic cause.

Case VI.—S. G., aged 41, abdominal hysterectomy (by Wertheim's method) performed for cancer of the uterus. The operation had to be incomplete owing to the extent of the disease. Eight days after the operation severe melæna came on. Post mortem: A chronic ulcer was found on the posterior wall of the duodenum.

In both these cases the operation was performed for cancer, and in both it seems that the ulcer, whether duodenal or gastric, had been practically latent, so that any symptoms due to it were wholly concealed by the major trouble.

It has been shown that previous sepsis is the main factor in the causation of gastric and intestinal hæmorrhage after operation; that the operation and the operator are alike guiltless save in the rare cases when the sepsis has followed the operation. Nevertheless, in a small proportion of cases (certainly under 10 per cent.), if some poison is responsible for the production of gastric or intestinal erosions, it is not an ordinary septic one. It will be interesting to test these conclusions by cases of similar nature where no operation has been performed, where acute ulcer or erosions have developed whilst the patient was under medical treatment for some other disease.

During the three years 1907 to 1909 the total number of post-mortems made at the London Hospital reached 3,755. Mr. Walton collected from this list sixteen examples of the kind referred to. Of these there were:—

	Cases
Multiple erosions of the stomach	9
Larger acute ulcers of the stomach	6
Ulcers of the duodenum and adjacent part of jejunum	1
	16

None of these patients were admitted with symptoms referable to the stomach or duodenum, the main cause of death being the following:—

	Cases
Ulcerative endocarditis	3
Pyæmic pericarditis and endocarditis	1
Purulent bronchitis, &c.	2
Purulent peritonitis	1
Severe burns	2
Tuberculous peritonitis, acute cystitis	1
Acute septicæmia after childbirth	2
Diabetic gangrene	1
	13

In all of these the septic element is either obvious or may fairly be invoked.

In addition, there are only three in which sepsis could be excluded—namely, two cases of chronic nephritis terminating in uræmic coma, and one of isolated cancer of the gall-bladder.

One of the cases of burns deserves to be quoted :—

Case VII.—N. D., aged 3 years, admitted with burns of the chest and abdomen, &c. Post mortem : Minute hæmorrhagic ulcers of the stomach, due, as the microscope showed, to miliary abscesses in the follicles, one of which had burst at the surface.

It is remarkable that the same proportion of non-septic cases (20 per cent.) prevails in the medical series as in the surgical one. An extreme lowering of the vital strength (e.g., from internal cancer, chronic nephritis) is the only cause of gastric erosion that seems to be common to these non-septic cases. A similar profound asthenia is certainly present in most of the septic ones, and may be more important than any particular micro-organism in the blood. It is this that probably explains the absence of marked fever in many of the cases, the temperature and pulse-rate remaining near the normal, and thus the nature of the infection may be made obscure.

CONCLUSIONS.

(1) Hæmorrhage into stomach and intestine due to small ulcers or erosions may develop after an operation, usually abdominal, has been performed. In 80 per cent. of the cases a severe septic infection, with secondary anæmia, is present before the operation, which in itself has no direct influence upon the hæmorrhage.

(2) Similar lesions of the stomach or duodenum are met with in certain cases of disease where no operation has been performed ; 80 per cent. of these cases are septic in nature.

(3) It is probable that a profound alteration in the blood, due in most cases to toxins of septic origin, is the chief factor in the production of these gastric erosions.

(4) Such hæmorrhage is of very grave significance, though a few patients recover from it.

(5) Operative interference does not appear to hold out much prospect of success in these cases.

DISCUSSION.

The PRESIDENT (Mr. Rickman J. Godlee) said that such a large dish had been set before the Section that it was almost impossible to assimilate it all at once. He had wondered whether some proportion of the cases under consideration might not be due to something else besides septic changes. In the kidney cases, for instance, one would have liked to know what was the specific gravity of the urine. It had been for a long time his opinion that in advanced calculous disease, when the specific gravity of the urine was low, there was very great risk of hæmorrhage, so that he felt very shy of operating on them. Last year he had such a patient at University College Hospital whose kidneys were full of stones, and he pointed out to the class how dangerous it was to operate, but, after considerable hesitation, he decided to tackle one kidney. The patient did well for a few days, and then began to bleed both into the bladder and from the wound, and continued to do so, until he ultimately died of hæmorrhage. And that was not the only case of the kind which he could mention. With regard to the cases of hæmorrhagic cellulitis, he did not remember seeing one in which pure blood only came away, but he had seen many in which the amount of pus was but small and the amount of blood large. If they were acute inflammations, he supposed they should come under the heading of erysipelas. But he would like to hear more about the acuteness of some of the cases which had been recorded. Where the inflammation was more chronic and the amount of blood was large and the pus small in amount a careful look-out should be kept for actinomycosis. With regard to the interesting series of gastric cases with which Mr. Hutchinson's paper dealt, one would like to know about the anæsthetic. He had often thought an interesting paper might be written on the influence of anæsthetics in producing black vomit. He was aware that Mr. Hutchinson was not dealing only with cases where there was black vomit, but he (Mr. Godlee) thought the anæsthetic might have something to do with the bleeding even in those mentioned by Mr. Hutchinson where chronic ulcer of the stomach or duodenum became manifest after the operation. That was brought forcibly to his mind in a case which occurred in the practice of Lord Lister, where he removed a patient's breast and she died of hæmorrhage from a gastric ulcer very shortly after the operation. He believed Lord Lister had a second case of the kind. It might also explain a case which occurred to him, in which he removed the rectum of an old gentleman by the combined method, the result of which was very satisfactory. After ten days he began to have melæna, which persisted for a long time. After it stopped a large lump was found in the region of the duodenum; but that disappeared in course of time, and the man was now quite well. But there were other cases which closely resembled those related by Mr. Hutchinson in which there was no evidence of septicity. For example he would mention that of a lady whose home was in India and who had passed gall-stones on several occasions. Shortly after one of these attacks he was urged by the physician under whose care she was to see if any gall-stones were left in the gall-bladder, because she was intending to return to India. Rather against his better judgment he agreed to explore, and found all the parts apparently quite normal, so the gall-bladder was not opened and the abdominal wound was at once closed. She vomited after the operation and the vomited matter was blood-stained; this continued for a week, so that her condition caused great anxiety. At the end of this time it stopped and there was no recurrence. It might be asked if this was due to the anæsthetic or to the stretching of some ulcerated surface caused by the recent passage of gall-stones. Another similar case was that of an old gentleman from whom he (Mr. Godlee) had removed a tumour of the sigmoid flexure, and whose wound followed an apparently completely aseptic course. Here the same thing occurred, but, although blood was vomited for several days, the general health of the patient did not appear to suffer at all. This patient had to submit to a second slight operation on another occasion for strengthening the scar, and the same thing happened. It had been suggested that some cases of this kind were caused by an obstruction, real or paralytic, of the bowel; but such an explanation would hardly hold good in the present instance. He suggested that some such cases were not septic at all. Two cases which he had seen corresponded with those which Mr. Spencer had brought forward.

A young unmarried woman, aged 23, was transferred to his care from Dr. Sidney Martin's ward at University College Hospital, on account of a left empyema, on March 12, 1909. She was, he believed, an Austrian, and of a highly nervous temperament. She was said to have had "sleeping trances" intermittently for two or three months, five years before admission. She had had "rheumatism," and was breathless ten weeks before admission. The illness began three weeks before admission with pain in the side and general weakness, and there was nothing special to note about the empyema, except that in the medical ward it was tapped (March 7) and turbid fluid drawn off, which was said to contain pneumococci. The pleura was opened on March 12, giving vent to a large quantity of offensive pus and lymph, and nothing unusual occurred for three or four days, when hæmorrhagic sores appeared both beneath and beyond the dressing and the fingers became affected with what looked like chilblains. The gums became swollen and spongy, and, like the dry and cracked tongue, bled freely, as did the spreading sores and the swollen granulations about the opening into the chest. But apparently there was no bleeding from the pleural cavity. On March 16 she was given 10 gr. of calcium lactate three times a day, and on March 24 this was replaced by calcium chloride 30 gr. three times a day, though by this time the bleeding was less, the sores were healing, and the chilblains were clearing up. On March 24 some pus was examined by the pathologists, and was said to contain staphylococci, streptococci, and *Micrococcus tetragenus*; but no pneumococci were found. It should be added that early on in the case cyanide dressings had been replaced by boric-acid fomentations. The following day 20 c.c. of antistreptococcic serum were injected. From this time there was a gradual improvement, and she left the hospital with the wound almost closed on May 6, and a short time afterwards healing was complete. Three months afterwards she died of an acute illness, the cause of death being certified as uræmia. This patient was very ill during the period when the bleeding was occurring. Her temperature, which was 103° F. before the operation, did not fall to normal until April 27, and, though during this time it usually ranged between 98° F. and 100° F., there were two or three sudden temporary rises to 102° F. or thereabouts. The pulse was frequent—100 to 120. The urine had a specific gravity of 1025 and contained no abnormal ingredients. The pathologist's report on the blood on March 24 was: Red cells, 3,400,000 per cubic millimetre; vacuolation, some anisocytosis and poikilocytosis; white cells, 22,000 per cubic millimetre; large lymphocytes, 5 per cent.; small lymphocytes, 13 per cent.; hyaline, 1 per cent.; polymorphonucleophiles, 81 per cent.; polymorpho-, basi- and oxyophiles, 0 per cent.; coagulation time, two and a half minutes.

Another case was that of a young lady who, after her first confinement, had pelvic cellulitis and a pelvic abscess which burst into the vagina. An abscess formed over the sacrum, which was opened by a small incision when it was almost pointing. No vessel of importance was divided, but copious and prolonged oozing took place from the abscess. This was followed by effusion, apparently of blood, into the left pleura, as only a small quantity of blood could be drawn off which contained streptococci. Later on an empyema developed on this side. After a long and very serious illness, during which numerous peritoneal bands formed, causing intestinal obstruction which necessitated a formidable operation during which five of these bands were divided, this patient made a complete recovery.

Mr. SPENCER, in reply, thanked the President for his remarks. He (Mr. Spencer) did not include in his series any case of Bright's disease. He remembered, as a dresser, being taught by Mr. Bowlby that Bright's disease was a common cause of hæmaturia, and his colleague, Dr. de Havilland Hall, had constantly referred to it. In cutting into his two kidney cases he did not perceive any disease of the kidney. In one case he cut out a stone, but that stone had not set up obvious necrotic changes preliminary to the hæmorrhage. With regard to hæmorrhagic cellulitis, he had often to stop an incision and plug quickly, but there was always pus. But in the case he related fluid blood simply poured out, and there was no pus afterwards at all.

Mr. HUTCHINSON, in reply, thanked the President for his valuable suggestion to inquire into the question of the influence of anæsthetics in the production of the subsequent hæmatemesis. But he thought some of the cases could not be explained in any such way, owing to the time that elapsed between the operation and the onset of hæmatemesis. In some the hæmatemesis had come on within twenty-four hours of the operation, but in some it was delayed for a week or ten days. It should also be remembered that in the sixteen cases quoted from the medical wards no anæsthetic had been given at all. With regard to the emphasis which he laid—possibly unduly—on the septic nature of most of the cases recorded, he had expressly pointed out that from 20 to 25 per cent. did not show a definite septic origin. This was interesting, as tending to disprove the idea that there was a specific germ in these cases. It seemed certain there could not be. Although his paper dealt with only a small part of a big subject, he thought it had been worth putting together, as showing, amongst other things, that there were definite gastric erosions in the majority of such cases. The autopsies had been most carefully done, and in twenty-one out of twenty-four there were definite small multiple erosions, nearly always in the stomach, though in some in the duodenum. In the case on which he operated there could be no doubt as to their existence; he saw them plainly, and the arterial blood coming from them. That knowledge might affect our treatment as well as our views as to the cause of the hæmorrhage.

Transverse Fractures of the Patella, without Separation of the Fragments.

By A. E. Johnson, F.R.C.S.

The causes of separation of the fragments of a transversely fractured patella, and the causes of failure to obtain bony union in cases in which much separation has occurred, have been of the greatest interest to surgeons for some considerable time. The subject was first studied scientifically by Gulliver [19], whose classical experiments laid the foundation of our knowledge of this subject. He performed eleven experiments on dogs and rabbits, in five of which he divided the patella transversely together with its aponeurosis, and in the other six divided the bone only, leaving the aponeurosis intact. In the five in which the aponeurosis was divided there was separation of the fragments, and only fibrous union resulted. In the six in which the bone only was divided there was no separation of the fragments, and firm union was the result in all. The union was bony in five of these cases, but in one patella, removed only twenty-seven days after the experiment, there was short ligamentous union only, and no new osseous tissue had appeared. Amongst Gulliver's conclusions we find the following: "Osseous union is simply the effect of immovable coaptation of the fragments, the provision for which, in certain forms of fracture, is the integrity of the aponeurosis in front of the bone." Gulliver, therefore, so long ago as 1837, only just failed to grasp fully the pathology of the failure of bony union in most of these cases. He knew that the rupture of the aponeurosis was the primary cause, but missed the fact, observed and pointed out by Macewen [29], nearly fifty years later, that the aponeurosis does not give way opposite the seat of fracture, with the result that the fringe attached to one part, generally the upper, falls between the fragments.

Macewen's clinical observations established the fact that the interposition of the præpatellar fibrous structures between the fragments was the cause of failure of bony union, and his deductions from his observations drew a picture of the pathology of transverse fracture of the patella which has been amply borne out by later observations made with greater facilities. Since the introduction of the X-rays further proof has been forthcoming, and the recognition of transverse fractures, without any separation of the fragments, which could not be diagnosed with certainty before the days of skiagraphy, has furnished additional evidence of the truth of Macewen's statements.

From the ease with which such fractures can be missed, and the frequency with which they are found when specially looked for, one is led to infer that before the introduction of the X-rays many cases without separation of the fragments were not diagnosed at all, or only after a further trauma had "completed" the fracture. Support is given to this view by the following: Malgaigne [34] described the case of a man who, several years earlier, broke his left patella, and in February, 1839, fell and broke the union. In March, 1839, he died, and at the post-mortem examination it was found that the right patella had also been broken and united by bone. This fracture of the right patella had not been suspected during life, and Malgaigne's drawings [35] show how close the union was. Again, Broca [8] [9], described a case of fracture of the patella by direct violence, in which the fragments were held together for fourteen days, when a false step and the slight muscular exertion necessary to prevent a fall led to tearing of the aponeurosis and separation of the fragments. Broca also quotes two similar cases reported by Malgaigne. In one of them a medical man received a violent blow on the knee from the wheel of a carriage and continued to walk; but the next day, whilst descending a staircase he slipped, made an effort to recover himself, and the fracture was "completed." In the second case a woman, who had a violent contusion on the knee, continued to walk, and at the end of a month, whilst making a slight exertion, separated the fragments.

Broca's case is of special interest, in that there was positive proof that the first fall really did lead to a fracture of the patella by direct violence, for, at the subsequent operation, it was found that the lower fragment was split into two halves, the strain at a later date having caused separation at the site of the transverse fracture.

In 1887 Macewen [30] himself related an interesting case. A young medical man received an injury to his knee in a football match. He could, however, walk without much difficulty. On examining his patella no line of fracture could be found. Still, osseous crepitus was detected on lateral movement. The patient went about with a bandage applied for three weeks. Firm, bony union took place. Macewen first saw the limb some years later, when there was only a transverse ridge on the patella to mark the seat of the former fracture. After describing this case, Macewen expresses the opinion that "it is highly probable that those specimens which are occasionally seen of osseous union after transverse fracture of the patella have been obtained from patellæ which were fractured without accompanying rupture of the præpatellar ligamentous structures," an opinion which has been endorsed since the introduction of the X-rays, and the frequency of the occurrence of such fractures has become more apparent. I would here mention a case reported by Vallas [53], in 1889, in which a man fractured his patella by direct violence and became unconscious. He therefore made no attempt to rise, and there was no separation of the fragments. No operation was performed. In 1905 the same man died from Ludwig's angina, and the patella was obtained. It was described by Vallas [54], and it was impossible to find a solution of continuity, either by direct examination or by the X-rays.

Since the use of X-rays has become more of a routine measure in cases of possible fracture a few further remarkable cases have been reported, the most striking being three described by Wegner [57], who, by employing the X-rays systematically in all cases of injury, was able to demonstrate some cases of fracture of the patella which had not been diagnosed before. Since they were the first cases of this nature which had been clearly described and demonstrated by skiagrams, I venture to quote them rather fully, especially as at present they are only on record in the German language. They are of the greatest interest when studied with some cases of my own which, as far as I have been able to find, are the only other similar cases clearly demonstrated.

In Wegner's first case a man, aged 35, slipped and wricked his knee. He did not fall, and after walking about twenty paces in considerable pain he was driven home. His medical attendant diagnosed an internal derangement, and treated it for four weeks with fixation and then adopted massage. Two months after the accident the patient returned to light work, but had to abandon it, and four months after the accident he went to hospital. There was some atrophy of the thigh muscles, but the patella could be easily moved, was not enlarged nor uneven, and movements in the knee were free and without grating. A skiagram revealed a transverse fracture of the patella, and the plate which Wegner publishes shows that there was practically no separation of the fragments. With massage and exercises the muscles were restored and a good result was obtained.

The second case was that of a workman, aged 41, who is stated to have fallen on his left knee as he was carrying a chest of drawers downstairs, thinking he had only one instead of two stairs to go down. He walked home in spite of acute pain in the knee, stayed in bed for one day, and then went a twelve minutes' walk without stick or other help to see his medical man, having to ascend two steps to get to the house. He walked home again alone. He was admitted to hospital three days after the injury. The effusion which was present disappeared after four weeks' compression, but on account of pain on the inside of the knee a further examination was made, and it was decided that the patient had a loose body in his joint. A skiagram, which Wegner publishes, was taken to decide this point, when it was found that the patient had also a transverse fracture of the patella, with practically no separation of the fragments, although a careful examination had failed to detect any pain at the site of the fracture or any crack or separate mobility of the fragments. The patient had had no difficulty in extending the limb or raising it from the splint. The loose body was removed and the patient made an uninterrupted recovery. Bony union occurred, and on the X-ray plate, with the exception of a small ridge on the anterior surface of the patella, Wegner states that there was no trace of injury.

The third case reported by Wegner is that of a workman, aged 44, who struck his right knee against the rung of a ladder which he was ascending. He felt a crack, but, in spite of acute pain, went a seven minutes' walk and ascended three storeys to his room, descended again to see the surgeon, again ascended, and two days later, on the advice of his medical man, went to hospital, again descending the three storeys alone. With the exception of a slight bruising of the soft parts at the sides and in front of the patella there was no sign of injury, and no sign of fracture of the patella, though this was specially looked for. A skiagram, which Wegner reproduces, shows the presence of a transverse fracture of the patella near its middle, without separation of the fragments. Treatment consisted of enclosure in plaster of Paris for six weeks, after which massage and exercises were employed. Convalescence was uninterrupted, and after three months Wegner states that no signs of a fracture could be detected by the X-rays.

In 1900 Barker [2, 3] showed a somewhat similar case at a meeting of the Clinical Society. This was a case of fracture by direct violence, with no palpable displacement, which was discovered by the aid of the X-rays. The plate which was published, however, shows a separation of about a third of an inch, which is much more than in Wegner's cases or my own. Cauthier [10] reported a case of transverse fracture of the patella due to direct violence, without appreciable separation of the fragments, in which it was not until the man had been under treatment for eight days that a fracture was diagnosed. Cases which fall into the same category have been mentioned also by Theim [49], Mayer [38], Doberauer [16], Smith [47], and Humblet [22]. Trésoret [51, 52] has also reported a case of fracture by direct violence which should be included amongst those we are considering, for though the man went about his usual work in the field, walked 2 km. directly after the accident, and 13 km. the next day, he did not seek advice for more than two months afterwards, yet the union between the fragments was so narrow that it only measured some lines in breadth.

Blackham [7] has reported a case where the lower tip of the patella was broken off, without the fragment being separated from the main part of the bone; and Bennett [6] showed, at a meeting of the Dublin Pathological Society, a specimen obtained from the dissecting room in which the same thing had occurred, though there was no history to the case. In Scudder's book [46] there is a tracing of an X-ray plate of a similar fracture. In these cases of fracture of the tip of the patella the line of fracture is extra-articular, and separation of the fragments seldom takes place, since at this point the bone lies in the thick and strong ligamentum patellæ. They have been fully described by Rödiger [45]. Such fractures are very rare, and of but little importance compared with transverse fractures through the body of the patella without separation of the fragments. The fractures of the tip of the bone get well without treatment; whereas in fractures through the body of the patella there is danger of some slight muscular exertion at a later date leading to separation of the fragments. Moreover, the joint being involved makes the injury a more serious one.

An extraordinary variety of transverse fracture of the patella is referred to by Reichel [43] and Magruder [32], in which the fracture is incomplete, not involving the cartilage. I have never seen a skiagram of such a case, which, of course, could not be diagnosed without the use of the X-rays, and there do not seem to be any published. Such cases would be very like cases of incomplete fracture of the olecranon, which I have described elsewhere [24, 25], but if an incomplete fracture of the patella does occur, it is much more remarkable than a similar injury to the olecranon. It must be remembered, however, that it is easy for the fracture to appear incomplete in a skiagram when the fissure does really extend through the whole thickness of the bone, and a careful examination with the screen should be made before the plate is exposed in order to avoid the chance of this error being made.

Now it is, of course, well known that cases of fracture of the patella from direct violence often do not result in much, if any, separation of the fragments. These fractures may be of any nature and the line of fracture may be in any direction. They are often comminuted, sometimes vertical, and in many cases transverse. It is not a matter for surprise, therefore, to find no diastasis in transverse fractures of the patella if due to direct violence, as in Wegner's last two cases, and in other cases which have been quoted.

As in many ways a fairly typical example, I will mention a case which occurred in the practice of the Middlesex Hospital.

Case I.—The patient, an able-bodied man, aged 28, was descending some steps into the street on May 26, 1909, when he slipped and fell on his left knee. He got up without help, and walked half a mile without stick or aid of any kind. On attempting to walk again, after resting for half an hour, he had much pain in the knee, and had to obtain the help of a friend to walk about a quarter of a mile to the hospital. He was seen by one of the resident officers, who transferred him to the out-patient department as a case of "traumatic synovitis" of the knee. The surgeon who saw him suspected the possibility of a fracture of the patella, and recommended him for admission. The patient could move the knee freely, there was no palpable separation of the fragments, and no crepitus was obtained. A radiogram (fig. 1) revealed a fracture of the patella without separation of the fragments, the line of fracture being mainly transverse, but the bone being slightly comminuted. There was considerable effusion into the joint, and the patient was kept in bed for a few days, after which he had a light removable plaster-of-Paris splint applied, and went to the convalescent home. There massage and passive movements were carried out daily, and convalescence was uninterrupted. Bony union occurred, as the accompanying skiagram, taken on December 20, 1909 (fig. 2), shows, and the man recovered with a strong knee and perfect movement.

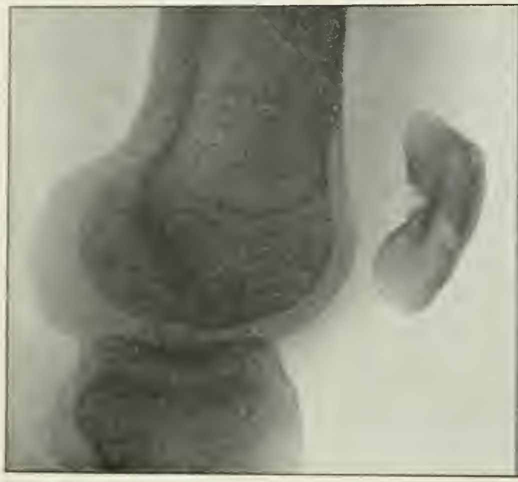


Fig. 1.

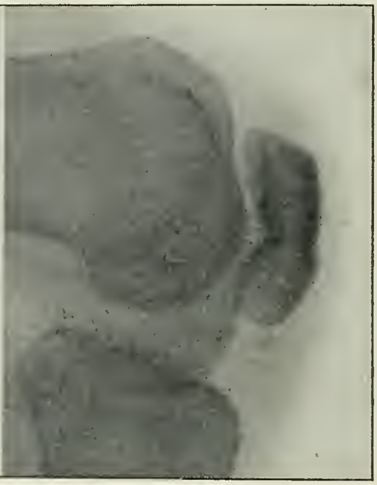


Fig. 2.

This case, as I have said, is fairly typical and by no means uncommon, though, like many similar cases, it presented some difficulty in diagnosis. But the next three cases I will describe were much more remarkable, and of the greatest pathological, as well as clinical, interest, for the fractures were caused in each case, as in Wegner's first case, by indirect violence—that is, by muscular contraction breaking the patella across the lower end of the femur—the well-named "tear fracture" of the patella.

Case II.—The patient, a man, aged 43, a driver of a horse tramcar, was descending the stairs to open the door to some visitors on the evening of January 15, 1909, when he caught the heel of his boot on the edge of a stair. He stumbled, but with an effort recovered his balance. He noticed a slight pain in the right knee, but did not pay any attention to it, finished his descent of the stairs and returned to his room, mounting two storeys. Next morning he noticed that his knee was rather swollen, but went to work. Owing to some pain and weakness in the joint he came, later in the day, to the hospital. He walked in with a slight limp, and, on examination, it was found that there was a little effusion into the joint, but otherwise no sign of any injury. The history being very suggestive of a fracture of the patella, particular attention was paid to the signs of this injury, and he was seen by several members of the hospital staff. There was no crepitus, no local tenderness, no cleft to be felt, no interference with movement (apart from what would be accounted for by the effusion alone), and no loss of the power of extending the leg. A radiogram, however, revealed the presence of a transverse fracture of the patella, without separation of the fragments (fig. 3). The patient was kept at rest in bed for a few days when a light removable plaster-of-Paris splint was applied, and he was sent to the convalescent home, where he had massage and exercises daily, getting about during the rest of the day with his splint on. Convalescence was uninterrupted, and bony union the result (fig. 4, from a skiagram taken on February 14, 1910).

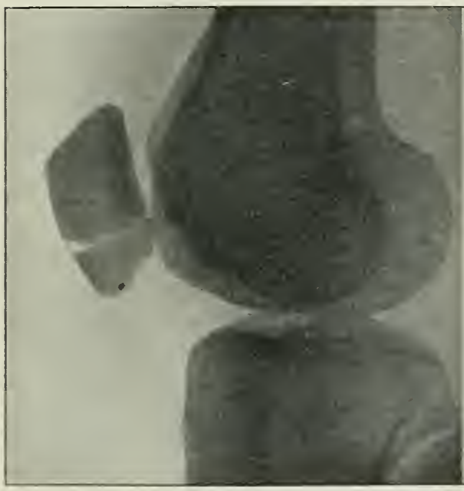


FIG. 3.

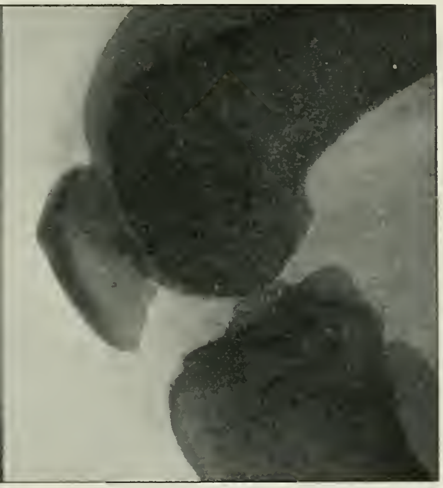


FIG. 4.

This last case was demonstrated at a meeting of the Middlesex Hospital Medical Society on March 25, 1909, and the importance of being on the look-out for such an obscure injury was emphasized. It was somewhat alarming, a few weeks later, when a house surgeon informed me that he had filled the last surgical bed with a traumatic synovitis of the knee, because he suspected that it might turn out to be a case of a similar fracture to the one I have described. However, my fears that the wards would be filled with cases of suspected fracture were not justified, for the case turned out to be a transverse fracture of the patella, without separation of the fragments. The history of the case is as follows:—

Case III.—The patient, a barmaid, aged 29, was turning round quickly in a stooping position when she felt a pain in the left knee like "something stretching." The pain increased, so she went to get some camphorated oil, her panacea for all ills, to obtain which she ascended three flights of stairs. She then returned to her work at the bar, but as the knee got more painful she sent for a medical man, who referred her to the hospital. She was admitted to The Queen's Ward on April 14, 1909, the day of the accident, under Sir Alfred Pearce Gould, who, knowing I was interested in these cases, kindly transferred the patient to my care, and I am indebted to him for permission to publish the case. There was good power of extension of the leg; no line of fracture could be felt; there was no local tenderness, no crepitus, and but little effusion into the joint. A radiogram (fig. 5) showed a transverse fracture of the patella, without separation of the fragments. After a few days' rest in bed a light removable plaster-of-Paris splint was applied, and she was sent to the convalescent home, where massage and passive and active movements were carried out daily. Convalescence was uninterrupted, and the result was excellent, bony union occurring, as the accompanying plate shows (fig. 6, from a skiagram taken on September 5, 1909).

Case IV.—The remaining case was that of a carman, aged 31. He was descending some stairs on February 7, 1910, to deliver a parcel, when he slipped and fell downstairs, failing to recover himself. He did not fall on his knee, but his leg was bent under him. He got up, went upstairs again, mounted his van, and drove back to the depôt. He got off his van without help. The knee, later in the day, became painful, so he drove round to the hospital and walked in. The case was diagnosed as one of synovitis of the knee, a back splint was applied, and the patient instructed to attend as an out-patient. The next day he remained indoors, but the following day (February 9) he called at the hospital again, when the casualty surgical officer, Mr. C. H. S. Webb, ordered a skiagram to be taken (fig. 7), and it was found that there was a transverse fracture of the patella, without separation of the fragments. In this case, as in the others, there was no local tenderness, no crepitus, and no separation of the fragments to be felt. The power of extending the leg was quite strong. The same treatment was adopted as in the other cases, and the result was excellent, bony union being obtained (fig. 8, from a skiagram taken on July 18, 1910).

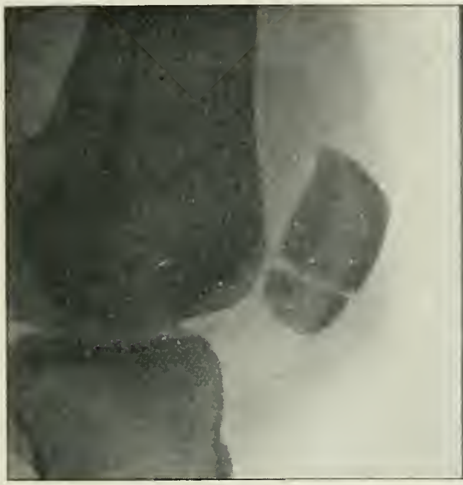


Fig. 5.

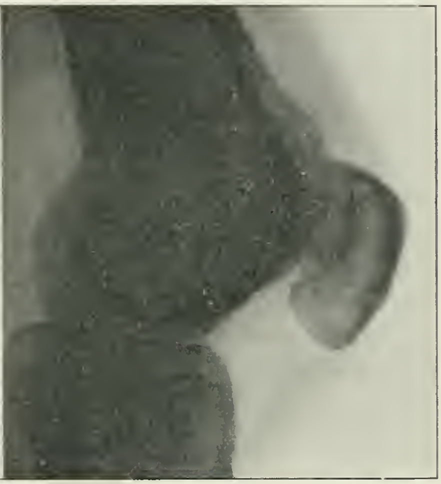


Fig. 6.

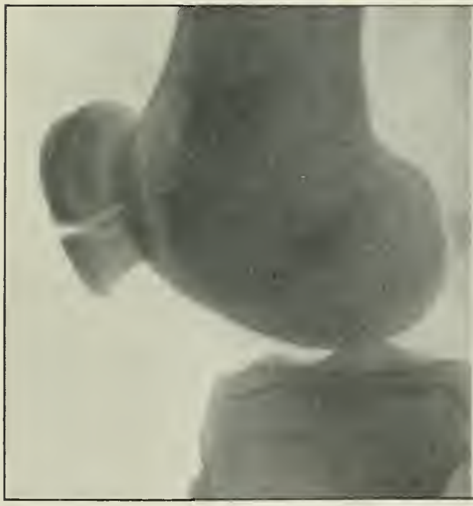


Fig. 7.

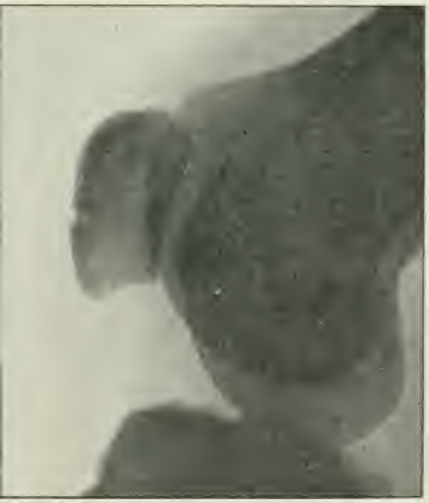


Fig. 8.

The problem of the satisfactory treatment of fractures of the lower limb in hospital is no easy one; for if they are to be treated by massage and exercises, these cannot be carried out for a sufficient length of time in the wards owing to the great demands on the beds, so that some splint has to be applied while they are being treated as out-patients, with the result that stiffness in the neighbouring joints and adhesions at the site of fracture are but too common. At the Middlesex Hospital a serious attempt has been made to deal with this difficult problem, and we are fortunate in having at our Convalescent Home a most able and enthusiastic matron who takes the greatest interest in these cases, and supervises treatment by massage and exercises, which is carried out by nurses skilled in massage after the patients are fit to be discharged from the wards of the hospital.

All the cases of fracture of the patella which I have described were, after a few days' stay in bed, transferred from the Hospital to the Convalescent Home, where massage and movements were carried out daily. In all the cases, recovery was absolutely complete—in fact, a perfectly sound limb was the result, and the patients were fit to return to their occupations within three months of the date of the injury.

In every instance the union was bony. Unless great care be taken to make the exposure to the X-rays in the exact line of fracture, it is quite possible for a plate to appear to show bony union when none is present. In all my cases the patella was most carefully examined through the fluorescent screen before the exposure of the plate, to make absolutely certain that no cleft between the fragments was still visible.

The cases are extremely interesting in many ways. First, they are of great pathological interest, for they bear out very clearly the statements of Macewen with regard to the mode of production of a transverse fracture of the patella by indirect violence. As Macewen [31] stated, "the fibrous and aponeurotic tissues lying in front of the patella are more elastic than the bone. By muscular action the bone snaps and retracts, and the fibrous and aponeurotic tissues over it still holding, they bridge the interspace between the two fragments. If the contraction continues, the aponeurotic and fibrous tissues become overstretched and lose their resiliency. Finally, if the force be great enough, the bridge gives way . . ."

The clinical interest of these cases is enormous, and their importance cannot be exaggerated. The fact that the patients walked without much difficulty is remarkable in itself, though it is often possible if there be no separation of the fragments. Still, it must be remembered that patients can walk even when there is an obvious fracture of the patella, with marked separation of the fragments. Jessop [23], for example, reported the case of a bargee who broke his patella clean across, tied a handkerchief round the knee, but never lay up or received any treatment at all. Dubrueil [17] also relates how a man fractured his patella and was able to walk forward to his home. Later, he re-fractured it, and was again able to walk. Martin [37] has also reported a similar case; while Carl Beck [4] mentions that in his experience patients, in spite of appreciable diastasis, were able to walk considerable distances without apparent discomfort. The fact that a patient walks in to see the surgeon should not, therefore, lead him to exclude the likelihood of a fracture of the patella having occurred.

From a study of these cases without separation it will be realized that the diagnosis may be extremely difficult, and that an absolutely certain diagnosis may be impossible without the aid of the X-rays. This fact has been rather neglected in most text-books, though recently attention has been called to it in some American and German works. Besides those writers which have already been referred to, these cases of fracture without separation of fragments have been mentioned specially by Reichel [44], König [26], Magruder [33], and Heineck [21] the last named stating that they often escape recognition. But even in the latest work by Cotton [13], it is stated that "tear fractures" always lead to separation of the fragments. This is shown to be a misstatement by Wegner's first case, and by my three cases.

The difficulties of diagnosis being proved, the question arises whether neglect to recognize cases of this character is of great importance. Undoubtedly it is. If these cases are treated as simple contusions or sprains, and the patients are allowed to go about and return to their ordinary work when the swelling has subsided, separation of the fragments is likely to occur, and the fracture may be very easily converted from one which will get quite well with rest and care into one requiring operative treatment to ensure a satisfactory result. The case reported by Broca, and Malgaigne's cases which he refers to, all of which I have already described, are good examples of the dangers to which these patients are exposed. It is essential, then, in injuries to the knee, or any other part, to have a skiagram taken of all cases in which there is the slightest possibility of fracture. In fact the X-rays, by the revelations they have made, have proved the importance of having a plate taken in all injuries of the joints, and there can be little doubt that many cases diagnosed as sprains are really associated with fracture. The medico-legal importance of these cases can hardly be over-estimated, for if the patient obtain a skiagram elsewhere, which shows a fracture, when the surgeon has assured him that there is none, the consequences are likely to be extremely unpleasant, if not disastrous, for the surgeon. While not in any way wishing to discourage the most careful manual examination of cases of injury, the fact must be recognized that manual examination, however carefully carried out, will fail to demonstrate some bone lesions which only the X-rays will reveal. By the systematic use of the X-rays obscure injuries will be revealed, and much needless suffering and inconvenience prevented.

The next aspect of these cases to which I will direct attention is the process of repair and the importance of obtaining bony union. The average length of treatment in my cases was about three months, when the patients returned to full work. Skiagrams were frequently taken to make sure that no separation of the fragments was taking place. The new bone does not throw a clear shadow for some months after it is laid down, and it is not possible to be sure that bony union has occurred until five or six months after the injury. I attach great importance to obtaining bony union in these cases, for even a short fibrous union is liable to stretch, an occurrence attended by some disability, not to say many dangers. Not the least of these dangers is the liability to re-fracture of the patella or rupture of the ligamentum patellæ. It is a pity to my mind that cases of fracture of the patella, including even those which have been submitted to operation, are not more carefully followed up and skiagraphed to see whether bony union has resulted; and Stimson [48] has stated that, even in operation cases, the union, which was so close that no independent mobility could be recognized, has sometimes been found to be fibrous, while in some bony union has taken place only in the posterior half or three-quarters.

It has been maintained that fibrous union was as good as, or even better than, bony. Hamilton [20], for instance, stated that a fibrous band, with only $\frac{1}{2}$ in. or $\frac{3}{4}$ in. separation, was as useful as bony; whilst many writers contended that because the bone, and not the fibrous union, often breaks in re-fracture, bony union has no advantage over a close fibrous union. Such was maintained by Morris [40], Desprès [14, 15], Wagner [55], and Murray [41], amongst others; but these surgeons were probably not aware of the fact that re-fracture was more common after non-operative than after operative treatment, and that it was the fibrous union and consequent lengthening of the patella which predisposed to re-fracture.

That fibrous union of a fractured patella does seriously impair the stability of the knee-joint must now be undoubted. Re-fracture is much more frequent in the patella than in any other bone—Lauper [27], Thiem [50], Corner [11, 12]; and more common after massage and other forms of non-operative treatment (when the union is generally fibrous) than after open operative treatment (when the union is probably bony)—Alessandri [1], Gibbon [18], Meyers [39], Powers [42], Bégouin and Audérodias [5]. Not only does a fibrously united patella increase the liability to re-fracture, but the whole extensor arc is weakened, the joint being uncertain and unreliable, which leads to frequent falls if the patients return to their usual occupations and habits of life. There is evidence of this in the frequency with which other injuries occur—for example, rupture of the ligamentum patellæ. In 140 cases of rupture of the ligamentum patellæ, Walker [56], found that previous fracture of the patella had been an ætiological factor seventeen times. Although bony union presents but slight advantages over a close fibrous union so far as immediate results show, when the future history of the limb is taken into account the advantages are much greater.

The period of treatment in my own cases might possibly have been cut down slightly, but, for the reasons I have stated, I was particularly anxious to make sure of a sound bony union. There has been great keenness on the part of surgeons to cut down the length of treatment of fractures of the patella after suture of the fragments, but I cannot help thinking that this has been rather overdone. Cases of re-fracture after wiring are too common, and since they generally occur during the first few months after operation it looks as if too great a risk were being taken in allowing these patients so much freedom of the limb; some support, such as a bandage or even some check apparatus, such as that described by Howard Marsh [36], would seem advisable to prevent extreme flexion for the first week or so after getting about. No doubt, however, many wonderful cases have been reported; for instance, one recorded by Lucas-Championnière [28], where a clown was able to return to his occupation, involving the turning of somersaults, within two months after operation.

In bringing the subject of transverse fracture of the patella, without separation of the fragments, under notice, I thought it would give food for thought in many ways: pathologically the cases are interesting; the clinical picture is most striking and teaches us to hesitate very much before giving a definite diagnosis of a bone lesion without the help of the X-rays; and, historically, in accounting for probably most of the cases of bony union which were recovered from the post-mortem and dissecting rooms before the days when wiring was the usual treatment.

By far the most important point for the surgeon is the difficulty of diagnosis, and he must now realize that he is absolutely dependent on the X-rays in such cases. With regard to the frequency with which these cases occur it is difficult to speak with certainty, and until it is the rule to examine all injuries of joints with the X-rays we cannot say how many of these cases are being missed at the present time. The cases I have described occurred in the practice of the Middlesex Hospital in a period of thirteen months, during which time we treated in all twenty cases of fracture of the patella. Failure in diagnosis may not only involve the surgeon in a suit for damages, but if the character of the injury be not realized by somebody else an unusual strain may lead to great hardship for the patient, owing to separation of the fragments and the necessity of an operation which would otherwise have been unnecessary.

BIBLIOGRAPHY.

[1] ALESSANDRI. *Centralbl. f. Chir.*, Leipz., 1902, xxix, p. 472.
[2] BARKER. *Trans. Clin. Soc. Lond.*, 1900, xxxiii, p. 225.
[3] *Idem. Brit. Med. Journ.*, 1900, i, p. 145.
[4] BECK. "Fractures," 1900.
[5] BÉGOUIN et AUDÉRODIAS. *Gaz. méd. de Par.*, 1897, sér. 10, i, p. 505.
[6] BENNETT. *Dublin Journ. of Med. Sci.*, 1881, lxxii, p. 450.
[7] BLACKHAM. *Journ. Roy. Army Med. Corps*, 1907, ix, p. 610.
[8] BROCA. *Journ. de méd. et de chir. prat.*, Par., 1875, p. 58.
[9] *Idem. Centralbl. f. Chir.*, 1875, ii, p. 223.
[10] CAUTHIER. *Lyon méd.*, 1905, cv, p. 627.
[11] CORNER. *Ann. of Surg.*, Lond., 1900, xxxii, p. 749.
[12] *Idem. Practitioner*, 1908, lxxxi, p. 498.
[13] COTTON. "Dislocations and Joint Fractures," 1910.
[14] DESPRÈS. *Progrès méd.*, Par., 1878, vi, p. 735.
[15] *Idem. Lond. Med. Rec.*, 1878, p. 476.
[16] DOBERAUER. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1905, xxxiv, p. 195.
[17] DUBRUEIL. *Semaine méd.*, 1892, xii, p. 297.
[18] GIBBON. *Ann. of Surg.*, Lond., 1904, xxxix, p. 1021.
[19] GULLIVER. *Edin. Med. and Surg. Journ.*, 1837, xlvii, p. 160.
[20] HAMILTON. "Fracture of the Patella," 1880.
[21] HEINECK. *Surg., Gyn. and Obstetr.*, Chicago, 1909, ix, p. 177.
[22] HUMBLET. *Arch. méd. belges*, Brux., 1883, xxxiv, p. 236.
[23] JESSOP. *Med. Times and Gaz.*, 1871, ii, p. 280.
[24] JOHNSON. *Arch. of Middlesex Hosp.* (Clin. Ser., No. 5), 1910, xvi, p. 47.
[25] *Idem. Med. Rev.*, 1910, xiii, p. 604.
[26] KÖNIG. "Lehrbuch der speciellen Chirurgie für Aerzte und Studirende," Berl., 1905. iii, p. 626.
[27] LAUPER. *Corresp.-Bl. f. schweizer Aerzte*, Basel, 1904, xxxiv, p. 321.
[28] LUCAS-CHAMPIONNIÈRE. *Arch. internat. de chir.*, Gand., 1903, i, fas. 1.
[29] MACEWEN. *Lancet*, 1883, ii, p. 847.
[30] *Idem. Ann. of Surg.*, St. Louis, 1887, v, p. 177.
[31] *Idem. Lancet*, 1883, ii, p. 847.
[32] MAGRUDER. *Journ. Amer. Med. Assoc.*, Chicago, 1910, liv, p. 1843.
[33] *Idem.* Loc. cit.

[34] MALGAIGNE. *Journ. de chir.*, Par., 1843, i, p. 244.
[35] *Idem.* "Traité des fractures et des luxations," 1855, plate xiv, figs. 2 and 3.
[36] MARSH, HOWARD. *Brit. Med. Journ.*, 1898, i, p. 613.
[37] MARTIN. *Gaz. des hôp.*, 1895, lxviii, p. 1345.
[38] MAYER. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1905, xxxiv, p. 189.
[39] MEYERS. *Ann. of Surg.*, Lond., 1901, xxxiii, p. 194.
[40] MORRIS. *Lancet*, 1883, ii, p. 865.
[41] MURRAY. *Brit. Med. Journ.*, 1898, i, p. 817.
[42] POWERS. *Ann. of Surg.*, Lond., 1898, xxviii, p. 67.
[43] REICHEL. Von Bergmann's "System of Practical Surgery," 1905, iii, p. 573.
[44] *Idem.* Loc. cit.
[45] RÖDIGER. "Extracapsuläre Brüche der Patella," Trier, 1887.
[46] SCUDDER. "The Treatment of Fractures," 1907.
[47] SMITH. *Amer. Journ. Med. Sci.*, Philad., 1873, lxv, p. 429.
[48] STIMSON. "Treatise on Fractures and Dislocations," 1905.
[49] THIEM. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1905, xxxiv, p. 392.
[50] *Idem.* *Ibid.*, 1905, xxxiv, p. 374.
[51] TRÉSORET. *Gaz. des hôp.*, Par., 1881, liv, p. 732.
[52] *Idem.* *Med. Times and Gaz.*, 1881, ii, p. 267.
[53] VALLAS. *Rév. de chir.*, Par., 1899, xx, p. 419.
[54] *Idem.* *Lyon méd.*, 1905, civ, p. 1001.
[55] WAGNER. *Deutsch. med. Zeitung*, 1888, No. 11.
[56] WALKER. *Amer. Journ. Med. Sci.*, Philad., 1896, n.s., cxi, p. 638.
[57] WEGNER. *Deutsch. Zeitschr. f. Chir.*, Leipz., 1900, lvii, p. 157.

DISCUSSION.

Mr. JONATHAN HUTCHINSON showed skiagrams of two cases bearing upon the subject of the paper, in which he had long been interested. The slides both showed a form of fracture of the patella which could not be diagnosed without the aid of the X-rays. In one case only the tip had broken off; it appeared as if it had been cut off with a chisel. The next slide showed a form which Mr. Johnson had not referred to, where there was a double transverse crack through the patella, without the least separation between the three fragments. The correct diagnosis might perhaps be occasionally made by the detection of the slight groove. He thought Mr. Johnson would agree that that was the only sign, apart from the tenderness and slight bruising, which pointed towards the diagnosis of fracture. He agreed with all Mr. Johnson said in his paper, except his last conclusion as to wiring. If two fairly thick silver wires were put in, the patella was stronger than before the accident, and if such a patella broke again it must have been due to some very violent cause, such as a fall in getting out of a tramcar, as he had known to happen in one case he had wired some two or three months before. He had operated again, removed the ruptured wires, and inserted fresh ones with complete success. If only *one* silver wire were put in it might give way on slight provocation, but the second wire was a safeguard. He had seen a case in which a single wire snapped a few months after its insertion, whilst the patient was dancing. The wire cut through and the fragments separated. As a rule, if a double wire had been inserted, the patient might safely be allowed to bend the knee within three weeks of the operation. That was the only point upon which he differed from the views expressed by Mr. Johnson in his admirable paper.

Mr. BRUCE CLARKE congratulated Mr. Johnson on bringing forward cases to show that there were many fractures which had not been thought of in connexion with the patella. Some years ago he had a man under his care who had fallen on his knee, and he (Mr. Bruce Clarke) did not know he had fractured it until he opened the joint. He seemed to have a loose cartilage. An incision was therefore made, and a piece of cartilage found loose which had been knocked off the under side of the patella, which was cracked across, but there was no separation of the fragments, and he saw the man some months later with sound union of the bone. He had no doubt it was the kind of fracture which Mr. Johnson had described. That might have been discoverable by X-rays, but in no other way. The case confirmed Mr. Johnson's contentions.

The PRESIDENT (Mr. Rickman J. Godlee) asked what happened when those cases were examined—could the fragments be moved from side to side, or did attempted movement elicit pain? He had a skiagram of a person who had a gumma in her patella and fractured it. It was very difficult to know whether the fractures were due to direct violence or not, as the histories given were often not dependable.

Mr. JOHNSON, in reply, expressed his great gratification at the reception of his paper. Mr. Hutchinson's remarks had interested him, but he feared without convincing him. If tension were put upon sutures in any part of the body the result was absorption of the tissues, and consequently the suture became loose. He did not see any reason why the patella should be regarded as an exception. He wished it to be clearly understood that he had not argued in any way against the employment of a suture in cases of fracture of the patella when there was separation of the fragments, but felt that there was good reason for advising a little more caution in the after-treatment of these cases. Of the two skiagrams shown by Mr. Hutchinson one was a fracture of the lower tip of the patella—a variety of fracture which was often extra-articular, and had been described by Blackham, Rödiger and others—while the other was not a very rare injury, since, from its character, it was probably caused by direct violence, though Mr. Hutchinson could not state whether this was so or not. The case mentioned by Mr. Bruce Clarke was extremely interesting. Here, again, since there was a small piece broken off the patella as well as a transverse fracture, the injury was probably caused by direct violence. The special interest of the case was its resemblance to the second case of Wegner's which was described in the paper. With regard to the President's remarks about the difficulty of deciding, from the patient's account, whether a fracture was caused by direct or indirect violence, Mr. Johnson agreed that it was often impossible. It was only in those cases in which the patients did not fall at all and did not receive any direct blow on the knee that it could be stated with absolute certainty that the injury was not due to direct violence. Though in the cases related fracture of the patella was suspected, there was no definite sign of this injury, though the knees were most carefully examined—sometimes by more than one member of the hospital staff.

Surgical Section.

January 10, 1911.

Mr. Rickman J. Godlee, President of the Section, in the Chair.

The Delimitation of the Rectum and its Subdivisions.

An Anatomical Introduction to a Discussion on the Operative Treatment of Cancer of the Rectum.

By F. Wood Jones, B.S.

In the whole length of 30 ft. or so of the intestinal canal of man certain portions are marked off from each other by various definite and easily recognized characters; so that in common usage the limits of these subdivisions are easily known, and the comparison of similar parts of different intestinal tracts presents no difficulty. Such subdivisions as the stomach, or the œsophagus, are so clearly marked off from the rest of the alimentary canal that no confusion arises in the study of these parts as described by different authors, or at different periods of the evolution of anatomical literature. The same satisfactory precision of limitations does not, however, hold good in the case of the rectum and its subdivisions.

The description of the rectum has from time to time undergone great changes, so that the portion of the gut indicated by the term "rectum" is not identical in the descriptions of anatomists of different historical periods. Even anatomists of the same period often differ widely in their conceptions of the portions of the hind gut properly designated by the term "rectum." Some slight account of the evolution of the present orthodox description of the rectum is a necessary prelude to any attempt at the introduction of any modification of this description.

One method of delimiting the rectum, which held good in English anatomical literature for a period of one hundred and fifty years or so, may be given in the words of Thomas Gibson,[1] who wrote in 1697: "This hath its beginning at the first vertebra of the os sacrum, where the colon endeth, and passeth straight downwards to the extremity of the coccyx." The first historical changes which were made in this description consisted of slight individual alterations in the limits set upon the rectum above and below. Cheselden (1756) and most English anatomists adopted the description quoted above, but Tuson (1828) extended the limits slightly by starting the rectum at the last lumbar vertebra, and terminating it "a little beyond the extremity of the coccyx." Cloquet, whose descriptions of the viscera show so great an excellence, marked off the rectum (Knox, trans., 1831) as extending from the left side of the lumbo-sacral articulation to the anus, and by the anus he meant the cutaneous ring-like orifice which lies "at about an inch before the coccyx."

From this time onwards for another fifty years this description was only varied by a gradual substitution of the left sacro-iliac joint for the joint between the last lumbar and first sacral vertebræ as the upper boundary of the rectum. The part of the intestinal canal so delimited is best described in the words of Treves when summing up the orthodox description of the rectum current in English literature until 1885: "The rectum is divided into three parts. The first part commences at the sacro-iliac synchondrosis and passes obliquely from left to right, forming a gentle curve to the right, and ultimately gaining the middle line opposite the third piece of the sacrum; it is entirely invested by a fold of peritoneum called the meso-rectum. The second part extends along the concavity of the sacrum as far as the coccyx, at which point the third part begins, and inclines at once backwards to terminate at the anus." So much for the generally accepted description of the rectum in English literature in 1885.

The next step owed its origin to the work of Treves and of Jonnesco on the pelvic colon, made familiar to English surgeons by the investigations of Treves, published in his Hunterian Lectures.[2] The changes necessitated by these new views consisted in regarding the old first part of the rectum (which possessed a mesentery) as the terminal part of the pelvic colon, and limiting the term "rectum" to only the original middle and lower portions—i.e., that part from the third sacral vertebra to the anus. This description became stereotyped in English anatomical literature by the appearance of a paper published by Professor J. Symington in 1888.[1] Symington followed Treves and Jonnesco in eliminating the original first part, and he went a step further in taking away the old third part of the rectum proper and making it a separate part of the alimentary canal named the "anal canal"—an arbitrary subdivision which has been followed in descriptive anatomical work ever since. Symington's findings with regard to the anal canal were the necessary outcome of the ordinary results of changed anatomical nomenclature; for he was, in fact, making no new subdivision of the intestinal canal, but giving a new name to a portion of the rectum long recognized as distinct. Of those authors who had described the rectum previous to Symington, two different groups had used the word "anus" as denoting different anatomical structures. The anatomists, from about 1830 onwards, meant by the anus that puckered rim of skin by which the bowel opens to the surface of the body—"the verge of the anus" of the third Monro. They used the word "anus" in its correct significance of a ring—the ring which surrounds the lower end of the rectum. Gibson, however, and the English anatomists as late as Tuson in 1828, reckoned all that part of the tube from the end of the coccyx to the skin as anus; for the rectum, as they described it, ended at the tip, or just beyond the tip, of the coccyx.

---

[1] "Anatomy of Humane Bodies," Book I, 5th ed., 1697, p. 58.

[2] "Anatomy of the Intestinal Canal and Peritoneum," Hunterian Lectures, Royal College of Surgeons, 1885.

Symington described the anal canal as follows (op. cit., p. 109): "It is about 1 in. in length, and its long axis is directed downwards and backwards at about a right angle with that of the second part of the rectum. It includes what is frequently described as the third part of the rectum and the anus." The "anal canal" was therefore the same thing as the third part of the rectum of authors immediately preceding Symington, and as the anus of the authors of a still older generation. Symington, however, introduced a new conception into this anal canal, for he looked on it as the duct of the rectum, which he regarded as "a receptacle for fæces"; and he pointed out that it was closed by the approximation of its lateral walls, whereas the rectum proper was closed by the opposition of its anterior and posterior walls. This side-to-side flattening of the anal canal is due to the lateral pressure of the levatores ani muscles, and the exact extent of the flattening produced in different subjects has caused the anal canal so defined to be a rather uncertain subdivision of the gut. Symington asserted its length to be "about 1 in."; in various anatomical text-books its dimensions vary from ½ in. to 1½ in.; and Mr. Harrison Cripps gives 2½ in. as the limit of levator ani pressure.

---

[1] *Journ. Anat. and Physiol.*, 1888, xxiii, p. 109.

It will therefore be seen, from this very brief review of some of the changes in description, that the limits of the rectum have been far from settled in the minds of anatomists; that the rectum of modern authors consists only of the second or middle portion of the older subdivisions; and that even its limits are by no means definitely established at the present day. It is possible that this uncertainty is due—and always has been due—to attempting to limit what merely is a subdivision of the intestinal tube by reference to fixed landmarks on the bony parietes. The present limitation of the rectum is not, in fact, a natural one, for it consists of including as one morphological entity a portion of a very plastic tube which happens to lie between two skeletal levels. That the pelvic viscera are not always set at the same level in relation to the bony boundaries of the pelvis (for instance, in elderly multiparous women and in young people) is well known, and it is therefore easy to see that an attempt to delimit the extent of a pelvic viscus by reference to these boundaries cannot be met with constant success.

Other subdivisions of the intestinal tract are established either by some distinction in their development, or by some more or less well marked peculiarities of the structure of their walls, or of their lining membrane. It is therefore natural to see if some such more satisfactory basis cannot be used in making a criterion for determining which part of the large intestine is to be properly regarded as the rectum, and for subdividing, if necessary, the portion of the gut so determined. I am inclined to believe that anatomists possess the material for making a more natural delimitation of the rectum, and Professor Paterson (1909), in his paper on the form of the rectum[1] has indicated some of the lines which should be followed.

In 1901, in the course of an investigation into the development of some of the common malformations of the hind end of the body, I had occasion to examine the early stages of the development of the urogenital sinus and rectum. I came to the conclusion that in the adult rectum two very distinct parts were combined, and that the division between these two parts was clearly indicated in the normal adult rectum (fig. 1). The results obtained from the study of development, of which a brief résumé was published,[1] were afterwards confirmed by Professor Arthur Keith, from the study of the comparative anatomy and the malformations of the rectum. The work of Professor Keith was delivered as demonstrations at the Royal College of Surgeons, and published in 1908.[2] With these papers for reference I need only very briefly state the actual results.

---

[1] *Journ. Anat. and Physiol.*, 1909, xliii, p. 127.

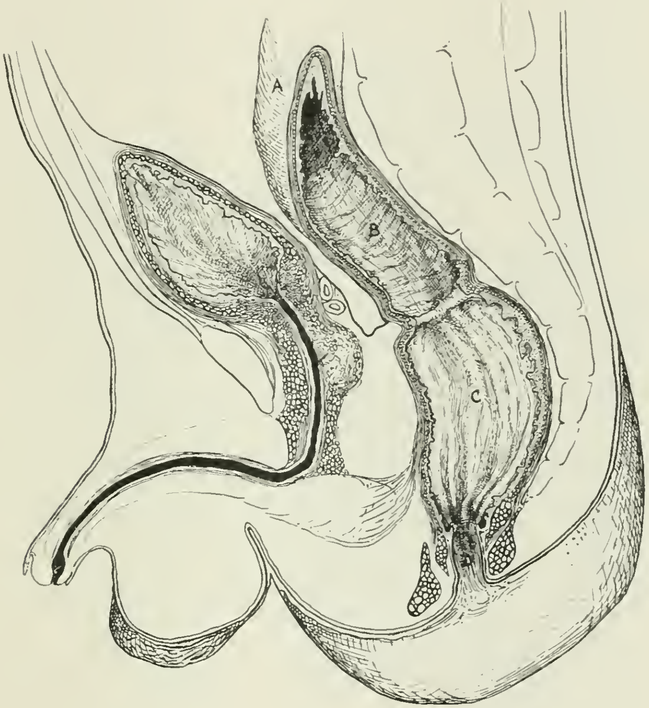


Fig. 1.

Section of the pelvis of a full-term male fœtus showing, **A**, pelvic colon; **B**, pelvic chamber of rectum; **C**, perineal chamber; **D**, anus.

At one stage of human development (embryo of twelve somites), and at one stage of evolution (amphibia), the rectum ends by opening into the cloaca in its upper part, in the neighbourhood of the openings of the genital ducts. From this original opening the rectum migrates downward, both in the development of the human embryo and in evolution, until it comes to open at the surface of the body at the anus (fig. 2). There are therefore two parts in the rectum—an old part which ends in the neighbourhood of the openings of the genital ducts, and a new part which runs from this point to the anus. It is natural to inquire if these two parts may be recognized in the conformation of the adult rectum, for if this is possible, then some rational subdivision of the rectum is indicated: also if their entire limits may be fixed, for then a definite boundary can be assigned to the rectum as a whole. Without repeating a mass of evidence which is available in the published papers already quoted, it may be said that the lower limit of the old portion (derived from the hind gut) is shown in cases of imperforate rectum, where the rectal cavity so commonly terminates in the neighbourhood of the prostatic urethra (fig. 3).

---

[1] *Brit. Med. Journ.*, 1904, ii, p. 1630.

[2] *Brit. Med. Journ.*, 1908, ii, pp. 1736, 1804, 1857.

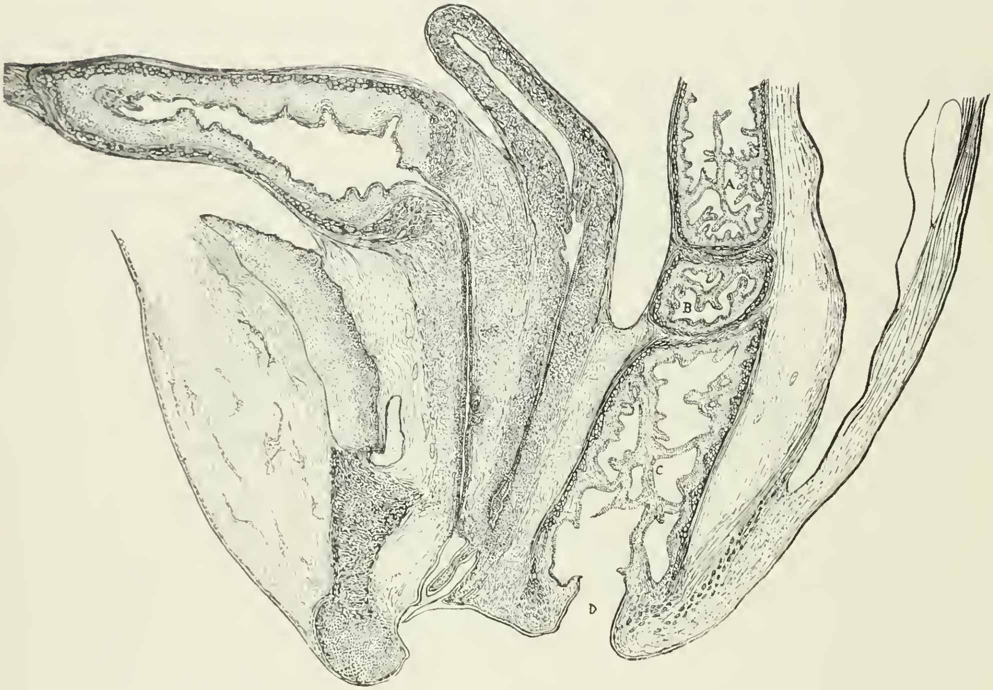


Fig. 2.

One of a series of sections of the pelvis of a female fœtus of about the fifth month, showing the rectal chambers and the two plicæ transversales recti. In this specimen the pelvic rectum, **B**, has a short course; **A**, pelvic colon; **C**, perineal portion of rectum; **D**, anus.

This is its termination in the abnormal rectum, and its termination in the normal rectum, I would contend, is at the peritoneal reflection from the anterior wall of the rectum, a point on the outside of the rectal wall which coincides with the main fold of Houston, or plica transversalis recti, within the cavity of the gut (fig. 1). With regard to the folds of Houston, and the significance of them, it is, I think, fair to say that the work of this Dublin anatomist has not had sufficient importance attached to it by subsequent authors. The reason for this is not difficult to see. Houston, in 1830, described four folds partly encircling the wall of the rectum: two of these folds—the first and the third—are undoubtedly definite anatomical entities; but two—the second and fourth—were so easily shown to be transient and highly variable

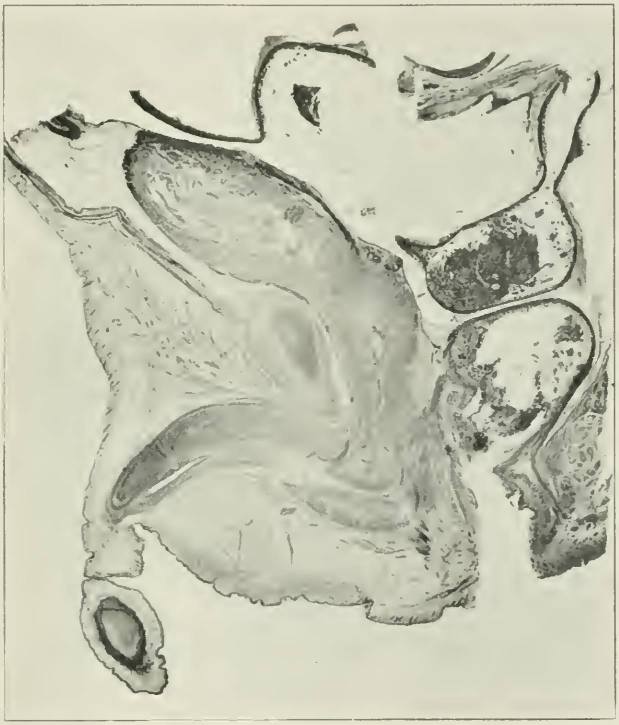


Fig. 3.

One of a series of sections of the rectum, &c., of a seven months' male fœtus, showing the normal subdivisions of the rectum.

puckers of the mucous membrane, that the importance of the two constant ones was lost sight of (figs. 1—5). Most authors have been able to show that the second and fourth of Houston's folds were quite unreliable landmarks, and it has been generally taken for granted that the first and third fall into the same class, and are worthy of no more attention than that bestowed upon occasional and unimportant inflexions of the intestinal wall. Far from considering these two folds as variable or unimportant, I would regard them as being the natural and essential landmarks for the delimitation of the rectum and its subdivisions. A few authors (such as O'Beirne, in 1833, Jonnesco, Paterson, &c.) have recognized the constancy of the first plica transversalis, which is situated at the junction of the pelvic colon and the rectum; and many have given evidence as to the great constancy of the third plica, which is situated immediately below the peritoneal reflection (fig. 5). In my experience both these folds are constant in the normal rectum, and are not mere inflexions of its wall, but are actual thickenings of the muscular coat which justify the name of "third sphincter," or "Nélaton's sphincter," which has been given to the lower one. Houston's second and fourth folds I admit as mere inconstant puckers of the rectal wall. In describing the rectum I would therefore set Houston's first fold as the upper limit, and make one chamber of the rectum—the pelvic rectum—extending from this fold to the peritoneal reflection, marked inside the gut by Houston's third fold (*B* in figs.). This (pelvic) part of the rectum has several characters which render it very distinct from the pelvic colon above, and the remaining portion of the rectum below. Its front and sides are clothed by peritoneum, it is a pelvic organ and expands into the peritoneal cavity. It is marked off from the pelvic colon by several very definite characteristics. The pelvic meso-colon ceases at its upper limit; the tæniæ coli blend together and constitute a uniform muscular coat for the pelvic rectum; the appendices epiploicæ disappear from the intestinal wall at its upper extremity, and the arteria hæmorrhoidalis superior divides into two branches which run upon the sides of the gut along the lines of peritoneal reflection (fig. 6). The walls of the tube are thick, and their muscular fibres are disposed uniformly around the circumference; inside the cavity of the gut the ridges of mucous membrane run, for the most part, round the wall of the gut in circular fashion, and at its upper limit, especially on its right-hand wall, is the prominent spur of the superior plica transversalis. This portion of the rectum is derived from the hind gut, and is the old rectum which opened at the lower limit of the peritoneal cavity into the cloaca in the neighbourhood of the genital ducts. It is a morphological entity, quite distinct in its development; and in cases of imperforate rectum it is the only portion of the rectum developed. Its anatomical delimitation is quite distinct, and although its upper limit is usually at the level of the third sacral vertebra that is by no means essential. Below this chamber of the rectum—which may be named the pelvic portion of the rectum—is a second very distinct chamber, the perineal portion of the rectum, which has a very different developmental history, and very different anatomical relations. The perineal part of the rectum is altogether below the peritoneum, outside the pelvic cavity, and its expansion can only take place into the cellular intervals around its walls. It is developed from a portion of the intestinal tube, which I have named the post-allantoic gut, and which is a new development serving to carry the hind gut from its old cloacal opening to its new opening at the surface of the perineum. This is the

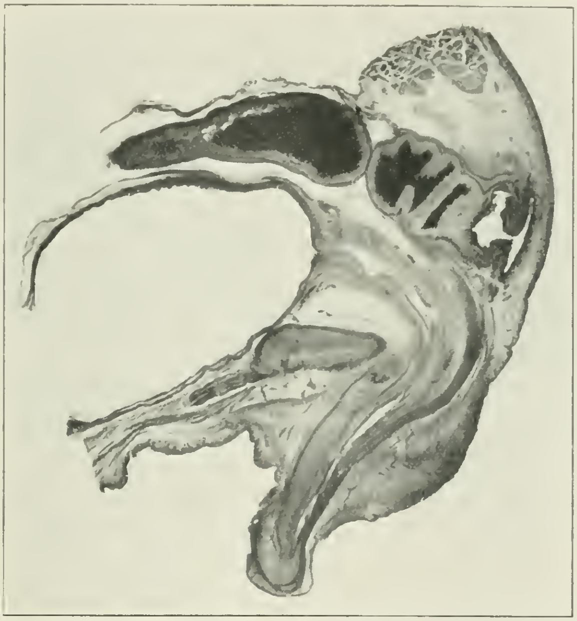


FIG. 4.

One of a series of sections of a five months' male fœtus, showing the normal subdivisions of the rectum. The section of the anus is not median.

portion of the gut which fails to develop in cases of imperforate rectum, and it bridges over the gap between the hind gut above and the proctodeum below. In the adult its boundaries are established by determining the limits of the hind gut and of the proctodeal depression. The hind gut extends, as we have seen, to the main fold of Houston, which encircles the anterior and right walls of the rectum at the lower limit of the peritoneal cavity marked by the level of the peritoneal reflection from the anterior surface of the rectum. The limits of the proctodeum are plainly marked in the adult rectum as the free edges of the rectal sinuses (valves of Morgagni), which are the remnants of the broken-down anal or proctodeal plate, in every way analogous to the hymen in the vagina (figs. 1, 2 and 4).

The perineal portion of the rectum is a very distinct embryological entity (post-allantoic gut), and it is also very distinctly delimited by its anatomical features in the adult. The arteria hæmorrhoidalis superior, which branched and ran down each side of the pelvic portion of the rectum, branches still more as it approaches the anterior peritoneal reflection from the rectum; and, at the level of Houston's main fold, its terminal twigs pierce the muscular coat of the gut and penetrate to a situation beneath the mucous membrane of the perineal chamber of the rectum (fig. 6). These terminal twigs run beneath the mucous membrane, accompanied by the veins and lymphatics, to the lower end of the perineal rectum. In their journey down to the annulus hæmorrhoidalis they lie beneath longitudinal folds of mucous membrane—the columnæ rectales. A distinction is therefore made between the pelvic and perineal chambers of the rectum in the disposition of the main folds of the mucous membrane, for in the pelvic rectum these folds encircle the gut, but in the perineal rectum they lie for the most part in the long axis of the gut (figs. 1 and 8). Much confusion exists in text-books of anatomy concerning these columnæ rectales. By many they are said to lie within the "anal canal," and if this is so, then the anal canal must be considered as extending to the main fold of Houston 3 in. from the anus. In the original figure of Morgagni (after whom they are named) they are very inadequately represented, and his drawing would appear to depict merely their swollen lower ends, although he gives some indication in the figure and in the text that they are in reality continued upwards, as the mucous membrane folds covering the vessels (fig. 7). It is at the lower end of these columns that the annulus hæmorrhoidalis is situated, for the veins contained within the columns are apt to form varicosities at this point. Between the lower ends of these columns are the rectal sinuses, whose free edges constitute the valves described by Morgagni, which mark the upper limits of the proctodeal invagination. The second chamber of the rectum is therefore a well-marked portion of the alimentary tract, distinct in its development (post-allantoic gut), and distinct in its anatomical limitations in the adult. It includes the ampulla recti, which is the bulging of its walls above the constricted proctodeal portion and the anal canal, which is a variable portion compressed laterally by the action of the levatores ani muscles.

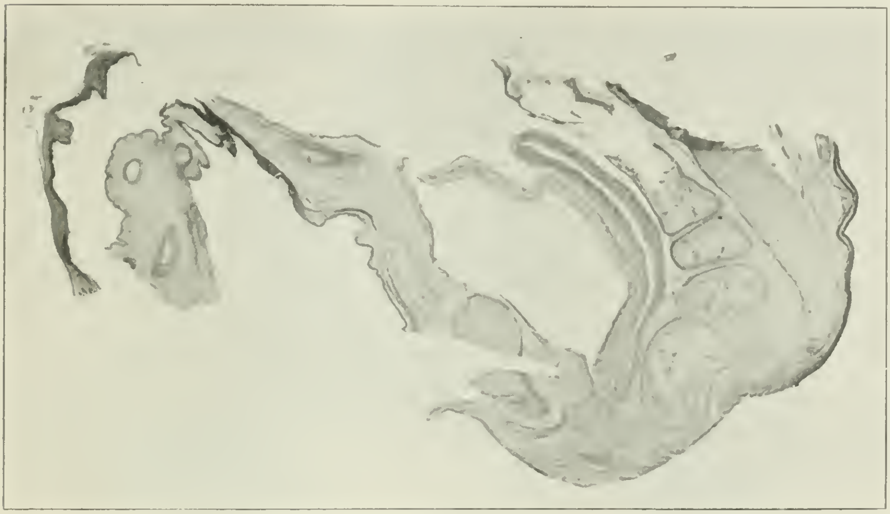


FIG. 5.

One of a series of sections of the pelvis of a six months' female fœtus, showing the normal subdivisions of the rectum. The section of the anus is not median.

The third subdivision is the anus proper. This corresponds to the original proctodeal ingrowth, and it stretches from the puckered pigmented anal skin to the under side of the anal valves.

It is surrounded by both the internal and external sphincters of the anus, and its lining epithelium is thrown into longitudinal puckers consequent on the constriction caused by these muscles (fig. 8). These longitudinal anal folds have been at times mistaken for the columnæ rectales, from which they are quite distinct, and this confusion has led to the erroneous description of the valves of Morgagni joining the *upper* extremities of the columns of Morgagni. This third subdivision is the

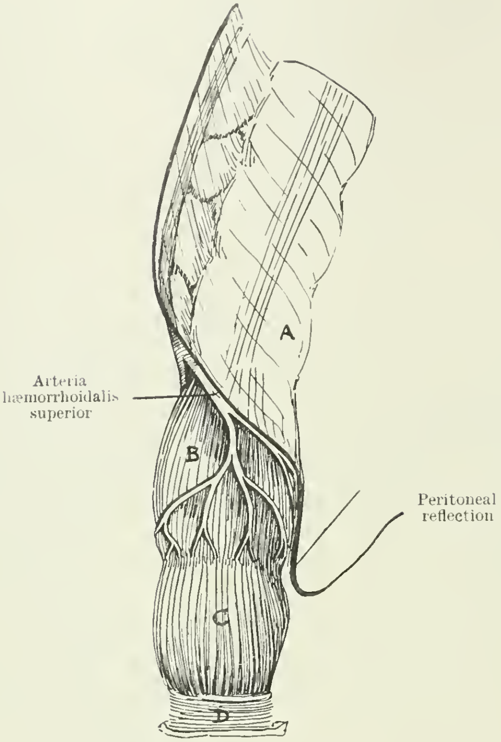


Fig. 6.

The two chambers of the rectum as marked out on the outer wall of the gut. **A**, pelvic colon ; **B**, pelvic portion of rectum ; **C**, perineal portion ; **D**, anal portion.

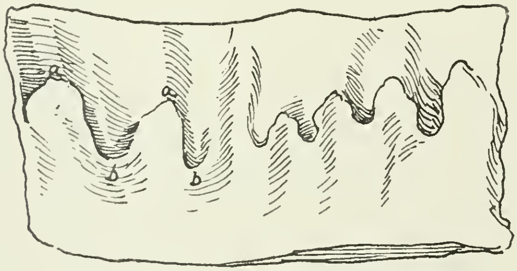


Fig. 7.

Tracing of Morgagni's original figure. From "Adversarià Anatomica," 1719. Description at III, vi, 10, 11.

one which fails to develop, or which fails to communicate with the perineal chamber of the rectum (post-allantoic gut), in cases of imperforate anus.

Following this description of the rectum, therefore, there are three well-marked embryological portions: (1) the hind gut, (2) the post-allantoic gut, and (3) the proctodeum; and these three portions, distinguished in development, are equally distinct in the anatomy of the adult rectum as (1) the pelvic chamber, (2) the perineal chamber, and (3) the anal portion (figs. 3, 4 and 5).

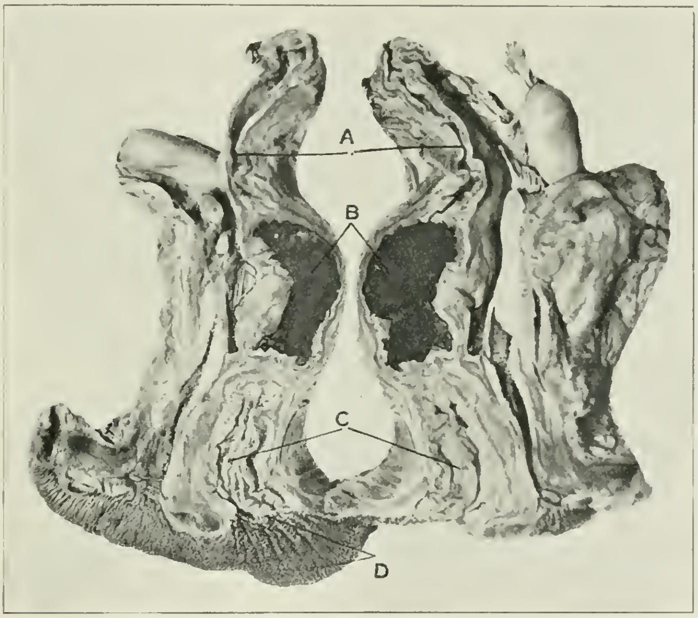


Fig. 8.

Section of the rectum of a young female in which the pelvic chamber, **B**, of the rectum is rendered conspicuous by containing fæces. The pelvic colon, **A**, and the perineal chamber of the rectum, **C**, are empty. The columnæ rectales are seen in C. The anal portion is marked **D**.

These subdivisions are submitted to surgeons in the belief that they are more practically useful than those current in anatomical literature, as well as being more embryologically and anatomically true; for the recognition of two main parts, the one intra-peritoneal and the other extra-peritoneal, marked off without reference to bony levels, seems more likely to lead to a rational classification of the diseases of the rectum. That anatomists work at a disadvantage when dealing with the rectum must be admitted, for the distension of the rectum with fæces, so common in dissecting-room subjects (fig. 8), and the artificial alteration of its form caused by stuffing with tow or cotton-wool, mask those natural subdivisions of the normal empty rectum which are well known to surgeons, and which I have attempted to describe. The rectum is not to be regarded, as many anatomists are disposed to regard it, as a normal storehouse for fæces; and work with X-rays upon the living has tended to show, and, I believe, will still further show, that the current anatomical description is not a good or a natural one.

Finally, I would submit that the very common anomalies in the development can only be understood and classified when the subdivision and limitations of the rectum are defined by some such method as I have attempted to follow.

The Operative Treatment of Cancer of the Rectum.

An Address Introductory to a Discussion on the Subject.

By F. Swinford Edwards, F.R.C.S.

I cannot but consider it a very great honour that I should have been asked to open from the practical side what is sure to be a very interesting and important debate on the Treatment of Cancer of the Rectum, a disease which, from all available statistics, appears to be on the increase. Had I to treat the subject from an historical and statistical point of view I know I should fall short of reaching even the lowest ideal to which I might wish to attain. But for an extensive review of the subject time does not permit, and I feel that the wishes of the Section are more likely to be met if I briefly state my own views on the present position of the operative measures necessary for the removal of a rectal cancer and my experiences of the various methods I have employed. I may at once say that the chief object of my remarks is to enter a protest against what I believe to be a growing tendency—viz., the performance of an unnecessarily severe operation attended with a heavy mortality for cases of cancer which can be cured by older methods, attended with but little risk to life, and which, moreover, entail no crippling of the patient.

All forms of treatment other than operative, whether by radium, X-rays, high-frequency currents, trypsin and other digestive ferments, have, I think it is generally admitted, proved unavailing, so we can confine our attention to the best means of effecting a cure by excision. The first successful extirpation of the rectum was, I believe, performed by Lisfranc in the year 1826. From this time to the year 1885 perineal excision for low-lying growths, as advocated and carried out by the Allinghams and Harrison Cripps in this country, was the only method in vogue—even to-day it has its proper place in operative surgery, which is for the extirpation of anal growths.

The work of Kraske, of Freiburg, in Breisgau, given to the world in 1885, marked a great advance, for by his trans-sacral method, cancers which before, owing to their high-lying position, were considered inoperable could be successfully removed and the function of the part, in many cases, restored by an end-to-end anastomosis.

The sphere of Kraske's operation was enlarged by the modifications of such men as Bardenheuer, Hochenegg, and Rosé, whilst the names of Billroth, Rehn, &c., are known in connexion with the formation and preservation of bone flaps. About this period the vaginal route found favour with some surgeons, and is comparable to the perineal operation as amplified and carried out by Quénu.

The next onward step was undertaken with the object of removing cancers situated in the upper rectum and lower sigmoid colon which did not permit of removal or could only be excised with difficulty and incompleteness by Kraske's route. It consisted in freeing the bowel through an abdominal incision and then excising it through the perineal or sacral route, with various modifications according to the exigencies of the case. This is called the abdomino-sacral, abdomino-perineal, or combined operation. It is an operation of considerable magnitude and is attended with a mortality of about 20 per cent. In the ideal operation no artificial anus is necessary, the gut being brought down through the sphincters and stitched to the anal margin. It should be restricted to cases where the disease has not passed beyond the walls of the bowels.

Nowadays the position of the growth—i.e., its distance from the anus—is no bar to extirpation. The only real bar to a successful operation is the fact that the growth is no longer confined to the bowel but has become adherent to neighbouring parts, whilst the lymphatics are infected. Another condition which quite bars any attempt at removal is metastasis in the liver, often not discovered until the abdomen has been opened, either for exploration of the growth or for the purpose of forming an artificial anus.

In considering the best method to employ for removing the disease, due regard must be paid to the immediate mortality of any given operation, the risk of recurrence, and the amount of crippling—if any—likely to follow the operation.

In a paper read by Mr. Ernest Miles before the British Medical Association last year, and published in the *Journal* of October 1,[1] the author advocates a very extensive proceeding which he calls the radical abdomino-perineal operation for *all* malignant growths of the rectum and pelvic colon, no matter how small they may be or where placed. This operation consists in the formation of an iliac anus with the entire removal of the rest of the sigmoid colon, the rectum with the soft parts adjacent thereto, and the anus. He states that his reason for advocating this extensive operation in *all* cases of rectal cancer is the great frequency of recurrence which followed his perineal operations, under which heading he includes para-sacral excision with end-to-end anastomosis. Out of fifty-eight operations there were fifty-four recurrences. This paper was accompanied by an elaborate table of cases, whilst the investigation into the spread of the disease—involving a large amount of work—reflects, if he will forgive my presumption in saying so, great credit on the author. I for one sympathize with Mr. Miles in the unfortunate results attending his perineal and sacral excisions, nor can I offer any explanation to account for them, unless it be that many of these cases were unfavourable for any operation, the disease having probably passed beyond the limits of the bowel at the time of the operation, and had better have been left alone. In reading through his tables, I find, in reference to "the spread of the disease found during the operation," by which is meant extension beyond the bowel, that in no less than thirty cases this in fact did obtain. In some cases the peritoneal coat was permeated, in others the growth was adherent either to the sacrum, prostate, or vagina, whilst the pre-sacral glands were usually involved. Concerning his table of twenty-six abdomino-perineal operations, there was evidence of the extension of the disease beyond the limits of the bowel in all but three cases, and yet of those who did not succumb to the operation (and of those who did there were ten) there had only been one case of recurrence. From this it seems to me we learn that cases which should be deemed inoperable when attacked from below can be successfully treated by the radical abdomino-perineal operation, though the immediate mortality up to the present is very high, but, then, desperate diseases may need desperate remedies!

---

[1] *Brit. Med. Journ.*, 1910, ii, p. 941.

I have always held the opinion that all growths fixed anteriorly or laterally, even when involving only a part of the bowel circumference, are best left alone, and indeed most of those fixed posteriorly—slight sacral or coccygeal fixation need not bar operation. If the meso-rectal glands and lymphatics are markedly enlarged, and remain so either after a preliminary colotomy or after rest in bed with frequent rectal douching, recurrence after operation is not improbable. It must be admitted that enlarged glands are often only inflammatory and not malignant. I have several times met with these glands when carrying out a para-sacral excision and have been much disappointed on finding that I was unable to remove the entire chain. Some of these cases are alive and well to-day, having passed the three years' limit—a proof, I think, that these glands were not cancerous.

Mr. Sampson Handley's paper in the *British Medical Journal* of April last is full of interest.[1] By the help of a muci-carmine stain he claims to show that the spread of rectal cancer, even in early cases, extends in the submucosa, or superficial to it, far beyond the tangible disease, and therefore joins with those who advocate the freest removal possible even for early and small cases. He records a case (evidently a very favourable one for the posterior operation) in which, by the combined method, he removed the growth and 10 in. of gut, including the sphincters and anus. He states that the disease, as proved by the muci-carmine staining process, extended up to the point of section 6 in. above the growth, "and for all he knew it might have extended higher." I ask then, wherein did the patient benefit by this extensive operation? Would she not have been better off after a para-sacral operation, with restoration of bowel continuity, and use of normal anus, and at least as well off as far as the risk of recurrence goes? If Mr. Handley is right, I cannot understand how we have managed to obtain so many cures when contenting ourselves with the removal of the growth and 1 in. of apparently healthy bowel above and below it. Either, it seems to me, that Mr. Handley's stained foci are histological and not pathological, or the cancer cells degenerate still more rapidly after the removal of the growth, until they become quite inert. Mr. Harrison Cripps published a long letter in the *British Medical Journal* last May[2] criticizing Mr. Handley's paper, and pointing out that it was quite at variance with his (Mr. Cripps's) clinical experience. With this letter I am in full accord.

A few words now as to my own results. For some ten or twelve years I have abandoned the old perineal operation, excepting for cases of epithelioma of the anus, and in the majority of cases have performed the extirpation by the sacral or coccygeal route. I have not concerned myself with the preservation of a bone flap, as patients appear to suffer no inconvenience from the loss of the coccyx and lower part of the sacrum. If the growth is extensive, involving the removal of, say, 4 in. or more of bowel, or if it extends so low down that end-to-end union is problematical, I advise a preliminary colotomy. If the anus has been preserved and the continuity of the bowel re-established the artificial anus can be closed subsequently, but bearing in mind the chance of recurrence, this may well be postponed for a year. When performing the colotomy the hand should be passed into the abdomen for the purpose not only of defining the limits of the growth, but also of exploring the liver and ascertaining the presence or otherwise of enlarged glands and lymphatics, or of secondary growths elsewhere, the existence of which would of course negative any attempt at excision.

---

[1] *Brit. Med. Journ.*, 1910, i, p. 927.

[2] *Brit. Med. Journ.*, 1910, p. 1323.

Extirpation by the sacro-coccygeal route I have carried out eighty-one times, with four deaths, or an immediate mortality of 5 per cent. There was recurrence in twenty-two cases. Hospital cases lost sight of numbered fourteen. Forty-one cases are well as far as is known; of these, one is still in a surgical home making a rapid convalescence.

The statistics of my cases in private since the year 1895 are as follows: Twenty-four cases, one death from septicæmia; eight recurrences, in the majority of cases at six months; fifteen are alive and well, and of these six have the use of the normal anus; six underwent a previous colotomy, and in one case this was closed after the lapse of two years; ten cases have passed the three years' limit. To sum up, the mortality after this operation is 5 per cent; recurrences about 33 per cent.; cures about 50 per cent.

In looking through my cases the following points especially strike me:—

(1) That a preliminary colotomy seems to offer some bar to recurrence.

(2) That when recurrence did take place it was with one exception *in situ*—i.e., at the level of the bowel section.

(3) That partial excisions are *not* to be condemned. By partial I mean the removal of only part of the circumference of the rectum. Theoretically they are unsurgical, but let facts speak for themselves. I have had five cases of partial excision. All recovered, and have passed the three years' limit. Three are quite well with use of the normal anus; one has a sacral anus; and one operated upon in 1895 died some years afterwards without recurrence. In all cases the growths were small, none larger than a florin, freely movable, and limited to the post-rectal wall. Microscopical examinations proved them all to be malignant adenomata. In one case, operated upon four years ago, I removed, in addition, four enlarged glands, in two of which metastatic deposits were found. This lady is now in perfect (rectal) health, and by a digital examination it is almost impossible to say that she has undergone any operation.

(4) Most of the cases of recurrence have been either in young subjects or in cases where the section of the bowel was perilously near

the disease—i.e., the specimen when removed did not show an inch of healthy mucous membrane above and below the growth.

In conclusion, I would express the opinion that rectal cancer in persons aged 45 and over is not prone to recur if removed sufficiently freely, but in the young it is very malignant and is likely to recur unless removed at a very early stage.

That cases of cancer of the sigmoid colon are very favourable ones for operation by the abdominal route, with end-to-end union unless obstruction is present, when a temporary colotomy should be the rule. Subsequent excision with intestinal anastomosis can then be carried out. In these cases I have had excellent results.

That for growths involving the upper rectum and pelvic colon an abdomino-perineal operation is necessary. Miles's amplification of it also has its field of usefulness for cases in which the disease has passed beyond the walls of the bowel and which otherwise should be considered inoperable.

The radical abdomino-perineal operation entails an artificial anus, which, when necessary, is no great evil, indeed, as I have said, it may be a positive advantage in preventing a tendency to recurrence. When the artificial anus is intended to be permanent, I prefer hypogastric colostomy through the left rectus muscle to an iliac one, as I find it confers a much larger measure of control. But where a subsequent abdominal section is purposed for an excision the iliac site is the better, for being placed further out the abdominal wound is not so likely to be infected.

To my mind the ideal operation for cancer of the rectum, when situated between the fourth sacral vertebra above and a point 2½ in. above the anus below, is Bardenheuer's modification of Kraske's operation with end-to-end suture. It is true that a dorsal fistula will persist for a time, but this usually heals in from two to three months if the stricture at the line of union is kept dilated. I have always found contraction take place at this point with a very marked anterior spur. This in time practically disappears if treated by dilatation, for which purpose I find nothing to equal Hegar's double-curved metal dilators.

In the face of my own results I cannot agree with those surgeons who advocate a combined operation for *all* cases of rectal carcinoma, any more than I agree with those who, for a small and limited cancer of the breast, would remove, shall I say, all but the ribs, and thus cripple the patient for life.

Mr. Harrison Cripps said he was aware of the limited time during which he could be allowed to speak, but as the subject was one which he had spent much of his professional life in studying, he found it difficult to condense his remarks sufficiently to keep within the proper time limit. It was now over thirty years since he was awarded the Jacksonian Prize for the subject, and between that time and the present he had had under observation over 1,000 cases. At first the pathology of rectal cancer might appear to be of secondary importance to a meeting like the present, as compared to its operative treatment. But that was not his view. He believed that a right knowledge of the pathology of rectal cancer was the great secret to successful treatment.

With the exception of two or three cases of melanotic cancer, and two or three cases of sarcoma, out of a large number of cases of rectal cancer he had seen, the whole of the remainder had been adenoid cancer. By that he meant a proliferation or overgrowth of epithelium of Lieberkühn's follicles and the reproduction of those follicles on a large scale. There were two ways in which those growths appeared from Lieberkühn's follicles: one when they commenced on the summit of some of the folds of the rectum, and the other where they appeared to commence at the base. He thought it was the same disease, but in a different situation. But there was a vast clinical difference. When the growth appeared on the surface of the fold, it had a free space to grow in; it grew like a tree in the free space of the rectal passage. What became of it? It became a polypus; it grew with a pedicle or trunk, it never grew to excess in size, it was not malignant, in that it did not affect the glands or general organs, and, if removed properly at the base, it seldom recurred. He regarded adenoid cancer as the same growth turned upside down. If, instead of growing free towards the surface, it grew into the muscular tissue, it no longer had the same beautiful tree-like arrangement, which made such beautiful slides, as when it grew free; but it was the same growth. When it grew downwards it became irregular, for it had to insinuate itself between the muscular fibres and the fibrous tissue of the rectal walls. In this situation it was invariably malignant. If he were shown a microscopical specimen of rectal growth and he saw no muscular fibres in any part of the specimen, he would say he believed it was innocent, though he could not say this to a certainty. If, on the other hand, the growth was between muscular fibres, he knew it was malignant disease. He would not enter, then, into the theories about that, but those two forms of growths were interchangeable. He had seen two or three cases in which there were multiple polypi of the rectum which had been going on for years. He knew of two cases, in one of which the patient was aged 25, which he published some years ago in conjunction with Sir Thomas Smith, in which polypi were removed at various times during fifteen years, and then a time came when growth started in the other direction, and the patient died of disseminated malignant disease.

With regard to treatment, while listening to Mr. Swinford Edwards, he felt that he might almost have been uttering the remarks himself, so fully in agreement with him did he find himself. He considered that the particular route by which rectal cancer was removed was a matter of quite secondary importance. The really important point in rectal cancer was whether it could be removed with advantage to the patient. Surgeons would differ very much as to what they considered the class of case suitable for removal. The class which he considered suitable was one in which the growth was still confined to the rectum, and did not extend beyond. In very many cases one could ascertain whether it was confined to the rectum by noting its degree of fixity. If he felt disease in the bowel and he was able, with his finger, to push it up or down, or rock it from side to side, he was sure it could be removed with advantage to the patient. But, if he found that his examining finger passed into a narrow part of the rectum and the disease extended round it, and he was unable to move the diseased part without at the same time moving the patient bodily, he generally found it was useless to attempt removal. He reminded the Section that he was speaking from some experience of those cases, and it took him some time to learn what conditions were removable and what were not. Some of his early cases on which operation was done should not have had that attempted, for the disease had gone through the rectal wall. Where that was the case and the neighbouring tissues were involved, he had not had a case of successful removal; and he believed that the recurrent growth was more virulent and rapidly fatal than the original. It was very important in the first examination to make out whether the cancer was movable or firmly fixed, and therefore whether it was removable or not. At the examination in one's consulting-room one could conclude that a case was not obviously suitable for removal, or that it moved nicely and was suitable for removal. But there were some cases on the borderline in which it was difficult to know whether the cancer was removable or not. As the point was a very important one, examination should be made in those cases under an anæsthetic. He had had many cases which, if he had been pressed for an opinion at the first examination, he would have said ought not to be removed, but which seemed more favourable when examined under an anæsthetic. No case should be condemned as irremovable until such further examination has been made.

With regard to recurrence of the disease, he was in accord with what Mr. Swinford Edwards said. It had been stated by Mr. Sampson Handley, as suggested to him by his pathological specimens, that rectal cancer extended by the lymphatics far beyond the actual growth. He (Mr. Cripps) did not think it did, and thought the lymphatics only became implicated very late in rectal cancer. Such widespread infection at an early stage was contrary to his clinical experience. He could not say he had ever seen cases which had recurred in the sense that large areas of bowel, 10 in., 12 in., or 14 in., had been involved. When rectal cancer did recur after removal, where did it recur? Mr. Edwards said the recurrence was *in situ*, and that was true; sometimes it recurred at the cut edge of the mucous membrane—i.e., in cases where there had not been a sufficiently free margin of ½ in. to 1 in. Where it generally recurred was not at the cut margin of the bowel, but in the tissue surrounding the part from which the growth had been removed. Cancer could be advantageously removed when the disease was still confined to the rectum, but not when it had passed this and penetrated into surrounding tissues.

With regard to the method of operating, a considerable number of cases could be reached from the anus by division of the sphincter. It would be generally agreed that a growth within 3 in. from the outlet could be removed in that manner. There were many cases situated in the common position for rectal cancer at about 4 in. to 5 in. which could be removed by the trans-sacral operation by sawing across the sacrum 3 in. or 4 in. up. That gave an astonishing view of the part. On the whole, he thought that was one of the most successful operations for the disease. There were a certain number of cases which obviously could not be felt *per rectum;* they were in the pelvis, and could only be attacked by the abdominal route. Therefore to lay down a rule and say one or other operation should be done in every case was absurd, and was discounting the surgeon's common-sense. Who would dream of removing a cancer in the upper part of the pelvis by the sacral route? or who would remove a cancer at the bottom of the pelvis below the peritoneum by beginning at the abdomen? With regard to the abdominal operation which was suitable when the disease was high up in rectum, especially if it merged on the sigmoid, there was a question of detail as to performing an anastomosis. Nothing was so successful in the sigmoid or the large bowel as the removal of a growth, and at once performing an end-to-end anastomosis. The operator would see what he was doing, there was plenty of room, and suturing could be accurately done. But when the growth was low down and there was difficulty in getting below it, and it was necessary to cut below the growth, and one had to cut across the growth above, there might be an interval of 4 in. between the two ends. Surgically, he believed it was impracticable to make a safe job of it in those cases, for even if the ends could be brought together, they had to be left in a condition of considerable tension, and there was nothing worse than to leave a junction of bowel with tension on the stitches. The junction would be sure to separate, and death ensue. It was better to cut off as low as possible, close by, invaginating the lower opening of the rectum, which would otherwise be open, into the abdomen, and then finishing the operation by fixing the upper end into the angle of the wound. That was far safer than attempting, in doubtful cases, to join the ends together by end-to-end anastomosis. In doing the trans-sacral operation it was good, if possible, to join the ends together. But in many cases that was not practicable at the time. He had had two very successful instances of doing the joining by a secondary operation. One of them was a lady who was brought to him by Dr. Rushworth, in whom he excised a piece of bowel by the trans-sacral operation, and removed 4 in., including the growth. He feared he would see the sacral nerves, but he did not. He could not join the ends together. He left the patient, and it was agreed that he should see her in a year's time, and if there was no recurrence, to see what he could do in the way of joining. He waited for more than three years, and there was no recurrence. But there gradually occurred a considerable prolapse of the upper end, and at the end of the three years he dissected out the prolapsed upper end, and the remains of the lower end, and made a very thorough anastomosis. He closed the soft parts over it. He did the original operation eleven years ago, and the second one eight years ago, and he knew that quite recently the lady was in perfect health, and she had absolutely perfect control over the anus. He wondered how such a result could have come about if he had taken the precaution of removing 13 in. of bowel, as Mr. Handley recommended?

With regard to statistics, after examining his notes, he found that not more than one in every four cases of rectal cancer which he had seen was, in his opinion, the least suitable for operation, and he believed to operate on all cases must bring discredit on the operation. With regard to recurrence, he was sorry to say his statistics were not quite so good as those given by Mr. Edwards. Out of eighty-five private cases which he had published of excision, and where he was able to follow up, thirty-five were cured—i.e., there was no recurrence after three years. The shortest case was three years and a few months, and the longest case twenty-three years. With regard to recurrence, it was fair to say that cure might be considered if there was freedom from recurrence after three years. If cancer of the rectum recurred, it almost always did so in the first year; in many cases there was recurrence in six months.

It was agreed that in selected cases the operation was very valuable, and if there was doubt in a case as to removal, the benefit of the doubt should be on the side of the operation. He would mention a case in illustration of that. One of his old pupils at St. Bartholomew's Hospital was in the Indian Medical Service. He had an obstruction, and was operated upon by Major Bird, of Calcutta, for complete obstruction. Colotomy was done. By passing his finger he could feel what he believed to be malignant disease. But owing to the hot weather the patient was advised to come to England for further treatment, and so consulted him (the speaker). By passing his finger down the colotomy opening, he could feel a growth of considerable size down the pelvis, the examination being made under an anæsthetic. It felt so fixed that when the patient came out of the anæsthetic he explained to him that he could hardly advise its removal. The patient replied that Mr. Cripps probably had in his mind that death might occur at the operation, but that he did not mind dying under the operation, but dreaded dying from cancer. He added that he would take the full responsibility for his life if Mr. Cripps would attempt the removal. He did the operation in St. Bartholomew's Hospital, and it was the worst he had ever done. It occupied two and a half hours. The growth was not anatomically fixed, but was firmly set in adhesions deep in the pelvis. He removed a growth as large as his fist, with 6 in. or 7 in. of bowel, and he never knew a patient who so nearly died. For forty-eight hours there was no sign of any pulsation at the wrist. His friends came, and he told them the patient was going in an hour or two. But the patient whispered to him that he was not going to die, and he did not. Only a short time ago he had a letter from him from India, which was dated as his eighth birthday, for he dated his life from that operation, and he sent him a 10-ft. tiger skin. His comment was, "I send you this tiger skin to show that I am in perfect health, and have not lost my nerve, for I shot this animal in one of the wildest parts of India, and I shot it on foot."

Therefore it was worth while in such a disease to err on the side of doing too much, rather than too little. Believing, as he firmly believed, that a number of those cases were curable if operated upon early—his statistics gave 33 per cent. of complete cures after operation—it was a pity that practitioners did not spend more time in the study of the symptoms of rectal cancer, as they were obvious. But time after time cases came to him which had been treated for long periods as chronic dysentery, intestinal catarrh, piles, &c., without any examination having been made, whereas if that simple precaution had been taken, the nature of the disease would have been early apparent, and they might have been treated by operation with permanent success instead of having drifted into a hopeless condition.

Sir Frederic Eve said it had been truly stated that the surgery of cancer was the surgery of the lymphatic system. It was only in recent years that the widespread permeation of cancer had been understood. Mr. Handley believed that the permeation in the bowel wall extended 5 in. above and below a rectal growth. Judged by clinical observation and the test of permanent cure, the speaker felt no doubt that operations of the Kraske, or perineal, type were not sufficiently thorough. Steinthal, taking the statistics of numerous surgeons, came to the conclusion that primary cure was obtained in only one-fourth or one-fifth of the cases operated upon. Gant, taking the work of nine surgeons, found permanent cure in only 15·6 per cent. Mr. Swinford Edwards's statistics, in his article in Burghard's "Operative Surgery," showed that out of fifty of his cases traced, in only seven did patients reach the three years' limit, and only three of them exceeded it. Every surgeon was aware that the three years' limit was too short, for many cases recurred after that time. Two of his own cases had recurred after five years. With regard to Miles's statistics, out of 58 cases traced, 54 were known to have had recurrence. Even allowing, with Mr. Cripps, that some of these cases were unsuitable for operation, it was impossible that any large number of them could have been. These facts showed very clearly that the Kraske or perineal method could not be relied on to give a permanent cure. For many years in clinical teaching he had emphasized the importance of exploring the abdomen in the majority of cases of rectal cancer. The necessity of that was obvious, to ascertain the condition of the glands, of the pelvic peritoneum, the amount of fixation of the upper part of the bowel as well as that of the lower, and finally the condition of the liver. For that reason he formerly advocated the performance of a preliminary colostomy. The advisability of exploring the abdomen was a strong point in favour of the abdomino-perineal method. In recent years his work in that department had been largely influenced by an important article by Rotter on the combined operation, which appeared in "Langenbeck's Archives" for 1906.[1] Rotter strongly advocated restoring the continuity of the bowel. The speaker, from his own experience, had come to the conclusion that the operation which fulfilled the two most important conditions—namely, a low mortality and a prospect of permanent cure—consisted in performing the abdomino-perineal operation, with the formation of a permanent colostomy. The method of controlling bleeding during the operation was of importance. After the abdomen had been opened the peritoneum was incised on the outer side of the pelvic colon and sigmoid flexure, and the large intestine, with the peritoneum, was stripped inwards to the middle line; the inferior mesenteric artery was thus exposed with the left ureter. A ligature was placed on the inferior mesenteric artery as high as was necessary, according to the condition of the glands which lay along it. That completely controlled the blood supply. The glands and cellular tissue were stripped from the sacrum, and the peri-rectal tissue removed. He agreed with Mr. Miles that in many cases the fat in the ischio-rectal fossæ and the levator ani should be taken away. Since many rectal cases occupied the ampulla, or true rectum, it seemed to him impossible to excise sufficient bowel below the growth unless the anus were sacrificed. He always separated the rectum as low as possible from the abdomen (as far as the levator ani), and then performed the rest of the operation through a perineal incision, having first closed the peritoneum through the abdomen. The operation of restoring the bowel with the combined abdomino-perineal operation could not be dissociated from the great risk of sloughing of the bowel or of leakage from giving way of the posterior sutures. It was to that sloughing and leakage, together with the more prolonged operation, that the high mortality of the abdomino-perineal operation was due. Among twenty-five cases published in 1906, Rotter's mortality was 44 per cent.; and in twenty-six cases published in 1908, Mayo's mortality was 26 per cent. During the last two years the speaker had performed the abdomino-perineal operation, combined with a permanent colostomy, with uniform success. He believed that that operation would present a very low mortality. Though he advocated it for a large number of cases of carcinoma of the rectum—viz., those occupying the ampulla or true rectum—he recognized that when the disease affected only the anal canal and anus it might be right to attack it from the perineum, but the coccyx should be removed, the sacrum cleared, and the groin glands extirpated. If the disease occupied the lower part of the pelvic colon, or supra-peritoneal portion of the bowel, so that it could not be dealt with entirely abdominally, then it was justifiable to perform the combined operation, with restoration of the bowel. The invagination method appeared to be least likely to be followed by sloughing. In performing it, there need never be any anxiety as to the possibility of bringing down the colon. Every surgeon had used the incision on the outer side of the sigmoid flexure in order to mobilize it. Mr. Moynihan had suggested that this method could be still further utilized in the combined operation to mobilize the descending colon by prolonging the incision upwards along it.

---

[1] *Archiv f. Klin. Chir.*, Berl., 1906, lxxxi, ii Theil., p. 33.

After closing the abdomen the operation was completed through a perineal incision through which the tumour, with the lower portion of the bowel, was brought and the junction effected. In this way soiling of the peritoneum was avoided, and the perineal wound was very useful for drainage of the pelvis; it also served to keep an observation on the suture line.

Mr. HERBERT F. WATERHOUSE said he was glad to find himself in almost complete agreement with what had been said by Mr. Harrison Cripps and Mr. Edwards. For once, he differed from Sir Frederic Eve, as he (Mr. Waterhouse) was an out-and-out supporter of the para-sacral method of excision. The administration for four or five days previous to the operation, by the mouth, of beta-naphthol, in 5-gr. cachets four times a day, was of much service in very greatly reducing the septicity of the bowel, and in diminishing the risk of pyogenic infection of the wound. He regarded the para-sacral method as one of choice, and thought the combined abdominal method should only be exceptionally employed. Of *operable* cases of cancer of the rectum, eight or nine out of every ten were best removed by some modification of Kraske's para-sacral incision. He thought the method was really due to Kocher, although Kocher only removed the coccyx. Though he had frequently performed the para-sacral operation, he had never found it necessary to remove any portion of the sacrum; it was always sufficient for a surgeon with a hand of ordinary size to remove the coccyx alone. He thought the removal of any part of the sacrum unnecessarily increased the shock of the operation. Moreover, as the wound was so prone to become septic it was a mistake to have in it an unnecessary section of cancellous bone. In the roomy female pelvis there was never any difficulty in performing the para-sacral excision without removal of any part of the sacrum. In the narrow male pelvis there was some difficulty, but time and perseverance would overcome it. That was also the experience of Professor Kocher, and of the late Professor Senn, of Chicago. With regard to the para-sacral method, it was not sufficiently appreciated how that could be made an aseptic operation if the anus were sewn up before the operation, or stuffed with sterilized gauze. The rectum with the cancerous segment could be drawn some inches outside the anus, and the para-sacral incision could be sewn up, before the bowel was amputated. In that way the para-sacral incision would often heal by first intention. He took it that the great question to be discussed was the relative advantages and disadvantages of some modification of the para-sacral with the combined abdomino-perineal or abdomino-anal operation. He practised the para-sacral method in nearly every case. He admitted that at first sight the combined abdominal method seemed attractive, as it seemed in accordance with surgical instinct to open the abdomen and remove the infected glands. But Mr. Cripps's, Mr. Edwards's and his own statistics showed that in the larger number of cases of rectal cancer which were really operable there were no infected abdominal glands, apart from the sacral glands, and that an operation for the removal of glands by the abdominal route was not necessary, and in many cases he thought it was unjustifiable. Moreover, the advantages of removal of glandular enlargements were theoretical in most cases of rectal cancer which were really operable. By the time the glands along the internal and common iliac vessels were enlarged the case was no longer amenable to radical treatment. Once glandular involvement had extended to the neighbourhood of the bifurcation of the aorta, the case was not suitable for radical cure. Thus the argument used by supporters of the combined abdominal methods fell to the ground. Against the para-sacral method it was urged that one could not reach high enough up the bowel. But there was hardly a limit to what could be reached in that way if the surgeon would freely incise the peritoneum at its reflexion from the rectum and divide every band which hindered the drawing down of the bowel. Many of those bands contained vessels and lymphatics, and in every operable case the surgeon could bring out the whole of the bowel below the brim of the pelvis through the para-sacral incision without any great tension being put on the vessels supplying the bowel. What one had to contend against in the para-sacral incision was tension. He would sooner practise too free division and draw down an unnecessary amount of bowel than use a stitch which was liable to cut through. He believed that many of the cases in which objections were urged against the para-sacral method were not rectal cancer cases at all, but cases in which the growth was well above the brim of the pelvis. For these the operation of choice was excision of the growth and end-to-end anastomosis by the abdominal route. The objection he had to the combined abdomino-perineal and abdomino-anal methods was that they were double operations, done at the same time, with the consequent additional loss of blood and of time occupied, and this in patients who were often worn out by pain, hæmorrhage, exhaustion and sleeplessness, and consequently ill fitted to stand the extra strain. The overwhelming objection to combined abdominal operations was their mortality. He believed it was as high as 30 per cent., or more, while that from the para-sacral excision was only a fraction of the above, either 5 or 6 per cent. He therefore asked surgeons to hesitate before choosing an operation with a mortality of 30 per cent., in preference to one of only 5 per cent. In the present state of surgical knowledge such a difference in mortality must be given great weight. One speaker had said that cancer which was not movable should be left alone. He agreed with that except with regard to posterior immobility. By the para-sacral operation excision of cancers which were fixed behind but not otherwise had given excellent, and in certain cases permanent good results.

Possibly in his remarks he had appeared to some to lay too much stress on the value of the para-sacral method, but he felt that it was a most satisfactory operation, and it had yielded him such excellent results that he welcomed the opportunity of recommending it cordially to his surgical colleagues as the routine method in cases of operable rectal cancer. His chief reasons for so doing in comparison with the more formidable abdomino-perineal or abdomino-anal operations, were its low mortality, the considerable proportion of permanent cures obtained, and the perfection of its results when either end-to-end union of the rectal segments, or suture of the upper end of the divided bowel to the anal margin, after shelling out the lower bowel segment, was practised.

(The debate was adjourned until January 18.)

Surgical Section.

January 18, 1911.

Mr. Rickman J. Godlee, President of the Section, in the Chair.

A Discussion on the Operative Treatment of Cancer of the Rectum.[1]

Mr. A. Boyce Barrow, in resuming the discussion, said he had intended to bring some patients, but they had disappointed him. His object in bringing them had been to demonstrate how a colostomy worked, and how comfortable it was to the patient. The patient got used to it, so that he came to almost disregard its presence. He had interviewed recently a man on whom he had operated three years ago, and he said he had kept very well, and had had no great discomfort. The patient reminded him that he (Mr. Barrow) had told him to take castor oil if he had trouble, but he had only to take three spoonfuls of that oil in three years, for his bowels acted twice a day, and he had no surprises at all. One patient, who had been unable to come, was operated upon by Mr. Barrow eight and a half years ago—September, 1902. He said he had kept fairly well and led a regular life; he could cycle and walk as well as of yore. He met that man after he had ridden thirty miles on his bicycle, which showed he was not much inconvenienced thereby. He was an enthusiastic advocate for colostomy in association with any operation for the removal of cancer of the rectum. He began operating for cancer of the rectum through the anus many years ago; he believed he commenced in 1880. He used to operate through the rectum, and the results were not satisfactory. The rectum became contracted and he had to do colostomy afterwards. It occurred to him that it would be wise to do a colostomy first, and so he established a colostomy opening first, as a preliminary to the operation, still removing the disease *per anum*. After a little time he did the para-sacral operation, but always with a colostomy first, and the para-sacral operation he found a highly satisfactory proceeding; but, of course, he left a blind piece of bowel above, between the colostomy opening and where the disease was removed through the sacrum. That always required washing out. It must be twenty years since he recommended the complete operation. The first complete operation he had performed he did in two parts, still doing his old colostomy first, then waiting for a week, and completing the operation by removing the whole diseased bowel, anus included. The patients did well and he was much encouraged. He therefore ventured to do the complete operation at one sitting. He thought he was the first to do that operation, but when he began to read more widely he found that was not so; that the operation had been already described, although then he had not known it. The operation when associated with colostomy was much simplified. There was not much stitching required to fix the colostomy, and, moreover, it enabled the surgeon to proceed immediately with the lower end and remove the disease most completely. He stitched up the upper end, and stitched up the anus, and he could remove the whole diseased bowel without consideration or thought of any junction afterwards. He was comparing the operation, with junction of the bowel, with that when colostomy was done. Another point in favour of colostomy was the fact that it was a much more aseptic proceeding. Once the upper end of the bowel was closed, and the anus closed and plugged, the whole proceeding was aseptic, and the course of the case afterwards was aseptic. He thought the asepticity of the operation, its completeness, and its simplicity, were very good points, and they made one advocate colostomy in association with the operation; but the patients objected very much to it. He thought, however, that everyone ought to make a sacrifice in order to get rid of a cancerous disease. He did not think many of those present would hesitate to have a colostomy associated with an operation, if they were unfortunate enough to have cancer of the rectum, and if the dangers of the operation, and the liability to return, were diminished thereby.

---

[1] Adjourned from January 10.

In any operation for cancer, in any situation, there were two elements requiring removal: that which was *recognizable* and that which was *unrecognizable* (the spread or extension of the disease, which might be called the lymphatic extension). Those two features ought to be well considered in any operation done for cancer. An operation which did not take count of both features was behind the times.

Everyone would allow that it was the wide removal of glands, &c., which produced so much better results in dealing with the disease; for instance, the removal of the axillary glands in cancer of the breast.

He had said that the abdomino-perineal operation was a very complete one, and that it was aseptic. He also contended that it was a very safe operation. He could not say he had not had a death, but that death was not exactly attributable to the operation. The fact of the operation being aseptic robbed it of many dangers; but it would be admitted that the greatest danger was that it was a long operation and a severe one, and so liable to cause much shock. Still, he had not found that shock. One of the greatest causes of shock was hæmorrhage, and in no operation was the hæmorrhage more under control than in the abdominal part of this operation for the removal of the rectum. In the abdominal part of the operation the vessels could be ligatured before they were divided. In the second part of the operation he trussed the patient up into the lithotomy position, and sat down and cut from the central point of the perineum to the coccyx. He did not find it necessary to take out the coccyx. He cleared out the whole pelvis behind the bladder and prostate, and it was surprising how little bleeding there was. The inferior hæmorrhoidals probably might bleed a little, but it could be easily controlled, and the deligation of the vessels in the abdomen had so restricted the bleeding that there was scarcely any in the perineal part of the operation.

He regarded the anæsthetic as the great element in causing shock, especially in very prolonged operations. Possibly some of those present might dissent from that view, alleging that the anæsthetic was given to prevent shock. If the anæsthetic was given very sparingly there would be very little shock, and for a long operation it was desirable to give the anæsthetic quite sparingly. He had put that matter to the test. He operated upon a patient, who was a very feeble person, for cancer of the stomach. It was found desirable to do gastro-enterostomy, but he said that if much anæsthetic were given him he would die. After the preliminary stages the anæsthetic was stopped, and the operation proceeded with Mr. Barrow talking to him during the time. The operation took one and three-quarter hours, because the Y-shaped operation was done. When the junctions were finished, he asked the patient if he would like a drink, and at his request one was given to him. Having drunk the glass of water, a little more anæsthetic was given, and the abdominal wound was then stitched up. The patient had no shock, and he did perfectly well after the operation. He asked the patient during the operation whether he was hurting him, and he replied that it was nothing of any consequence. He thought the case showed how much shock depended upon the anæsthetic.

Mr. Barrow therefore contended that the abdomino-perineal operation associated with colostomy was the safest and most satisfactory operation so far attempted for this disease.

Mr. SAMPSON HANDLEY showed two patients from whom the cancer specimens shown at the meeting were removed. The first was a man who was operated upon on December 1, 1910. He was a lead-worker, and the operation was done by combined method, with removal of the anus and its muscles, and the man had now a permanent colostomy opening. It was acting about twice a day. This was a lateral colotomy. He stabbed through the abdominal wall, so as to make only a small opening, a method which obviated subsequent weakness and bulging of the colostomy scar. The other patient was a lady upon whom excision of the rectum was done two years ago. She was now very well, and was able to travel about; in fact, she had since been to the Continent several times. The bowels were opened once or twice a day. Mr. Handley continued as follows:—

Mr. Harrison Cripps and Mr. Swinford Edwards consider that it is sufficient in rectal cancer to remove the growth as seen by the naked eye, together with an inch margin of the apparently healthy mucosa above and below. That is to say, they perform an operation which would suggest itself by the light of Nature to a surgeon entirely unacquainted with the pathology of carcinoma. Years ago a similar operation was habitually done in the case of breast cancer; the growth was excised from the breast, and the axillary glands were left untouched. It is strange to remember that as late as 1887, when Banks advocated the routine removal of the glands in breast cancer before the Harveian Society,[1] only a single speaker supported him. Step by step, as pathological knowledge has increased, the scope of the operation has gradually widened in some directions and restricted itself in others, until the modern operation for breast cancer described by Mr. Edwards with equal vigour and inaccuracy as "removing all but the ribs and crippling the patient for life," has established itself. And at each step of this progress there has been a marked improvement in results, so that local recurrence in the field of operation is now a rare event, and functional disability is practically absent.

In the operative treatment of rectal carcinoma we may anticipate with confidence a similar process of evolution. The necessity of removing a considerable length of bowel is shown not only by my own researches, but by the clinical results of Mr. Swinford Edwards, which are exactly in accordance with my observations. In all his cases of recurrence, with one exception, the growth returned locally at the level of the bowel section. It would be difficult to bring forward a stronger argument for the removal of an increased length of bowel both above and below.

---

[1] *Lancet*, 1887, i, p. 627.

Mr. Harrison Cripps argues that my observations on the spread of rectal cancer must be incorrect, because recurrence when it takes place involves only a small area at the site of operation. He implied that if permeation is so extensive the obvious recurrence must be equally extensive. To this I would reply that I have seen a primary sigmoid growth extending along the wall of the bowel nearly to the splenic flexure. I show a specimen of rectal cancer with an isolated oval patch of secondary growth extending up the bowel 3 in. above the upper border of the primary growth. As a rule, however, the obvious recurrence is very local for two reasons, first because permeation is a slow process which only produces naked-eye phenomena after a considerable period. It is only in those regions where permeation has been long present, that is to say, in the region close to the primary growth, that naked-eye recurrence is to be expected. Secondly, the area of the obvious recurrence is reduced by shortening of the affected length of bowel owing to contraction of the growth in a longitudinal direction. To remove an adequate length of bowel, to control hæmorrhage at the beginning of the operation, and above all for the careful removal with visual aid of the fat and glands of the meso-rectum, the perineal and sacral operations must be replaced by the abdomino-perineal methods.

The attempt to preserve the natural anus by bringing down the cut end of the sigmoid leads, as Continental experience has shown, to a largely increased operation mortality. Moreover, it involves the risk of recurrence in the anal region, a calamity which took place in the last case I operated upon by the perineal method. For these reasons, speaking generally, I advocate an operation on the lines described last year by Mr. Miles, and I believe his recent paper will lead to a great advance in the treatment of rectal cancer in this country. I have myself practised a similar operation for two years, and in three cases. All of these are living and have no signs of recurrence. Had the operation been the dangerous adventure depicted by Mr. Harrison Cripps I can hardly believe that fortune would allow me an initial sequence of successes. Goullioud has published a series of eleven cases in women, all of whom recovered.

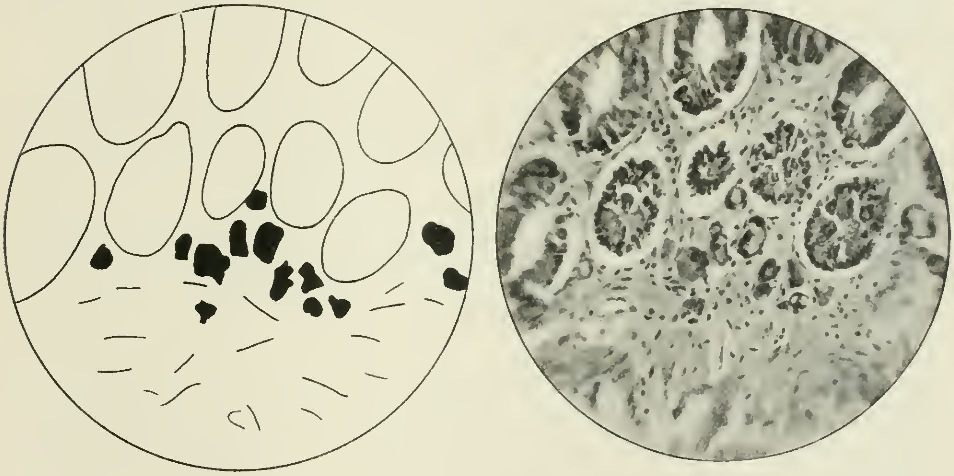
It is necessary to recognize that the radical abdomino-perineal operation is a severe one, and that its mortality is higher in men than in women. In old or feeble patients of either sex, but more especially in men, I should still employ a limited perineal operation if the growth were situated low down, or should content myself with a colotomy.

Mr. Harrison Cripps has stated in reference to a case of mine operated upon by the combined method, that according to my own statement I had done a perfectly useless operation because, on examining the specimen, I found evidence of permeation of the bowel as far as the line of section. I am quite ready to admit that I failed to attain the surgical ideal of complete removal, but my operation secured a better approximation to it than the limited operation which Mr. Cripps advocates, and in that way I believe that I minimized, so far as was possible in that particular case, the chance of recurrence.

Surgeons often claim far too much credit for their successes in dealing with cancer. An absolutely clean removal of every cancer-cell present in the body is probably a rare event. Freedom from recurrence is common because natural processes of cure destroy or inhibit the few remaining cancer-cells. These facts explain the not inconsiderable amount of success attainable by very incomplete operations. They in no way justify the abandonment of the surgical ideal of complete removal, for experience has shown, more especially in breast cancer, that the more extensive operations, if guided by pathological knowledge, produce greatly improved results. The ideal may be unattainable, but the struggle to reach it finds its full reward. Mr. Harrison Cripps's results have been attained by a very rigid selection of cases. I gather that only in one case in every three or four seen by him does he advise excision. The vast majority are left not only with their disease, but often, I presume, with the colotomy opening which Mr. Cripps so much deprecates in his criticism of Mr. Miles's and my own methods. One advantage of the combined methods is that they widen the limit of operability and enable us to aim more often at cure instead of merely at palliation. If the perineal method is applicable only to one case in four of rectal cancer, surely it cannot be the ultimate resource of surgery. It is the duty of surgeons, in dealing with rectal cancer, to advance along the lines laid down by pathology so far as they are able without subjecting the patient to undue risk.

It is, of course, to be much regretted that a reasonably safe, and at the same an adequate, operation can only be done at the price of a colostomy opening. But the disability, though real, is not inconsistent with a normal life of work and social intercourse. The methods which aim at a restoration of the normal function of the anus are only successful in a small percentage of cases. The ideal result of these cases is dearly bought at the cost of many operative fatalities, or at the cost of preventible recurrences. Other cases remain with a permanent sacral anus, or suffer from incontinence or stricture. If the surgery of rectal cancer is to advance, the ideal of restoring anal function must be abandoned as inconsistent with the other and far more important ideal of complete and safe removal of the disease on the lines laid down by pathology.

It must, however, be admitted that the pathology of rectal cancer is not yet fully worked out. For some years I have been endeavouring to trace the spread of cancer of the rectum, and until recently without success. Owing to the proneness of the carcinoma cells to mucoid degeneration and the consequent difficulty of recognizing them in microscopic sections, about two years ago it occurred to me to employ mucicarmine, a specific stain for mucus, and by the aid of this stain I thought I was able to demonstrate the spread of permeation along the mucous lymphatic plexus of the bowel a distance of 6 in. from the primary



Permeation of the mucous lymphatic plexus in rectal cancer (case of J. S., patient shown at the meeting). The figure shows, in its upper part, the deeper portion of a number of normal crypts of Lieberkühn. Between these and the muscularis mucosæ, which is seen at the bottom of the figure, are a number of irregular, darkly stained, vacuolated cancer-cells, which are marked black in the attached key-diagram. The section was taken from a point considerably above the growth, where the rectum appeared normal to ordinary naked-eye and histological examination. Section stained by hæmatoxylin and muci-carmine. (Photo × 180, by Mr. R. W. Annison.)

growth. Mr. Cripps has no doubt that I was mistaken. I am able to-night to demonstrate another case showing similar appearances extending to a distance of 4 in. from the obvious or naked-eye edge of the growth. It rests with Mr. Cripps to explain what these appearances are, if they are not those of permeation of the mucous lymphatic plexus by cancer-cells. I have been unable to find anything similar in a small series of normal rectums. Though an occasional isolated element may take on the muci-carmine stain, nothing can be seen in the normal bowel in the least comparable to the small groups of sharply defined mucoid cells, irregular in size and shape, which may occur at frequent intervals in the cancerous rectum to a variable distance from the macroscopic edge of the growth in the plane of the mucous lymphatic plexus. Beyond a certain point they cease to be visible, and that point is, in my opinion, the true microscopic growing edge of a rectal carcinoma, within which the surgeon's knife must not willingly intrude.

The PRESIDENT (Mr. Rickman J. Godlee) asked Mr. Handley at what distance from the original growth the last section shown was taken. Also how far from the original growth had he found cancerous cells?

Mr. SAMPSON HANDLEY, replying to the President, said the section was taken from fairly near the growth because, wishing to demonstrate the process clearly, he took one of the best fields he could. In one case he had found scattered foci 6 in. from the growth, and in a second case 4 in. away.

Mr. F. C. WALLIS said that during the last ten years the surgery of the rectum had made great progress and this was more particularly the case in other countries. But in this country also in the last few years a much more intelligent interest had been taken in this branch of surgery by practitioners at large, who more and more appreciated what a valuable asset a proper knowledge was of this subject and what before were looked upon as more or less mysteries were now treated in a rational manner, and thus the relief of pain for the patient was much earlier and of a more practical character. The first essential for good results was early diagnosis, and that could only be obtained by a proper examination being made at the earliest moment the symptoms were complained of, instead of the whole thing being shirked as long as possible by both practitioner and patient. This was a grave responsibility which rested with the practitioner, and it behoved those who had the opportunity of early examination to do this as early and as thoroughly as possible. If this was made a routine practice the diagnosis would be made much earlier, and so the results of the operation would be greatly improved. Before deciding if any operation should be done in cases of rectal carcinoma, one of the most important matters to decide was whether the growth was a fixed one or not. This decision, as a rule, should not be made until the patient had been examined under an anæsthetic, and even then the decision might be of some difficulty, especially if the growth was adherent in front to the bladder, vagina, or uterus. In such cases the growth might be easily pulled down with the viscus to which it was adherent, and thus give a false idea of free mobility. When any such possibility as this occurred during diagnosis, it would be obviously wrong and might also lead to serious disaster if an operation was undertaken either from the perineum, or by one of the posterior routes, and when the growth was arrived at, it was found to be fixed. Also other parts of the colon and even the small intestines might adhere to the growth. To obviate any such catastrophe, and at the same time to obtain a more thorough knowledge of the growth and its surroundings, an exploratory laparotomy should be made in the mid-line. This was an expediency which he suggested should be made much more use of than was at present the case. The question as to what particular operation should be undertaken was one which was almost acute in its present interest. Any new operation which involved a decided alteration of technique was usually commenced with enthusiasm. The enthusiasts often overdid it, and operated on all sorts of cases which were in one way or another unfitted for such a procedure, with the result that the operation was discredited by those whose practical experience, having extended over some years, had settled into a groove in which work might run smoothly, but perhaps not so much progress was made, and thus an important advance had often been checked for a considerable time. It was to be sincerely hoped that such would not be the fate of the abdomino-anal operation which was now so much advocated, and was a good one in suitable cases. The technique of this operation had been thoroughly described, and the main details of it could soon be learnt by one or two operations on the cadaver. But this was the least difficult part of the work. The age of the patient, the character and size of the growth, the situation, particularly with regard to its surroundings, the length of the sigmoid loop, the size of the pelvis, and the amount of fat generally in the abdominal cavity, were all matters of clinical importance, which it was essential to consider before undertaking this operation. It could be truly said that most of these points must be considered in any operation for a high-lying cancer of the rectum. But it was probably for the low-lying carcinomata that this operation would obtain the best results. The involvement of the lymphatics in the submucous plexus in certain cases of cancer of the rectum was admirably demonstrated by Mr. W. Sampson Handley, but whether this was satisfactory to everybody was not certain. Personally, on reading Mr. Handley's views, and thinking over his (Mr. Wallis's) past cases, he was much impressed with the fact that a certain proportion of such cases which had done extremely well, had had a return of the growth in the lower segment after an interval of a year or eighteen months. And he (Mr. Wallis) thought that in such a case if the abdomino-anal operation had been performed, and the bowel above the growth more freely removed, recurrence would not have occurred. It was for such cases that the the abdomino-anal method was desirable, and from which probably the best results would be obtained.

He could not feel enthusiastic as to the ultimate fate of the bigger operation which had been performed—namely, excision of the whole of the rectum and possibly a portion of the sigmoid, with the establishment of an iliac anus. It could only be very few cases in which such a measure was justifiable, and even amongst the few that were justifiable it would hardly be expected that anything but a high percentage of mortality would obtain. When a rectal carcinoma required such severe measures it was probably far better treated by an ordinary colotomy, and he could give various instances of people who had lived comfortably with a colotomy and a carcinoma for many years, the longest he knew of being ten.

With regard to the other operations, he was strongly of opinion that the best method of dealing with rectal growths not involving the sphincters was by the sacro-anal method. The great point which he claimed in its favour was that instead of suturing the last 2 in. or 3 in. of the bowel to that which was brought down, the anal mucous membrane was excised and the cut end of the bowel above the growth brought down and stitched to the anus. It was quite a simple matter and all possibilities of a sacral anus forming and the almost certainty of a stricture were done away with. This was a method which he had adopted for the last twelve years, and which could be well combined with the abdominal incision when necessary. When the growth involved the sphincters, then a wide excision of these muscles was necessary, and this operation could usually be performed through the perineum, especially if a preliminary colostomy had been performed, and indeed in such cases this was essential.

When a growth was inoperable there was no doubt that great comfort was given to the patient by a colostomy. He (Mr. Wallis) had already mentioned that patients lived in comparative comfort with good general health for many years after this operation had been performed, and he was constrained to mention it again, because he remembered a few years ago, at the Medical Society of London,[1] when a discussion took place on this very subject, that grave doubts were passed upon any possible benefit being derived from such a measure. There was no question as to the immense benefit derived, especially if the toilet of the colon, which was well known now to most good surgical nurses, was daily carried out, and if the growth itself was prevented as far as possible from becoming septic by appropriate treatment.

He could not advocate any of the various fancy operations for colostomy, which in his experience only caused trouble, and rarely, if ever, possessed those particular virtues which were claimed for the operation. One other point he would suggest in the operation, and that was that the bowel should be opened at the time of the operation. This saved much trouble, was much more comfortable for the patient, and, with ordinary care, was entirely free from all risks. He had no knowledge of any good effects which had been produced by the action of radium upon a rectal carcinoma.

Mr. P. LOCKHART MUMMERY said he thought it necessary to insist very strongly upon one point upon which Mr. Wallis also touched—namely, the large proportion of inoperable cases of cancer of the rectum which were seen. Mr. Harrison Cripps stated that only one case in four of those which he saw was operable. In his (Mr. Mummery's) own work, out of 100 consecutive cases from his note-books he obtained the following figures: Of the total, 35 were operable, 57 inoperable; 7 refused operation. His definition of an inoperable case was that it was not possible to remove the growth, even by an extensive abdomino-perineal operation, either because the growth involved structures which could not be removed, such as the bladder, or because there were secondary deposits; or, in a few cases, because the patient's condition did not warrant the risk of operation. The larger proportion in Mr. Cripps's figures was easily accounted for by the different view which that gentleman took on a question of what was and what was not operable. He thought he could claim to be as optimistic as most surgeons with regard to removal of growths from the rectum, and there was no doubt that 57 per cent. of inoperable cases was too large, and that such a proportion was preventible. There were two causes for this: First, that the patients often did not seek advice until too late. But what he believed accounted for the larger proportion was that the medical man who first saw the patient did not make a local examination when there were symptoms in the rectum. This omission was exceedingly common. He was continually seeing, at St. Mark's Hospital, patients who said they had been treated by their doctor for piles for six months, and that no local examination had been made. On examination a large carcinomatous growth was found. Such cases were seen almost every week. It was impossible to produce statistics on the point, but his experience led him to say that the time from the beginning of carcinoma to its inoperable stage, varied from six to nine months. At St. Mark's a common period for the cases to be seen was at the seventh month—i.e., just before they became inoperable. If such cases could be seen quite early, there would be an immense advance in the successful treatment of cancer of the rectum. At present there were means, by using the sigmoidoscope, of diagnosing with absolute certainty any growth in the bowel at its earliest stage, when it was little more than a papule, at any point below the middle of the sigmoid flexure. He would exhibit a drawing of the earliest case of carcinoma he had ever come across, the growth having been detected through the sigmoidoscope. He removed the growth. If the condition of the patient was favourable, and he was not subject to any other disease, operation should be done for removal of growth in the bowel whenever there was a reasonable chance of saving the patient's life, because the result was so bad if no operation were done; moreover, even in the cases which were apparently hopeless, he had frequently seen most brilliant results. What was apparently one of the most hopeless cases he ever saw, and only just operable, was that of a patient upon whom he operated eight and a half years ago. The case had been declared by other surgeons in London to be inoperable. He removed the rectum with half the sigmoid flexure and restored the bowel, and the patient was alive and in the enjoyment of perfect health to-day. He was 33 years of age at the time of operation. It did not do to pick one's cases too carefully, and it was not justifiable to try to get statistics which would yield a small percentage of recurrences. The duty of the surgeon was to do his best for the patient, and not to try to get satisfactory statistics. The surgeon should tackle border-line cases whenever they presented themselves, and the fact of doing that would spoil good statistics. Though there had been some failures from following that policy, the number of successes was sufficient to justify a continuance of that attitude. He was one of the earliest in England to advocate the removal of the entire rectum in every single case of carcinoma of that part, and he still believed that that was correct. And, whenever possible, the entire rectum should be removed, not only because it gave a better chance of removing the whole growth, but also—and there he differed from Mr. Handley and Mr. Miles—because it gave the possibility of restoring the bowel to the condition it was in before. It was possible, by removing the entire rectum, to bring the sigmoid to the anus and give the patient perfect sphincteric control. He had several such patients now with their sigmoid flexure opening at the anus, and their sphincters were normal. He admitted that if the growths were large and near the anus there was greater risk of recurrence, but no absolute rule could be laid down as to what should and what should not be done in rectal cancer. The surgeon must be guided by the circumstances, and where there was a small growth low down the means must be modified. An excellent result could be obtained by removing the rectum and bringing the sigmoid to the anus. One advantage of the complete abdomino-perineal operation, which Mr. Cripps had not given it credit for, was that it enabled one to resect the growth in many cases of carcinoma of the rectum which otherwise would be inoperable. There were many cases at the recto-sigmoidal junction which he would defy any surgeon to get at by any other operation. The ideal operation for cancer of the rectum must be one in which aseptic healing was the rule—in which the patient would not have a large sloughing, suppurating wound ; it would also be one in which the bowel could be restored. It was possible to remove a part of bowel as large as Mr. Handley's specimen, and also to bring the sigmoid down to the anus. The only difference was that it was necessary to leave the sphincters. By carrying the incision to the right side of the sigmoid colon, one could almost bring the splenic flexure to the anus. The ideal operation was not one which left a colostomy behind. After extensive removal, the bowel should be restored. The exception would have to be in those cases where the growth was near the anus ; then, of course, the sphincters could not be preserved. The point upon which surgeons ought to concentrate their attention was aseptic healing. He had had only three cases in which that had occurred after removal of the entire rectum. In a recent one, in which he did the abdomino-perineal operation, he took away everything from the sigmoid to the anus, and the patient was out of bed in eleven days, and in three weeks returned to his home in the country unaccompanied. Another case was that of a lady who was out of bed in ten days, and she also went away in three weeks. In neither of these cases was there any temperature or any anxiety. The third patient was one in hospital, and he also went out in three weeks. He believed that most of the mortality from excision of the rectum by the abdomino-perineal excision was due to peritonitis and sepsis. The reason the mortality was low in the operation advised by Mr. Cripps was that in those cases sepsis did not much matter, as it did not involve chiefly the peritoneum. In the more serious operations sepsis was a more important matter, and might cause the life of the patient to be lost.

---

[1] *Trans. Med. Soc. Lond.*, 1903, xxvi, p. 241.

Mr. C. H. Fagge said that his own small experience had been entirely opposed to any form of perineal or sacral operation, and now for nearly three years he had, except on one occasion, confined himself to the abdomino-perineal route; his experience of Kocher's method (for he agreed with Mr. Waterhouse that if the perineal route were chosen sufficient room could be obtained without removal of any part of the sacrum, and therefore a complete Kraske was unnecessary) had been unfortunate, for all except one of his cases, about ten in number, had died of recurrence after intervals of six months to three years. He thought the first speakers who were in favour of perineal methods alone had given their case away by admitting that this method was unsuitable when the growth had extended beyond the bowel wall, for in his experience many patients did not come under observation until the perirectal tissues were permeated, as one found by dissection after operation and by microscopical examination of the parts removed. In other cases, too, which had not advanced so far, to limit the excision to the bowel only was sailing too near the growth. He believed that the abdomino-perineal route would give some measure of success even in these cases.

Some three years ago, when becoming dissatisfied with perineal methods, he met with two cases which finally settled the question as far as he was concerned; in the first, Kocher's operation on a man was followed by death from inhalation pneumonia in a few days, and at the autopsy several secondary deposits were found in the liver; the second case was sent to him by one of his medical colleagues, who was very pleased to have detected an early case with the sigmoidoscope, but he (Mr. Fagge), after his last experience, refused to operate from below without first exploring the abdomen, when he found cancerous peritonitis, and of course no radical operation was done. He had carried out the abdomino-perineal operation six times on patients of his own, and had also on several occasions assisted one of his senior colleagues. In some cases they had operated together, one from the abdomen and the other from the perineum, but they had found it unsatisfactory. He had in one of these six cases carried out the operation in two stages, but the bowel, detached and pushed down into the pelvis, sloughed, and the patient died of peritonitis : he had seen the same thing happen in the practice of another surgeon. In one of the six patients abdominal exploration for acute obstruction led to a diagnosis of cancer of the rectum to which the left tube was adherent; at a second operation these organs were removed by the abdomino-perineal method, when the condition causing obstruction was found to be tuberculous salpingitis widely infiltrating the rectal wall. The patient got well. In the other five, microscopical examination showed typical carcinoma; three died as a result of shock or peritonitis shortly after operation, and two recovered, but neither had yet passed the three years' limit. As an alternative, when the growth was situated in the upper part of the rectum, the abdomino-anal method, as described by Miss Aldrich Blake, and as practised by Mayo and others, was to be considered, but he was of opinion that for growths in the ampulla—the usual site of rectal neoplasms—it was wise to sacrifice the levator and other tissues around the bowel. An attempt to preserve the sphincteric apparatus ran two risks, of which the first was local recurrence and the second an incontinent functionless anus, which was probable after an operation necessitating a perineal dissection, which rendered it difficult to ensure that all the muscles and their nerves should be left intact; he thought a patient was more comfortable with a colostomy under such circumstances, so that as a routine operation for rectal cancer he preferred the abdomino-perineal method.

Mr. GORDON WATSON said that most of the points he intended to speak on had been mentioned by others. Since April, 1909, he had performed the abdomino-perineal operation ten times, and had assisted others on nine occasions. He had also watched distinguished operators perform the operation. In the cases on which he had himself operated, he had only twice brought down the bowel to the anus. Of his ten cases four died, two of them three weeks after the operation—one of pneumonia and one from cellulitis—and two within a week of the operation for peritonitis. The chief objection which most people would raise to the abdomino-perineal operation was its high mortality ; but the same kind of thing occurred in connexion with the operation of panhysterectomy by the abdominal method when first introduced. For the first few years the mortality was about 40 per cent., whereas a gynæcologist told him recently that the present mortality was probably under 10 per cent. He considered that the latter figure would be reached in the abdomino-perineal operation if the cases were carefully selected. Patients over a certain age, with fat abdomens and thick arteries, were not suitable for such an operation. With regard to the technique of the operation, he would like to bring out certain points. No patient ought to die of shock after this operation. Each of his patients had been given a ¼ gr. of morphia an hour before the operation, most had had saline during the operation, given by means of Arbuthnot Lane's rubber bags. Some had had oxygen, passed through absolute alcohol, given with the anæsthetic. In not one of his ten cases had there been danger or grave anxiety on account of shock after the operation. He was sure shock could be eliminated. With regard to the length of the operation, the longest time he had taken was one hour forty minutes, and the shortest time was one hour. He did not think that was too severe a strain for that procedure. If the mortality could be reduced, he did not think any surgeon could object to it on pathological lines, as had been pointed out by Mr. Handley and others. He had not succeeded in discovering permeation in the portions of bowel above the growth, which Mr. Handley had demonstrated that evening, but he had kept all the specimens, with a length of bowel above and below, and would further investigate the point. He wished to emphasize another point, namely, the method which Mr. Wallis practised, and which he (Mr. Watson) had several times assisted him at, namely, bringing the bowel down through the sphincters without dividing the sphincter. But that was not suitable for cases in which there was a growth in the lower 5 in. of bowel, because he was convinced that later on, though the cases might do very well at first, recurrence would happen locally where the sphincters were not removed. The procedure was admirably suited for growths above the reflexion of the peritoneum from the vesiculæ seminales on the one hand, or the vagina on the other. He would like to know the opinion of Fellows with regard to prophylactic injections of coli vaccine. He had given it experimentally in several cases, but it was difficult to arrive at conclusions. The idea was to give it before operating so as to diminish the chances of peritonitis by raising the resistance of the patient. He believed that the great risk connected with the operation was the occurrence of peritonitis the result of sepsis. Mr. Handley mentioned that he had had three consecutive fatal cases. He (Mr. Watson) also had three consecutive successful ones, but they were followed by three consecutive fatal cases, though in two of them he felt he was partly responsible, because he gave coli vaccine within thirty-six hours of the operation, which was due to a misunderstanding between the pathologist and the house surgeon. Since that time he had given the vaccine a week before operation, and during that week he had had the teeth carefully attended to. Naphthalene tetrachloride was also given every four hours, in 5-gr. cachets, and the rectum irrigated daily with a view to diminishing the intestinal flora for a week before operation.

Mr. SAMPSON HANDLEY, in reply to Mr. Gordon Watson, said he had not used anti-coli serum, but he had used anti-streptococcic serum in one case. That case recovered, but he could not say what part the serum played in the recovery.

Sir FREDERIC EVE said he had used anti-streptococcic serum, as a routine measure, before excising the rectum. It seemed to do good; he could not say more than that.

The PRESIDENT congratulated the Section on the very interesting discussion which had taken place during the two evenings allotted to it. Before calling upon Mr. Swinford Edwards to reply, he said he would like to mention that he knew one case in which carcinoma of the rectum did not recur until ten years after removal, and then the recurrence took place in both the lung and the rectum. He had been much struck, when doing the combined operation, with the small amount of shock which resulted. It did not seem to matter whether the operation were done quickly or slowly; in fact, sometimes the more slowly it was done in moderation the better. The real point of importance was to avoid stretching and pulling on the structures. Even in the prolonged operations there was a small amount of shock compared with that from Kraske's operation. What appeared to be the great objection to the combined operation was that he at all events could not be sure, however carefully it was performed, that there would not be sloughing of the posterior part of the bowel. He had no dread of peritonitis; that did not occur if the peritoneum were sewn up carefully. He did not succeed in any large proportion of cases in getting healing by first intention; in fact, he only remembered one genuine case. Therefore, he was becoming convinced that the operation of choice in the immediate future was the removal of the rectum and lower part of the sigmoid flexure, leaving a permanent colostomy wound. An unsatisfactory anus in the right position was less satisfactory than a satisfactory colostomy wound.

Mr. SWINFORD EDWARDS, in replying on the discussion, said he could confirm the President's remarks as to the extraordinary absence of shock after the combined operation. His last case of excision of the rectum took place at St. Mark's Hospital a fortnight ago, when he did the combined operation, and occupied three hours and a quarter over it. But the patient was doing remarkably well, and had no shock. Therefore he was not entirely wedded to the para-sacral operation and he thought that all cases should be judged on their own merits. By the operation advocated by Mr. Miles, which he called the radical abdomino-perineal operation, he (Mr. Edwards) conceived that cases had become operable which previously would have been considered inoperable; for Miles showed, as Mr. Edwards said in his paper, that if the cases were analysed it would be found that the disease in all had passed beyond the confines of the bowel itself, having become adherent either to the organs in front, or to the sacrum behind. Most speakers had emphasized the point that, no matter how small the cancer might be or where it might be situated, the combined operation should be performed, though it was admitted that the operation entailed a mortality of 30 to 40 per cent. He very much doubted whether, if that fact were properly put to the patients, they would agree to undergo the operation, especially if they were told they would, if they recovered, have to have a colostomy opening. There were cases in which the cancer was situated low down, perhaps not larger than a shilling or a florin, and limited to one wall, which could be dealt with quite locally. He pointed out that he had had in private practice no less than five cases of partial excision for that particular condition. All those cases were till recently alive, and had passed the three years' limit, and had retained the use of their normal anus, the exception being that one died from lung trouble, but some years after the operation. With those facts before him he did not think he would be justified in recommending the radical abdomino-perineal operation for such cases. That operation had its own field of usefulness, viz., when the growth was either somewhat adherent or was situated in the upper rectum—i.e., at the junction of the sigmoid and rectum. If the combined operation were done, it was well to leave a permanent colotomy opening, and remove all the bowel below it. In the case operated upon a fortnight ago, he removed 18 in. of bowel, and established an artificial anus. If one attempted the procedure recommended by Mr. Mummery of bringing the sigmoid down to the anus, having excised all the bowel between those two parts, he thought there would be great risk of sloughing. He had seen it happen more than once, and of course the result might be fatal.

He was very glad the opportunity had been afforded of having the discussion, especially at this time, for evidently there were two opposite camps. He hoped he did not belong entirely to one, but rather to both.

Surgical Section.

March 14, 1911.

Mr. Rickman J. Godlee, President of the Section, in the Chair.

Traumatic Myositis Ossificans.

By G. H. Makins, C.B., F.R.C.S.

The following cases appear to be of some interest, because I am in a position to exhibit skiagrams illustrating the development and spontaneous cure of the condition, as well as a microscopic section of the bony deposit in one of the cases concerned.

In a paper by Schultz [3], dealing with the occurrence of traumatic myositis ossificans in the German Army during a period extending over ten years, the respectable number of 233 cases has been collated, showing that the affection is more frequent than is commonly supposed. I take it that the frequency there shown depends upon the fact that soldiers are carefully examined for any defects in physical efficiency, while amongst the civil workers the minor degrees of such a condition as this are often overlooked. Amongst Schultz's cases only three are noted as affecting other muscles than the quadriceps extensor femoris and the brachialis anticus. The exceptions are two instances of ossification of the masseter following horse-kicks and one of the pectoralis major due to a blow during bayonet exercise. In a paper by Ewald [2] examples are quoted of ossification of the deltoid, triceps, biceps, adductor magnus, and subclavius following accidental injuries, also Horvath's remarkable case, in which ossification of both iliaci followed forcible manipulation of a flexed and adducted hip-joint. The common history of all these cases is the reception of an injury, such as a kick or blow, rupture of the muscle from over-action, or, in the case of the brachialis anticus, laceration of the muscle accompanying a dislocation of the bones of the forearm backwards. The injury may be followed immediately by the development of a hæmatoma, the swelling consequent on which may persist until the more lasting hardening due to the ossification appears. More commonly a latent interval or a period of improvement is followed in from ten to twenty-one days by the development of local pain and limitation in the movements of the neighbouring articulation. A hard, resistant tumour is then discovered in the area of the affected muscle, which may persist for long periods, or become more or less completely absorbed with some rapidity.

The cases are as follows:—

Case I.—A. T., aged 19, hosier's assistant. Six weeks before admission in July, 1905, the patient received a kick on the front of the left thigh while playing football. He had received the same injury on two previous occasions, but treatment with hot fomentations caused the swelling to disappear in two or three days. On this occasion there was bad bruising and considerable swelling of the knee, which after a few days completely disappeared. Two weeks later the patient noted that the thigh had commenced to swell again and a "lump" formed which gave him considerable pain, especially on flexion of the knee. The swelling had not increased during the past two weeks, and the patient has been able to use his limb more freely. As a shop assistant he is on his feet most of the day. On admission an obvious swelling existed in the middle third of the front of the thigh, the superjacent skin was normal in appearance and temperature. The swelling was fusiform in outline, at its upper and lower ends the femur could be palpated at a deeper level. The inner margin of the swelling was somewhat sharply defined. There was no pain or tenderness on palpation, but flexion beyond half the normal range caused pain, while no movement was produced of the tumour. A skiagram showed a definite plate of bone somewhat to the outer side of the femur and unconnected with the bone (fig. 1). The patient was kept at rest for seven weeks, during which time the tumour somewhat diminished in size and became less defined in outline. He then returned to his work, and has since suffered no practical disability. A skiagram taken on February 3, 1911, six years later, showed no trace of the previous ossification. The movements of the knee were normal, and no swelling could be detected on palpation. The patient continues to box and play football.

Case II.—C. B., aged 19, railway porter. Three months before admission in July, 1905, the patient received a kick from a horse on the front of the right thigh. The kick was a severe one, necessitating rest in bed for a week. Swelling was noted on the day following the accident, and the patient does not consider that this has either materially increased or diminished since. There has been little pain, except on going upstairs; at times the limb feels as if "giving way under him." On admission to the hospital a swelling of fusiform outline and 5 in. in length was found over the front of the middle third of the femur. It was quite hard and immovable, apparently fixed to the anterior surface of the bone. The skin was somewhat stretched over it, the superficial veins were dilated, and a small pigmented area corresponding to the point actually kicked still existed.

A skiagram showed the tumour to be composed of bone of about the same consistence as normal cancellous tissue (fig. 2). For about an inch at the central part no interval could be demonstrated between the tumour and the femur, but the two ends of the tumour were quite free from the bone. The arrangement of the laminæ suggested that the tumour consisted of an ossification in the substance of the quadriceps. On July 19, 1905, Mr. Clutton exposed the tumour by a longitudinal incision on the front of the thigh and removed the greater part of it. The bony mass was surrounded by gelatinous layers suggesting bursal tissue. Some septa of similar tissue and what appeared to be muscle ran for

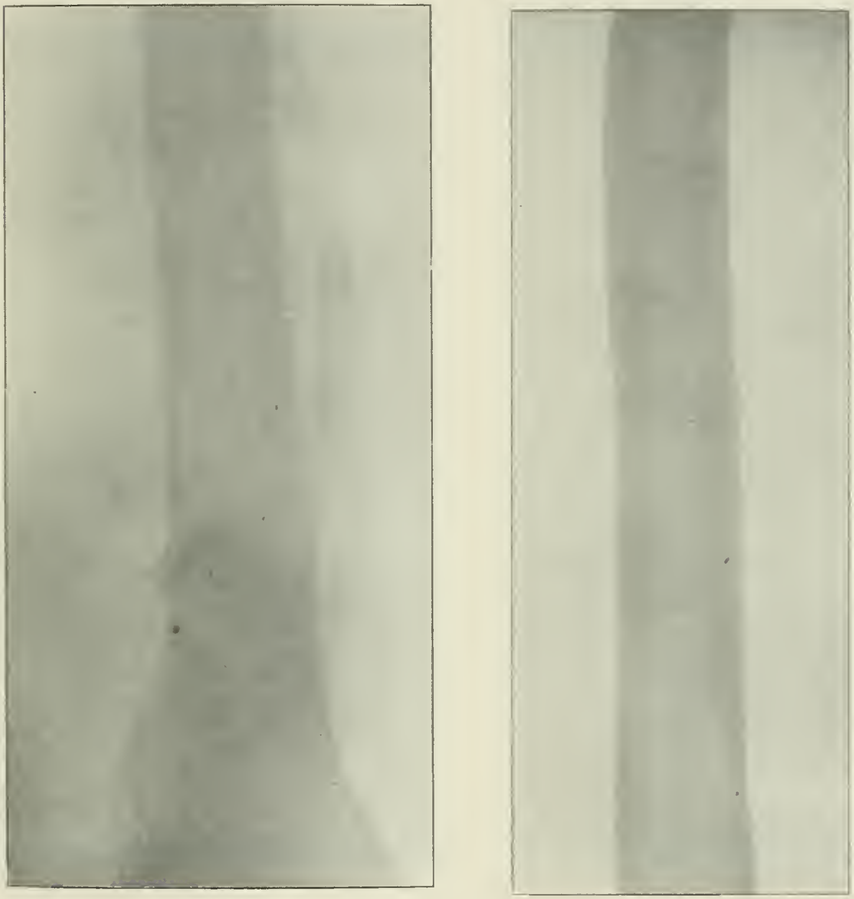


FIG. 1.

Case I, A. T., six weeks' standing.

Six years later, showing complete disappearance of the mass in the quadriceps.

some distance into the bone. The periosteum was not exposed. Except for the development of a hæmatoma, the wound healed normally, and the patient left the hospital on the nineteenth day after the operation. A skiagram taken on February 1, 1911, shows a plate of bone about ½ in. in thickness closely applied to the femur beneath the site of the upper part of the original ossific tumour. The patient has been doing his work as a railway porter for the past five years, suffering no disability, except slight restriction in the amount of flexion possible at the knee. No tumour is detectable on palpation.

Case III.—S. C. B., aged 23, student. Seven weeks before he came under observation, while playing football the patient had an accident, another player falling over him and kicking his knee. A week later, while playing football again, an onlooker pointed out to him that he had "water on the knee." For the next six weeks he was kept at rest and treatment directed to the injury to his knee. At the end of seven weeks he came under my notice, when a hard fusiform tumour was present over the middle third of the femur. Flexion of the knee could only be carried out to about half the normal range, and attempts at further movement were painful. A skiagram showed a large mass of bone in the deep part of the quadriceps, no interval existing between it and the femur. It was well-formed, delicate bone in irregular lamellæ. The femur was normal. The patient was kept at entire rest, and given small doses of potassium iodide for four weeks, at the end of which time the bony swelling had become less in extent, but somewhat denser. During the next three months the patient gradually resumed normal life and exercise, and at the end of the period was suffering no disability, although some swelling was still palpable.

Other skiagrams (figs. 3 and 4) illustrate two ossifications in the brachialis anticus, obtained from the X-ray Department of St. Thomas's Hospital, one of which was under the care of my colleague, Mr. Wallace. One ossification is of two years' standing, the other of some weeks only. The comparative density attained by the bone in the two cases is well illustrated.

The chief interest in these cases lies in the first and second, since the patients were of the same age; they came under observation at the same time, and, while one was treated by an active method, the other practically got well spontaneously. It is, however, to be remarked that the first case was of seven weeks' standing only, while the tumour in the second patient had been in existence three months. None the less, I am inclined to maintain the opinion that no active treatment should be adopted in the early stages of the condition, and I think that this view is supported by the observation here recorded, in spite of the difference in the duration of the condition in the two patients.

I regret that I am unable to attempt to shed any new light on the pathology of traumatic myositis ossificans. I think we can only assume that patients the subjects of the condition belong to the "ossific diathesis" of Virchow—that is to say, in the words of Mr. Shattock in describing a traumatic cancellous osteoma, that "they possess an idiosyncrasy for producing bone under conditions which do not avail under normal circumstances." These patients must, in fact, be placed in the same category with those in whom large masses are developed in connexion with fractures, even to the degree of such a case as the

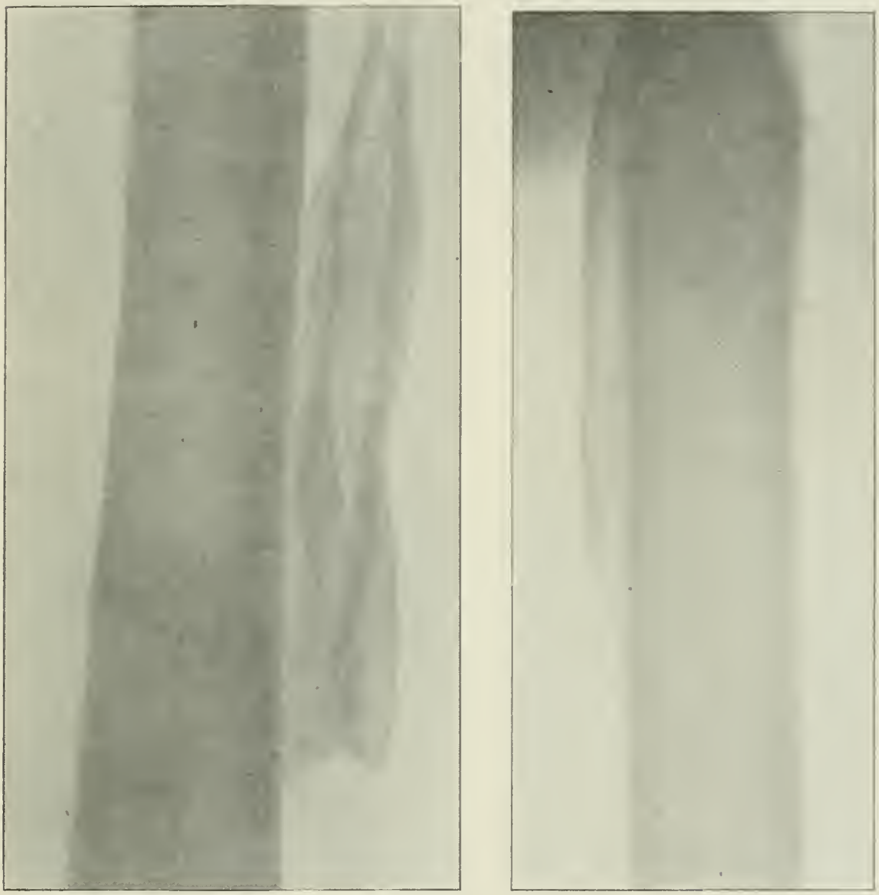


Fig. 2.

Case II, C. B., three months' standing (before operation).

Six years after operation, showing a thin plate of dense bone intimately connected with the surface of the femur.

remarkable instance recorded by Messrs. Battle and Shattock [1], in which the callus assumed the form of an actual cancellous osteoma.

The frequency with which two muscles of the body, the quadriceps extensor femoris and the brachialis anticus, are affected seems to me to support the theory that the exciting influence is to be referred to bone-forming cells detached from the periosteum. Both these muscles derive a great part of their origin from plane surfaces of bone to which the attachment of the periosteum is not so firm as at the bony ridges or eminences which afford so great a proportion of muscular attachments; hence it seems probable that the tearing free of fibres of origin of these muscles may more readily give rise to breaches of the continuity of the periosteum, which, while in themselves insignificant, may yet suffice to set free bone-forming cells from the osteogenetic layer of the periosteum in a number capable of providing the necessary genetic stimulus for the ossific process. These breaches of continuity of the periosteum may be very far short of a gross lesion, such as accompanies a fracture, and yet be sufficient for the purpose, while the subsequent apparent complete absence of continuity between the tumour and the bone is explained by the retraction of the separated fibres of muscle, together with their minute attached portions of periosteum towards the main mass of the muscle.

As to the common subsequent re-absorption of the bone, this appears to me to be a process in strict conformity with the reabsorption of superfluous masses of provisional callus in the case of fractures, the remodelling of bones to compensate for deformity due to curvatures, or the re-absorption of such cancellous osteomata as that described in the case of Battle and Shattock already referred to.

The structure of the bone is that of typical cancellous tissue, except for the absence of fine bone-marrow, not differing in any way from that developed on the surface of the long bones in cases of osteo-periostitis. To illustrate this fact I exhibit a skiagram of a femur affected with diffuse osteo-periostitis of unknown origin, in which the ossification extends into the origin of the vastus externus, while the compact tissue beneath the periosteum has been extensively absorbed. A microscopical section of the bony tissue in this instance (fig. 5), is identical in structure with the section obtained from the case of traumatic myositis ossificans (fig. 6), except in so far as the trabeculæ and intertrabecular spaces are more delicate in arrangement.

The fibrous tissue surrounding the bony masses may resemble bursal tissue, and in old-standing cases cystic cavities containing fluid

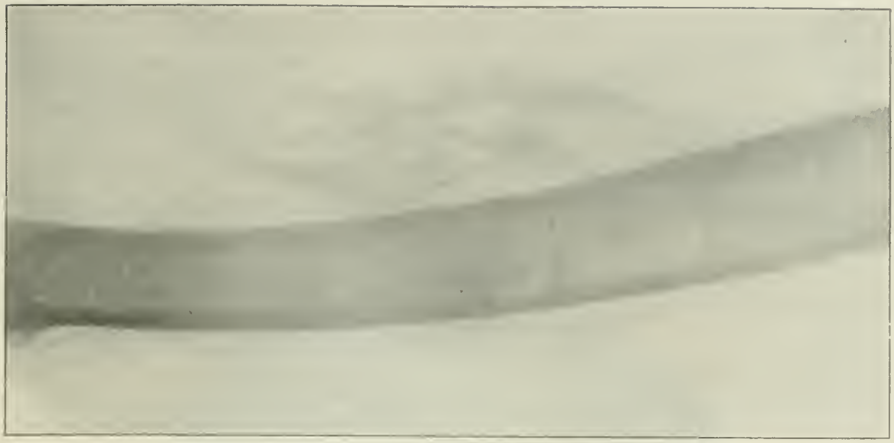


Fig. 3.

Early "ground glass" deposit in the brachialis anticus at the end of six weeks.

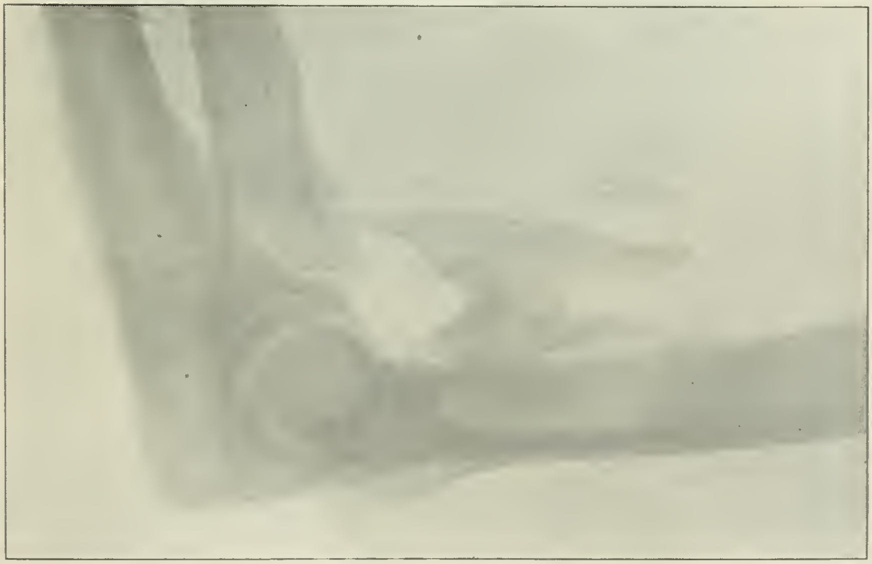


Fig. 4.

A mass of dense compact bone in the brachialis anticus at the end of two years.

resembling bursal fluid have been observed. Significance has been attributed to the development of a hæmatoma in the early stage of the condition, but the presence of blood is probably only of importance in so far as it dissects up the intimate structure of the affected muscle, and opens up the way and forms the basis for the connective tissue in which the subsequent ossification takes place.

It has been suggested by Ewald in a recent paper that the escape of synovial fluid from the knee- and elbow-joints respectively affords the stimulus for the ossific process. In support of this theory Ewald dwells upon the bursal character of the tissue surrounding the bony mass (mentioned in Case II), which has been described by many observers, and also on the synovia-like fluid filling the cystic cavities which sometimes develop. It may be pointed out that similar tissue and fluid are observed in some cases of false joint following non-union of fractures, where no rupture of a neighbouring synovial capsule exists.

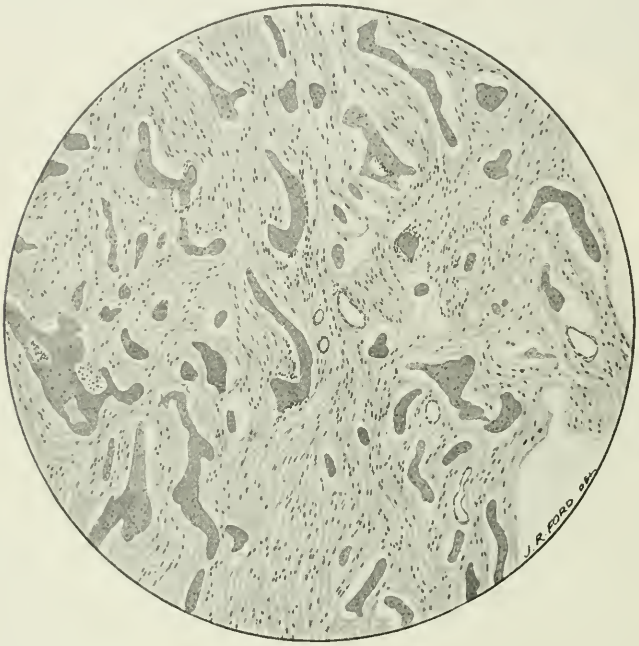


Fig. 5.

Microscopical section of cancellous tissue removed from a case of subacute periostitis.

We should separate clinically the condition of traumatic myositis ossificans from the ossifications accompanying the separation of small bony prominences, as in dislocations of the elbow, since these are much less extensive, usually confined to fibrous tissues, and in my experience more likely to be permanent. Again, traumatic myositis ossificans should be sharply distinguished from the so-called "rider's bones," which are really small permanent exostoses, and rather to be classed with the normal muscular apophyses which develop on bones at the points of attachment of powerful muscles as a definite response to the mechanical stimulus furnished by the pull of the muscles. In support of this view it may be pointed out that when the "rider's bone" attains large proportions, as in the ossification of the complete adductor magnus, the change is a permanent one.

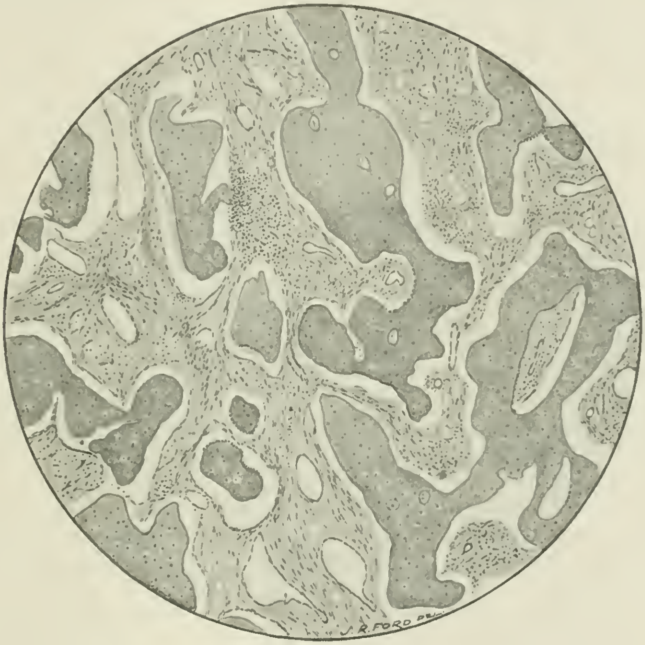


FIG. 6.

Microscopical section of the bone in Case II, showing typical cancellous tissue.

It does not appear to me to be correct to class the ossifications of muscle observed in various neuropathic diseases with traumatic myositis ossificans, since the former are permanent degenerations due to disturbance of nutrition, while the latter are rather to be regarded as connected with the normal process of repair.

Lastly, as to the general line of treatment to be adopted, a period of some weeks' complete rest should be maintained during the continuance of the active progress of ossification. When it is judged by clinical observation and X-ray examination that progress has ceased, or the process is retrogressive, massage and exercise should follow. Operative treatment should only be considered when the process has manifestly come to a definite standstill, and the patient suffers from functional disability which there is a chance of relieving.

REFERENCES.

[1] BATTLE and SHATTOCK. *Proc. Roy. Soc. Med.*, 1908, i (Path. Sect.), p. 83.
[2] EWALD. *Deutsch. Zeitschr. f. Chir.*, Leipz., 1910, cvii, p. 310.
[3] SCHULTZ. *Deutsch. militärärztl. Zeitschr.*, 1910, Heft 4.

The Formation of Bone in Periosteum separated by Injury.

By ANTHONY A. BOWLBY, C.M.G., F.R.C.S.

THE condition to which I wish to direct attention is well illustrated by the two following cases:—

Case I.—A young man, aged 18, was thrown from his horse on April 20 of last year and injured his arm, the pain being felt on the flexor aspect just above the elbow-joint. As it was evident that he could not have struck this region it appeared probable that the injury was caused by a muscular wrench when he fell on his outstretched hands. He was seen the next day by Dr. Bryce-Collyer, of Croydon, who found that there was a good deal of swelling and pain, but the X-rays confirmed the fact that there was no fracture. During the next fortnight the swelling rather increased, and the patient was not able to use the arm, while both flexion and extension became more limited. On May 26 another radiogram showed that there was an irregular piece of bone in the region of the swelling, and on May 30 I saw the patient and found the following condition: Situated on the front of the humerus, under the biceps tendon, was an irregularly rounded swelling the size of a hen's egg. It was very firm, was more or less fixed to the bone, but the muscle over it was free. It limited the movements of flexion and extension, and when the forearm was as nearly completely extended as possible it could be seen as a very definite swelling. An X-ray picture showed an irregular formation of bone an inch in front of the humerus over an area of about 1¼ in. in diameter. The arm was placed in a splint, and Scott's ointment was applied, with the result that in six weeks the swelling had almost disappeared, while in three months more no sign of it could be found, and the movements of the arm were quite restored.

Case II.—A young lady, aged 21, fell while tobogganing on April 15, and injured her elbow. A surgeon found that she had sustained a dislocation of this joint, and this was reduced under an anæsthetic, and the arm kept in a splint for one week, and in a sling for a week more. It was then found that there was a swelling on the front of the humerus, just above the elbow-joint, and this swelling continued to increase till I saw her six weeks after the accident. I found a swelling exactly like that described in Case I, and, like it, about the size of a hen's egg, situated beneath the muscles, and attached to the bone. It caused aching pain, and prevented free use of the arm. The X-rays showed an irregular bony lump, separated from the outline of the humerus by about an inch, while the shafts of the bones themselves were normal and uninjured. Here also rest on a splint and a mercurial ointment resulted in the complete disappearance of the swelling within three months, and the restoration of movements in this joint.

The formation of a considerable swelling as a result of the separation of periosteum by the muscles is not a common occurrence, although as the result of direct blows, such as kicks on the shin, it is frequently seen. But I believe that there are really a great many cases in which pain and impairment of the use of a limb is caused by the dragging of the periosteum off the bone where large muscles are inserted into it, and by the hæmorrhage which results therefrom, and from the formation of new bone. In most of these cases the swelling is slight and is often hardly noticed, but pain may persist for a long time unless the muscles are kept at rest. If there has been any old periosteitis which has left the membrane hypervascular and has loosened its attachment, separation is more easily caused, and I have seen in one patient, who had previously had acute osteitis and necrosis of a small piece of the humerus, several attacks of pain and swelling of the bone brought on by any violent use of the muscles attached to the upper part of the affected bone. The patient was liable to these attacks for several years, and they caused the arm to swell to at least double its natural size, but they only occurred as a result of some considerable muscular strain, and slowly subsided when the arm was kept absolutely at rest. In their less pronounced form I have seen cases of pain and tenderness with some swelling, which I have attributed to a separated periosteum, chiefly in the humerus and the femur, but the only cases where X-ray pictures have been taken to confirm this diagnosis are the two I have detailed this evening. In one of them the swelling was so considerable that it had been diagnosed as a periosteal sarcoma.

Myositis Ossificans Traumatica.

By Rickman John Godlee, M.S.

More than three years ago, on December 6, 1907, I saw a youth, aged 18, who a month before, on November 9, had been kicked on the front of the thigh at football. He continued to play for three minutes, though in bad pain. Swelling occurred the same day, and bruising came out later. He kept his bed for one day, then was about for two days, after which he rested for ten days, whilst the limb was fomented and afterwards painted with tincture of iodine. He was then allowed to walk a little, and massage was applied.

When I saw him there was apparent a very large oval swelling on the front of the lower half of the thigh. It was symmetrical and very hard, except just above the patella, where it fluctuated, and there was only slight pain on movement. I told him to leave it alone, and he returned to Oxford, where he did ordinary walking. In February, 1908, he tried football three times, but had to give it up on account of pain and stiffness. Otherwise it was all right till February 26, when it suddenly became stiff and hard again, and when I saw him on February 28 it was in appearance much as when I saw him in December, except that it was now hard all over. It was perhaps slightly smaller, but still measured 5 in. from above downwards and reached $1\frac{3}{4}$ in. above the patella. It was obviously fixed to the bone, and I then had the skiagram taken which I now produce (fig. 1). It shows a large dark shadow corresponding to the tumour, which it will be seen is even larger behind than in front, and there is a still more defined shadow of laminæ of what appears to be bone on the front of the femur. For the most part these are parallel with the shaft, but tail off at the upper part into layers which slant obliquely towards the bone.

On March 8 I saw him again with the late Mr. Clutton, who supplied three or four skiagrams of similar cases, and told me of two which had been operated upon (one by himself) in which the operation distinctly did harm. I was therefore fortified in my opinion to leave him alone, and after six months he had no further trouble.

His next visit was in March, 1910. The lump had almost gone. But another skiagram, which I also present (fig. 2), shows that there was then a distinct flake of bone on the front of the femur, fading off above into the surface of the shaft, but ending below in a sharp spicule. He now pays no further attention to the thing at all.

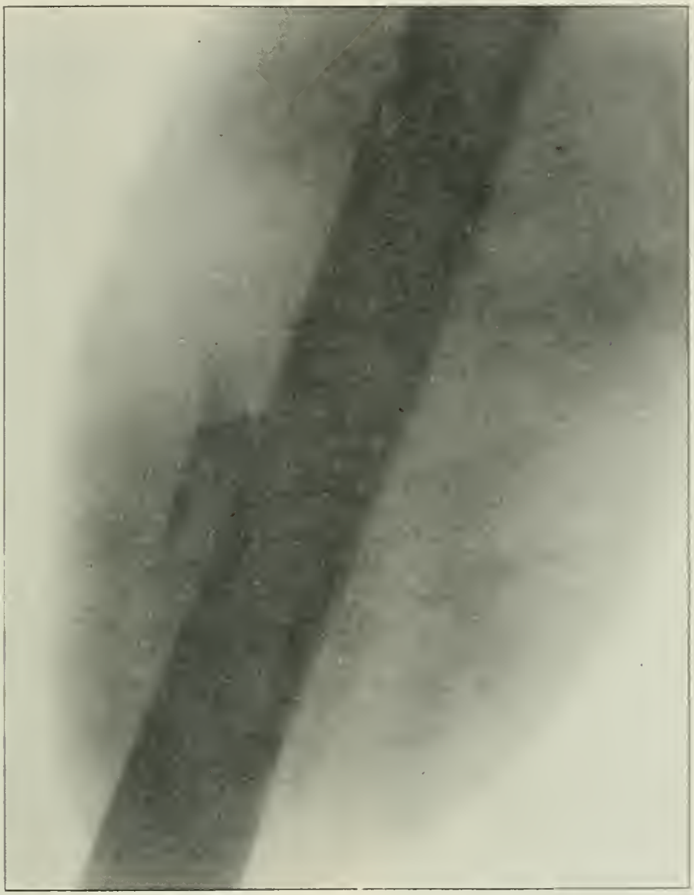


FIG. 1.

One object of bringing this case before the Section is to point out that a large swelling may, without treatment, diminish so much in size as almost to disappear and cause no further trouble or inconvenience. It should be remembered that the shadow in the earlier skiagram does not necessarily show the amount of the bony deposit at that time, as it would probably be, like early callus, transparent to the X-rays. This being the case, and the experience at all events of some surgeons having been that removal has actually done harm, I would urge the advisability of leaving these swellings alone until ample time has been allowed, at least a year, for the absorption of what may be called provisional callus. Even after this time I think that removal would only be justified if the mass were causing mechanical inconvenience and pain. It must be remembered that the operation is inflicting another traumatism upon a part which, for some reason, has shown a special tendency to the development of bone, and it cannot therefore be surprising if renewed activity of the process should follow.

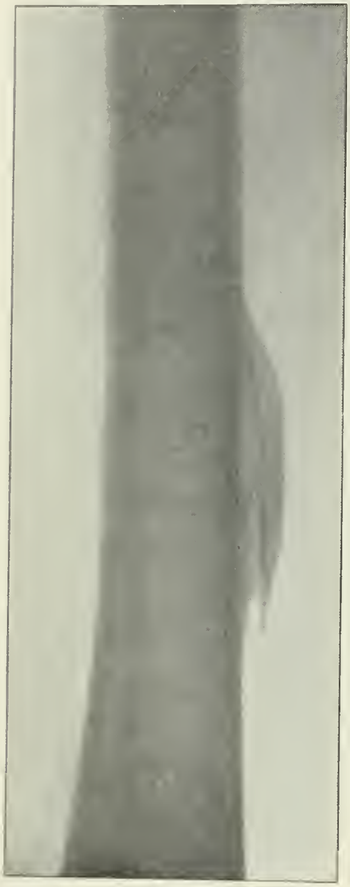


Fig. 2.

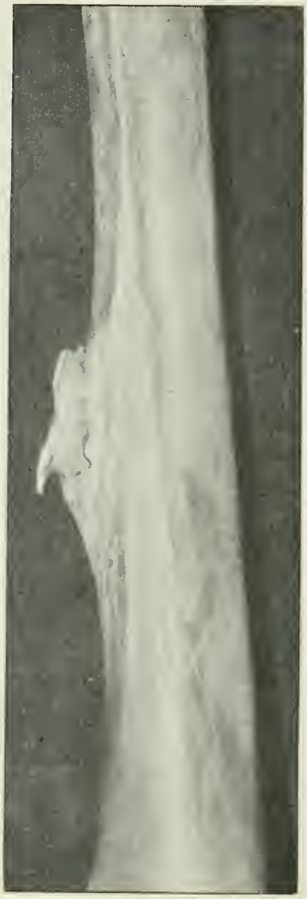


Fig. 3.

To support this proposition I have searched some of our museums for specimens of bone showing the result of such injuries. But I find that they are not numerous. By a curious coincidence, whilst I was being told that we have nothing of the sort in the anatomical museum at University College, the beadle brought in this fine example (fig. 3) which he wished to exchange for a normal femur. It will be seen that it occupies a part of the bone from which both vastus externus and crureus arise. It is sharply outlined, suggesting that the periosteum had been stripped up over a limited area. Its surface is finely granular and

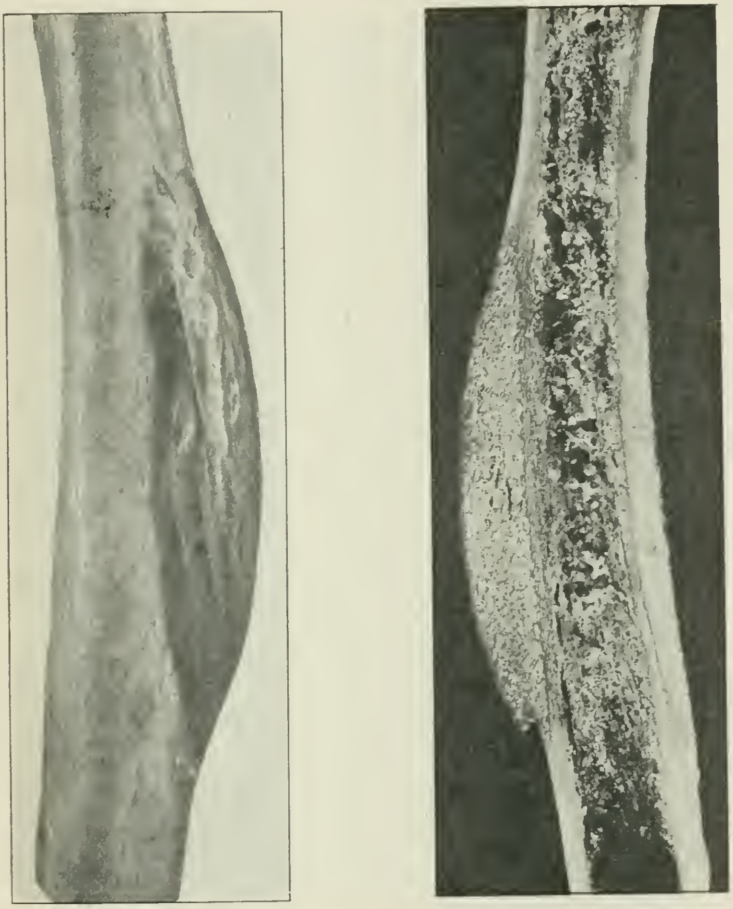


FIG. 4. FIG. 5.

No. 1611, Museum of the Royal College of Surgeons.

rough, contrasting with that of the vertically ribbed highly polished normal bone. It ends, like all the specimens, in a sharp point below. There are two similar specimens at University College Hospital Medical School, in which the flake of bone is not so large and the surrounding area of granular bone is less extensive.

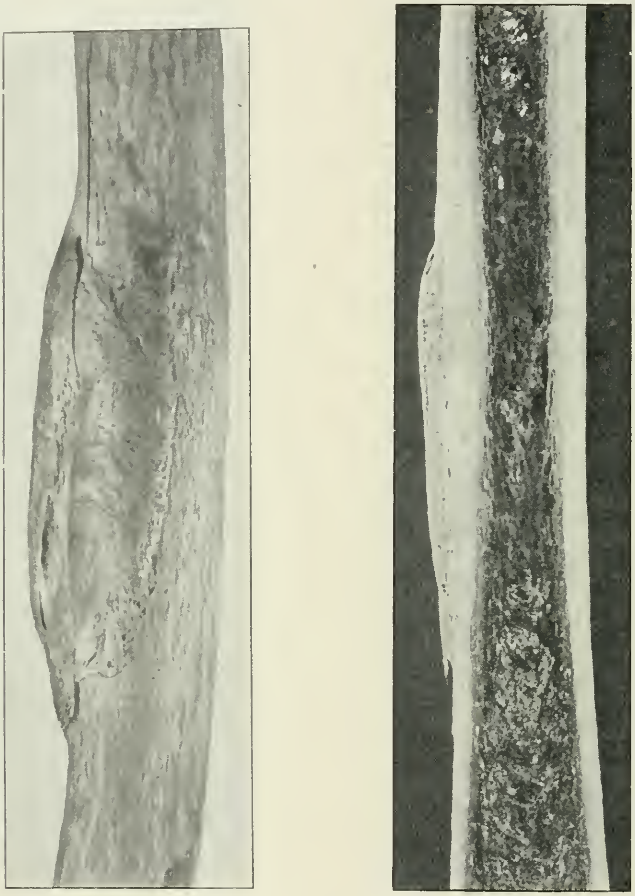


Fig. 6. Fig. 7.

No. 1613, Museum of the Royal College of Surgeons.

In the Museum of the Royal College of Surgeons there are two femurs (No. 1611 and No. 1613, both Hunterian, figs. 4, 5, 6, 7) showing similar plates of bone. In that of St. Thomas's Hospital is one in the crureus, and at St. Bartholomew's Hospital are three admirable specimens which, by the kindness of the authorities, I am permitted to show, and of which I produce photographs (figs. 8, 9, 10, 11, 12). Two of them are much like my own specimens, but one (fig. 12) is particularly interesting because it corresponds almost exactly with one removed by Mr. P. M. Heath, which is now in the Museum of the University College Hospital Medical School. This case was described by Mr. Heath.[1] It shows a large mass of bone, not smooth like most of the others, but raised into deep rounded vertical ridges, looking, it must be owned, like a

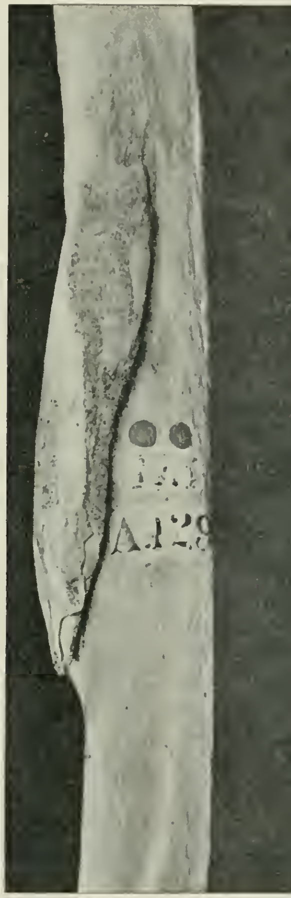


FIG. 8.

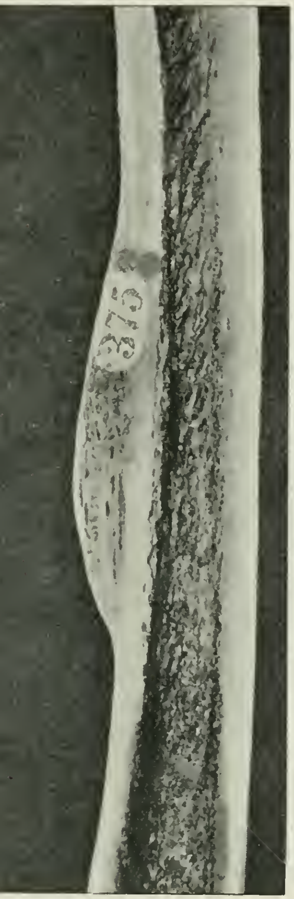


FIG. 9.

---

[1] *Proc. Roy. Soc. Med.*, 1910, iii (Clin. Sect.), p. 149.

deposit in the muscular fibres of the vastus or the crureus. I suggest this is only an earlier condition of that which the other specimens show, and that in time it would have been in great part absorbed, and have left the smooth, rounded swelling to which I have drawn attention. We have had a transverse section made of Mr. Heath's specimen, and of this I show a microscopical section. It will be seen that the mass of bone is hollow. On its surface there is muscular tissue undergoing hyaline degeneration, the interstitial tissue of which is increased, and amongst the layers of which is blood pigment. Between this hollow plate of bone is a thick mass of grey gelatinous material, rather like myxomatous tissue,

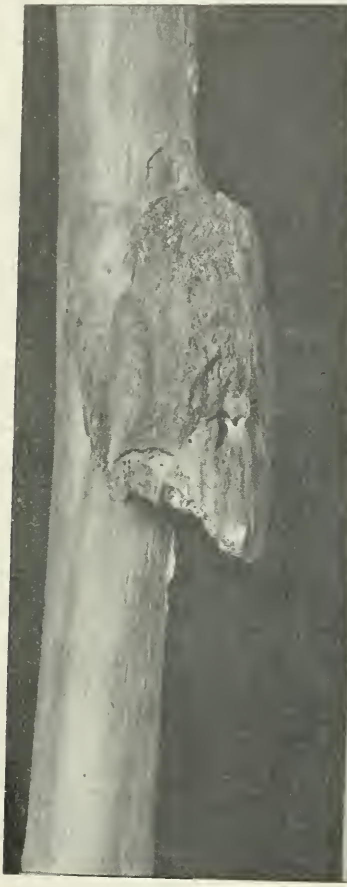


Fig. 10.

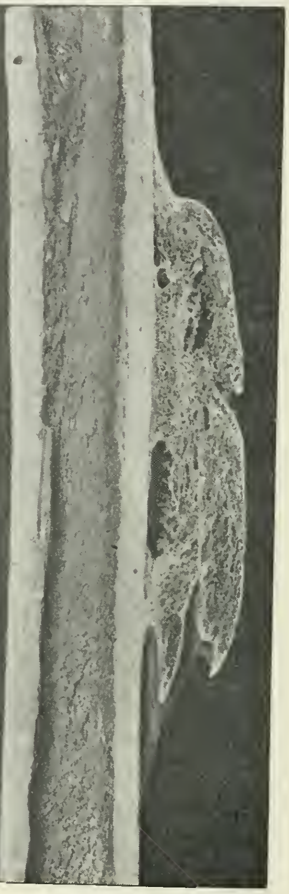


Fig. 11.

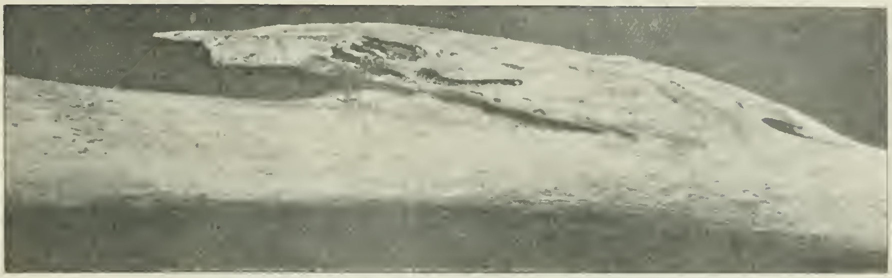


FIG. 12.

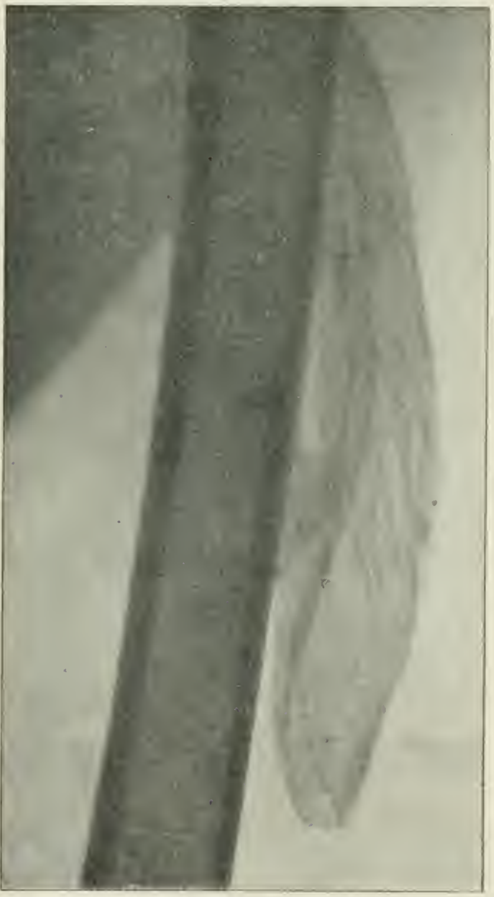


FIG. 13.

Mr. Heath's case before operation.

containing vessels scattered about in which are a few isolated groups of atrophied muscular fibres. These points are illustrated in figs. 13, 14, 15, and Mr. Heath has most kindly supplied two skiagrams of his case, one taken before operation and one afterwards, to which he refers in the discussion which follows (p. 156).

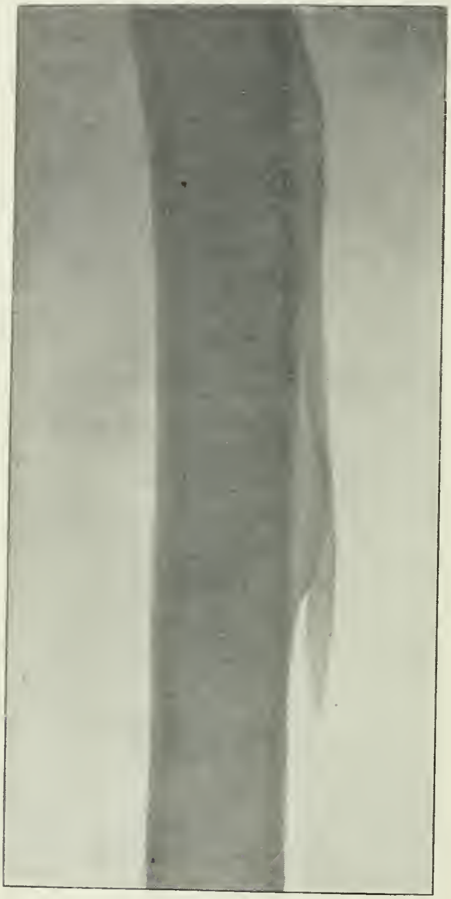


Fig. 14.

Mr. Heath's case after operation.

Naturally these cases would not come under the eye of the pathologist, and in the dissecting room would not excite much interest to the anatomist. It is therefore interesting that Professor G. Elliot Smith tells me he did not come across any examples amongst the large number of prehistoric bones he examined in Nubia, and that none were furnished by the great haul of bones from the East End plague pits which found their way to University College.

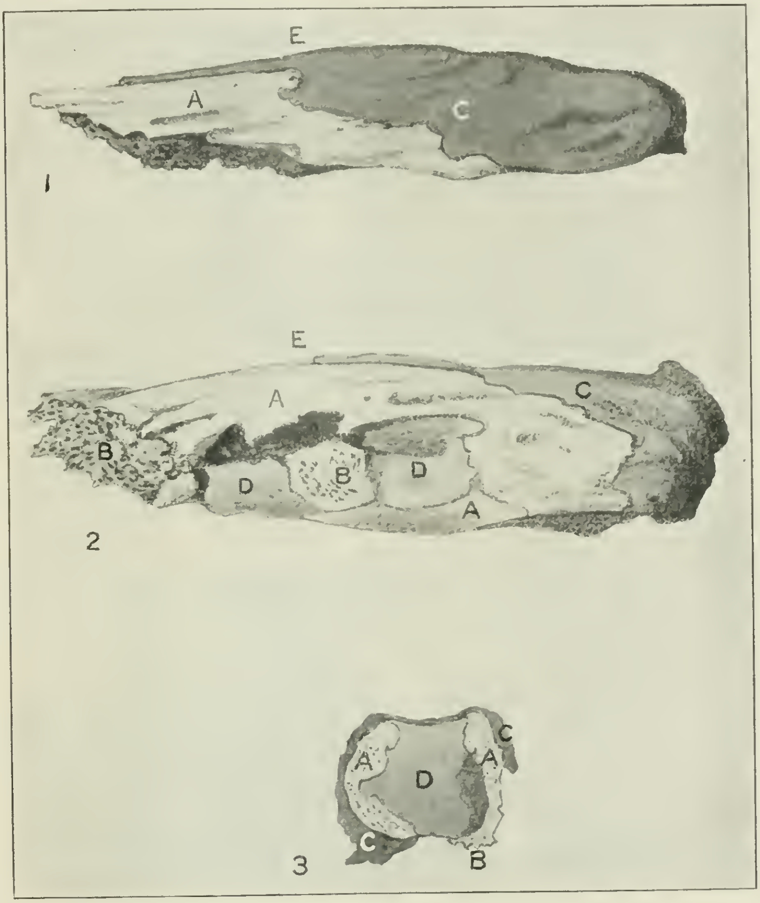


Fig. 15.

Mr. Heath's case; the part removed. **1**, from the outer side; **2**, from behind. **3**, transverse section at **E**. **A**, bone; **B**, bone where divided; **C**, muscle; **D**, gelatinous material between new bone and shaft of femur.

So far I have only referred to injuries to the femur, but a large number of cases of a similar process occurring in the neighbourhood of the elbow have been described, some in connexion with fractures and others with simple dislocations. I show as a typical illustration a skiagram (fig. 16) of the elbow of a boy, aged 6, who broke his humerus in 1903. It was refractured on account of stiffness seven weeks later, but soon was as stiff as ever. Six months after the accident there was a bridge of bone uniting the front of the humerus to the coronoid process, and absolutely locking the joint. This would be called a deposit in the brachialis anticus. I restored the joint to a fair degree of utility by removing it, but a later skiagram (fig. 17) shows a considerable amount of fresh bony deposit after my operation.

A most elaborate paper on the subject by Mr. Robert Jones[1] records seventeen cases of his own and over 140 by Cahier and Binnie. At that time Jones concluded that operative treatment was essential. It is therefore interesting that he should now write to me, under date of February 28, 1911: "Since writing the article I have come upon cases where the deposits, instead of increasing, have decreased, and I am not now at all convinced of the value of operation. The simplest looking mass in the bend of the elbow is a very difficult problem to negotiate operatively, and I have on more than one occasion wished I had left the whole thing alone."

Still it must be owned that in some of the elbow cases with complete locking, operation appears to be essential, and also that in some of the early operations on cases in the thigh, even if they were not, as I think, necessary, no harm has resulted. Such, for example, was the result in Mr. Heath's case, and in one other to which I must refer, because a careful examination was made of the parts removed.

Sir Frederic Eve described a case[2] apparently very much like Mr. Heath's, in which the bone was removed eleven weeks after the injury with good result. A microscopical examination showed atrophy or degeneration or disappearance of the muscle in the neighbourhood. But it is not clear that any sign of muscle was found in the thin layer of soft tissue which intervened between the bony mass and the femur. The author agrees that injury to the periosteum is an essential part of the condition.

Lastly, I would ask whether this condition should be called myositis, and what is the evidence that it is due to inflammation of muscle. To me it seems more probable that the essential things are, first, an injury to the periosteum, and, secondly, an effusion of blood, and that the injury to muscle and the inclusion of muscular fibres in the callus are, so to

---

[1] *Arch. Roentg. Ray*, 1905-6, x, pp. 10, 46, 72.

[2] *Clin. Soc. Trans.*, xxxii, p. 233.

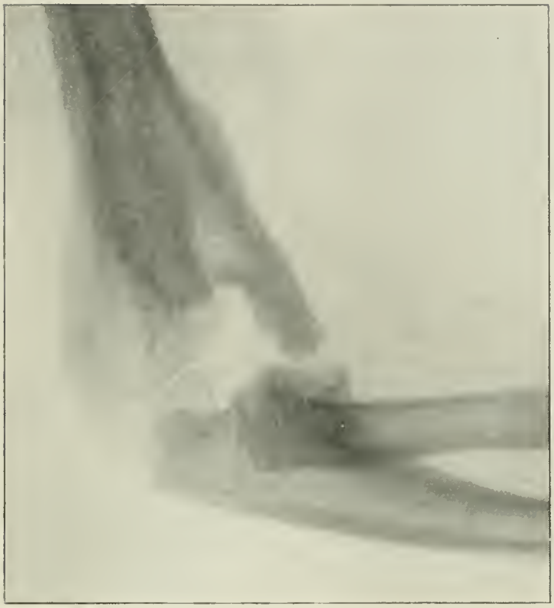


Fig. 16.

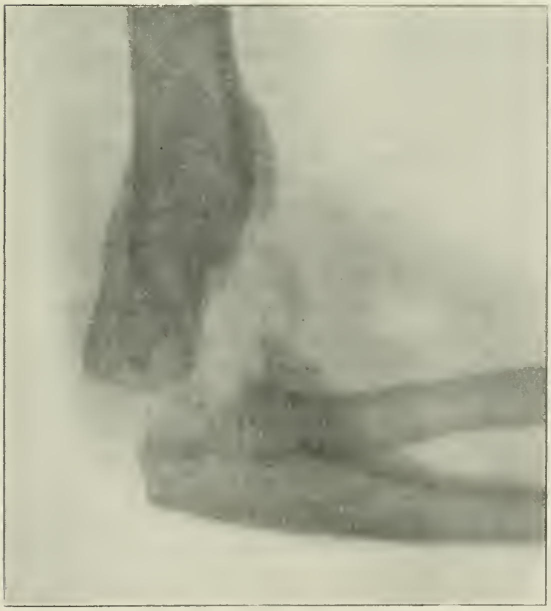


Fig. 17.]

speak, accidental, though it no doubt governs the shape and position of the new mass of bone. In other words, I think it is more analogous to what occurs when periosteum is stripped off in such operations as excision of the elbow, or in the cephalhæmatomata of infants, in which it is easy to demonstrate true bone at the margins of the clot, or to the formation of provisional callus in simple fractures. I know it is suggested that the process is the same as that which causes the "rider's bone" or the "exercise bone," but it appears to me to be a very different one. In these there is no evidence of injury to the periosteum, unless it be suggested that they are to be compared with the cases described by Sir James Paget where necrosis occurred at the insertion of a strained muscle. The strongest argument for the myositis theory is afforded by some recorded cases where the new bone was not in actual contact with that which had received the injury.

If the essential factor, then, is injury to the periosteum, it is surely a pity that the name which has been applied to this condition should confuse our ideas of myositis, and especially of myositis ossificans, about which, it must be allowed, our present notions are quite vague enough.

DISCUSSION.

Mr. MAYNARD HEATH said he showed, before the Clinical Section, on March 11, 1910, the patient from whom the more recent specimen shown by Mr. Godlee was removed.[1] The man was a horsekeeper by occupation, and the piece of bone was removed because the limitation of flexion in the knee-joint was so great that he could not do any work. He exhibited a skiagram of the condition. The man returned to his work in four months, and he had been free from trouble ever since. He saw the man yesterday for the first time since, and he had another skiagram taken. It showed a small scale of bone, replacing that which had been removed. He did not think that any of the new bone had been left behind, because the original piece of bone was attached to the femur by two pedicles, and it was easy to cut them through and remove the whole mass. The scale of bone shown in the latest skiagram had therefore developed since the operation, but it did not interfere with the movements of the knee. The man was satisfied with the result, and he (Mr. Heath) thought the operation was justified. Here there was a return to work without disability in four months, and in some of the recorded cases the interval had been much longer. The injury occurred in November, 1909. The first skiagram was taken on February 28, 1910. The piece of bone was removed on March 17, 1910. The second skiagram was taken on March 13, 1911.

---

[1] *Proceedings*, 1910, iii (Clin. Sect.), p. 149.

Mr. CARLING said he had had two cases which illustrated one or two of the points which Mr. Makins referred to. One was a typical case of the condition, traumatic myositis ossificans, in the middle of the shaft of the femur, resulting from a blow. An interval of six months had elapsed since he saw the man, which was two months after the injury, and it was now disappearing, and with it the limitation of movement in the knee. The other case, if it could be placed in the same class, was unusual. It was that of a young man who fell over a precipice in Switzerland, and probably sustained a fracture of his pelvis. Two months later he was found to have a tumour in his glutei, which, on X-ray examination, proved to be closely associated with the ilium. He thought that illustrated Mr. Makins's point, that many of these instances were closely allied to cases of exuberant callus. There was another point which he thought might be made, that whilst there were differences between these cases and those in which the affection developed in tendons, as a result of exercise, repeated stresses, &c., one saw occasionally cases in which both forms were combined. Indeed cases were sometimes exhibited as true myositis ossificans which belonged more properly to the development of bone in muscle as the result of injury, and especially of repeated injury. Last year a case was exhibited at the Clinical Section which seemed to fall into that category; there were masses of bone developed in the fascial planes around the joints in the tendons, and some in the muscles of the back, which simulated typical myositis ossificans. Possibly the name "muscular osteomata" would meet the objection raised by the President as to nomenclature: it had been adopted by French surgeons for such masses, and separated them from the cases which arose independently of injury.

Mr. WHITELOCKE said there were two points of special interest in the discussion, which were referred to by Mr. Bowlby. The first was as to the possibility of those affections occurring as the result of strain or sprain. He had had experience of them amongst athletes at Oxford, who sometimes brought the condition about suddenly, and at other times after repeated exercise, such as in punting, in which the strain was sustained especially by the muscles of the calf and of the thigh. He had seen two cases in which it occurred in connexion with the soleus muscle, at its origin from the oblique line of the tibia. That situation had not been mentioned that evening, and he had not noticed it referred to elsewhere. He had had two cases of it, in one of which he was tempted to operate on account of extensive swelling and the pain which resulted. The patient was a young lady, who did it in punt-racing. Her first accident caused some swelling, and after recovery from that she continued to carry on the same form of exercise. On the second occasion, some months later, there was a large swelling, a severe hæmatoma, and pain. Six weeks after that a radiograph showed evidences of spicules of bone, and he determined to cut down on the spot. He found a series of spicules passing from the oblique line into the soleus muscle, one of which penetrated into the gastrocnemius. He removed as many of the pieces of bone as he could find, but there were still a few small spiculæ left which had evidently developed since the operation, passing into the soleus muscle. It was now nearly two years since it was done, and occasionally she had pain. He thought the periosteum had been damaged, because when handling it in a recent state he could feel grits of bone all along the origin of the muscle. His opinion was that the condition occurred more frequently as a result of sprain than was usually thought. The other point of interest was as regards Mr. Bowlby's advice as to treatment. He agreed with him entirely as to the policy of rest in the early stages. Vigorous and prolonged massage had been productive of harm in more than one case to his knowledge.

Mr. MAKINS, in reply, said he thought the President's comments in regard to the name "myositis ossificans" were well justified. In the microscopic section which he (Mr. Makins) showed there was absolutely no trace of bone to be found in the muscle; the mass of bone was surrounded by the soft connective tissue, which might be the successor of the blood-clot; at any rate, the tissue occurred where the blood-clot was, and the marginating muscle was not invaded.

Mr. BOWLBY, in reply, said he agreed that the condition had nothing to do with myositis; it was connected with injury of the periosteum. He believed there would not be the formation of bone in most cases if it were not for the vigorous treatment they often received in these days of massage and forced movements. He thought it probable that in one of his cases the formation of bone was entirely the result of that.

The PRESIDENT (Mr. Rickman J. Godlee), in reply to Mr. Bowlby, reminded that gentleman of the case of the little boy he had mentioned, with a fractured elbow, who was put into splints for four weeks, and there was so much stiffness that re-breaking was necessary.

Large Congenital Diverticulum of the Bladder treated by Resection.

By JAMES BERRY, F.R.C.S.

C. B., AGED 21, a labourer, was admitted to the Royal Free Hospital on November 18, 1904, complaining of recent hæmaturia. Three years before admission the patient first noticed a lump in the lower part of the right abdomen; it came and went several times, disappearing after urine had been passed. Three weeks before admission he was playing football and thinks he strained himself. Two days later he first noticed blood in the urine, for which he consulted Dr. Pegg, of Reigate. The hæmaturia lasted two or three days and then stopped. Dr. Pegg examined him carefully and thoroughly, and not being satisfied as to the cause of the hæmaturia sent him first to the Reigate Hospital and then to the Royal Free Hospital, where he was admitted under my care.

He was a pale but otherwise healthy-looking youth. When examined under an anæsthetic, a large rounded, soft swelling was found in the right iliac fossa and extending thence up into the right loin. Although he had passed water shortly before the examination, nevertheless as much as 37 oz. of urine were withdrawn by catheter. A sound passed into the bladder deviated markedly to the right. The urine had a specific gravity of 1020, was very slightly alkaline, and contained a good many pus cells and some oxalate of lime and triple phosphate crystals. The patient also informed us that his mother had told him that when he was a very small boy he used to hold his water for a very long time, even a day or two, and then pass a very large quantity.

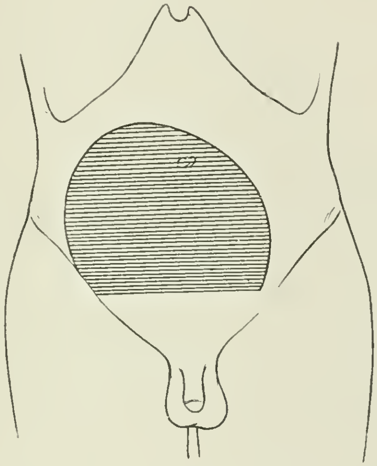


FIG. 1.

Abdominal tumour (bladder and pouch) distended. Before operation.

The diagnosis at this time was slight cystitis, probably connected with a pouched or dilated condition of the bladder of uncertain origin. He was sent back to Reigate and instructed to draw his water off with a catheter three or four times daily, and to report himself again in three months' time. During this time Dr. Pegg looked after him, and the water was drawn off regularly.

On February 28, 1905, he was readmitted to the hospital and his condition was found to be much the same as before. On March 4 he was again examined under an anæsthetic; 17 oz. of residual urine were withdrawn by catheter. Three pints of boracic lotion were injected into the bladder and the abdominal tumour reproduced to its full extent. It was then found to extend upwards in the middle line as far as 1 in. above the umbilicus and on the right side to within 2½ in. of the costal margin. Transversely it extended to within 1 in. and 2 in. respectively of the right and left anterior superior iliac spines (fig. 1). A silver catheter having been passed into the bladder, a rectal examination was made and the prostate found to be much higher up than normal. It was unusually small and much flattened. A cystoscope was then passed, and on its entrance into the bladder the same marked deviation to the right that had been observed on the previous occasion was again noticed. High up on the left side of the anterior surface of the bladder a large dark oval aperture in the bladder wall was seen. Only a part of the circumference of the opening could be seen; its margin was smooth and well defined. A careful search failed to reveal more than one ureteric orifice.

It seemed clear that the patient was the subject of a large diverticulum which was seldom, if ever, completely emptied. The symptoms thus seemed to be fully accounted for. As the patient was young and otherwise healthy, and as drainage of the bladder by catheter had been given a fair trial without success, it was decided to do an exploratory operation with the view of removing the pouch, if possible.

On March 5, 1905, a long median incision was made through the lower abdominal wall and the bladder freely exposed. The peritoneum was high up, and was not interfered with, the operation being wholly extraperitoneal. It was at once seen that the bladder consisted of two nearly equal parts, each about as large as a normal bladder. The lower and anterior one occupied most of the true pelvis, and had a tough fibrous wall in which very little muscular tissue could be seen. The posterior sac had a very thick muscular wall, and lay above and behind the other, constituting the tumour that had been felt before the operation. The anterior sac was then freely opened, and 41 oz. of fluid withdrawn from it. The opening between the two sacs was then clearly seen. It was as large as a five-shilling piece, and admitted five fingers easily. Posteriorly the sac had a somewhat conical projection, which passed down on the left side of the rectum, and was rather firmly attached to the coccyx. Removal of the sac, chiefly by blunt dissection, was then begun. When the posterior surface had been reached it was found that the left ureter, dilated to the size of a little finger, opened into this sac on its posterior surface. The original plan of removing the whole sac was therefore modified. A more or less triangular portion of the wall, including the orifice of the ureter and the large opening into the main portion of the bladder (fig. 2) was left in situ, and all the rest of the sac, shown in this jar (fig. 3), was cut away. The triangle of sac wall was then sewn up with a continuous catgut suture, so as to form a conical tube, $2\frac{1}{2}$ in. long, leading from ureter to bladder. As the whole of the sac and the lower end of the ureter had been freely separated from their posterior connexions and drawn well forwards, the suture presented no special difficulty. The only really troublesome part of the operation was the separation of the sac from the coccyx, because

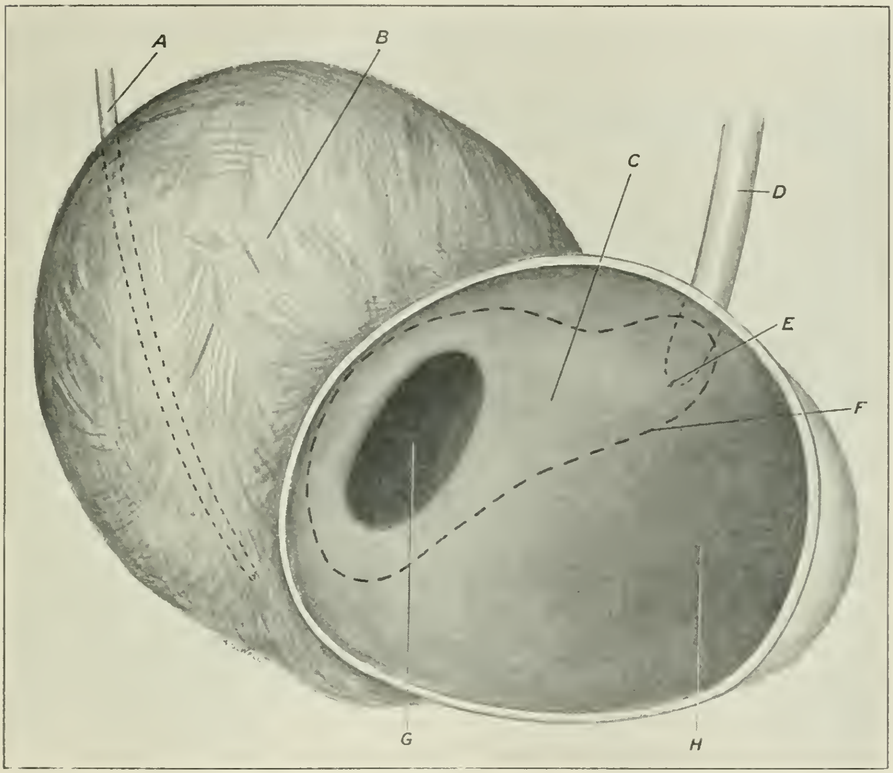


Fig. 2.

Front view of bladder and pouch. **A**, presumed position of right ureter; **B**, bladder; **C**, area of pouch wall, not removed; **D**, dilated left ureter; **E**, orifice of left ureter; **F**, line of incision; **G**, opening between bladder and pouch; **H**, interior of the pouch.

of the depth at which the parts lay. Hæmorrhage was but slight, and caused no trouble whatever. The abdominal wound was left widely open, with two large rubber drainage tubes passing into it. A large rubber drain was also inserted into the bladder through a median perineal incision.

The patient made an excellent and uneventful recovery. The temperature remained practically normal throughout, the highest being 100·2° F. on the day after the operation. No extravasation or leakage took place, and the wound healed aseptically. On the sixth day after the operation the granulating abdominal wound was closed by secondary suture of fishing gut. On the fifteenth day the perineal tube was discontinued, but for a few days longer the urine was drawn off by a catheter passed through the perineal wound. Three weeks after the operation the patient returned home to the care of Dr. Pegg. On June 7, six weeks after the operation, he came up to the hospital to report himself. He was in good health, and the bladder was found to contain only 4 oz. of residual urine. On October 30, 1906, he was seen again, and found to be in excellent health, and able to do his work as a labourer. I have not seen him again until to-day, when he has come up from the country, that you may see him and judge for yourselves of his condition.

He now tells me that he is in excellent health and has no urinary trouble at all. In the daytime he micturates every three or four hours, and he very rarely gets up at night to do so.

I venture to record this case on account of its rarity, as I have not been able to find a precisely similar one recorded elsewhere. Mr. John Pardoe has kindly drawn my attention to an excellent paper by Dr. Hugh H. Young, of Baltimore,[1] on "The Operative Treatment of Vesical Diverticula, with Report of Four Cases." A bibliography of the literature is given, together with a short account of the cases of excision of a diverticulum by Péan, Czerny, Riedel, Pagenstecher, and von Eiselsberg, besides his own three cases in which this method of treatment was adopted.

The chief points of interest in my case seem to be:—

(1) The large size of the cyst.

(2) Its congenital nature, shown by the existence of urinary symptoms in early childhood, the absence of any acquired obstruction in the urethra which would have led to the formation of such a pouch, and the opening of the left ureter into the pouch far away from the bladder.

---

[1] *Johns Hopkins Hosp. Report*, Balt., 1906, xiii, p. 401.

(3) The dilatation of the left ureter, which was not due to any stricture at its vesical orifice, since a probe could be (and was) readily passed into the ureter.

It was apparently due to the absence of muscular fibre in the wall of the pouch into which the ureter opened, thus permitting the intravesical pressure during micturition to be transmitted directly to the ureter.

The operation itself presented no difficulty of any importance, and does not seem to require further comment.

Centimetre scale.

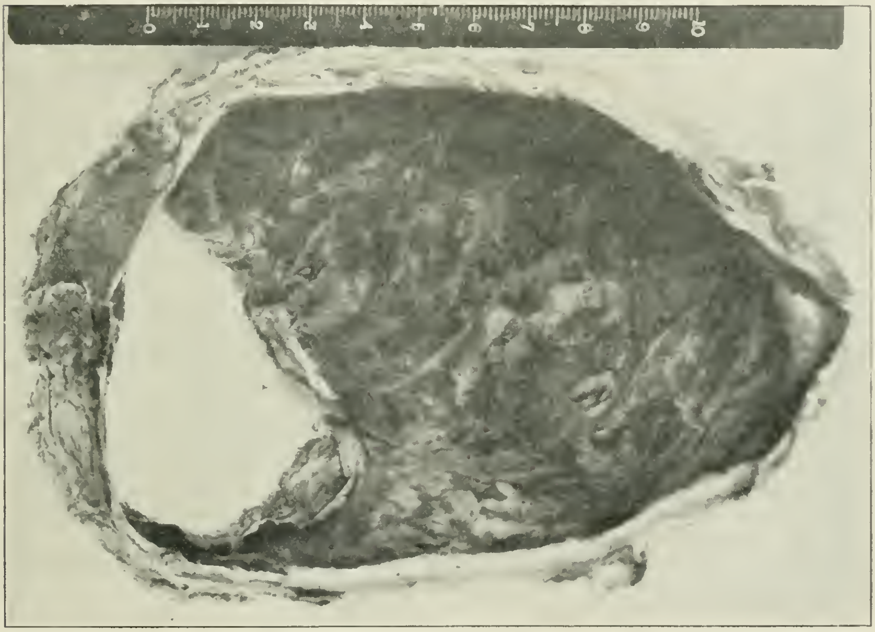


Fig. 3.

Photograph of the pouch after removal: the front wall has been cut away. The interior was smooth, the dark areas being due more to redness of the mucous membrane than to shadow. (Reduced $\frac{3}{4}$.) Royal Free Hospital Museum. Series xxv, 5*a*.

DISCUSSION.

Mr. Swift Joly said he had a case under his care suffering from a somewhat similar condition, the specimen of which he showed. It was a case of total extirpation of the sacculus.

Patient, a male, aged 51, messenger, was admitted into St. Peter's Hospital on January 15, 1910, complaining of difficulty of micturition. He states that as long as he can remember he had a weak stream, and always took longer to pass his water than other people. In

1901 he had pain along the left groin, for this he consulted a doctor, who passed a catheter. Up till this time his urine had been perfectly clear, but about a week after this consultation it became turbid and he had scalding during the act of micturition. Since then he has been constantly under treatment for cystitis, which, however, has steadily got worse.

Present condition: Patient has to pass water every hour by day and night. Difficulty in micturition, stream slow and weak, straining. Scalding pain in penis and perineum during the act. Never had retention. Patient has had five attacks of hæmaturia during the last eighteen months, has passed clots irregular in size and shape. No stricture. Prostate normal in size, soft and elastic. Above the prostate the bladder can be felt bulging into the rectum, soft and not indurated. Urine: Residual, ℥xiii; total quantity passed in twenty-four hours, ℥xxxv; acid; albumin present, no sugar; urea, 1 per cent.; deposit of pus and blood-cells. Cystoscopy: It was only with great difficulty that the bladder could be washed clear; distension, ℥xxiv; on introducing the cystoscope there appeared to be only about ℥x or ℥xii of lotion in the bladder. General cystitis. No intravesical projection of the prostate. The right ureter is displaced rather over to the right, the left is nearly in the middle line. Above and to the left of the left ureteric orifice is the opening of a large sacculus; this appeared to be large enough to admit a cedar pencil. Surrounding this opening, except at its lower part, was a polypoid mass of very œdematous mucous membrane. The left ureter protrudes into the bladder, and, when an efflux occurs, its lower end can be seen to be dilated. Abdomen: The bladder can be felt, even when empty, as a tumour rising just above the pubis under the right rectus. The displacement to the right becomes more marked when it is distended.

Operation, January 19, 1911: A vertical median suprapubic incision was first made, later the left rectus was divided transversely 1½ in. above the pubes. The peritoneum was stripped back from the left side of the bladder and from the sacculus, which was found to fill almost completely the left side of the pelvis. The sacculus was next freed from the lateral wall of the pelvis to which it was bound down by strong adhesions. A large vein was torn near the spine of the ischium, which gave rise to very troublesome hæmorrhage. To control this the wound was partially plugged with gauze.

The bladder was next opened, and the finger introduced into the opening of the sacculus. The wall of the latter was incised about ½ in. from the opening into the bladder, and the incision was then carried round this opening, leaving a small portion of the sacculus attached to the lateral wall of the bladder.

The left ureter was found to be greatly thickened and occupied a deep groove in the posterior wall of the sacculus, from which it was stripped with some difficulty.

The opening in the bladder wall was closed with a continuous catgut suture, both the pelvis and bladder were drained, and the wound partially closed.

Convalescence was very slow, the suprapubic sinus remaining open for some time. This sinus tracked round the bladder and opened into that viscus at the site of the old opening of the sacculus. Four months later the bladder was again opened, and the edges of the sinus were freshened and drawn together with interrupted catgut sutures. After this the wound healed soundly.

The sacculus was somewhat pear-shaped, the narrow end being below. On its right lateral wall at the junction of the upper and middle thirds was the opening into the bladder. It was about half as large again as the bladder itself, and could easily contain an orange. A microscopic section of the wall showed three distinct layers of muscular fibres, a well-marked submucous tissue, and a lining membrane of transitional epithelium.

He considered that Mr. Berry's case was the most successful instance of extirpation of the bladder which he had seen or read of in literature. Ten or twelve cases had been reported, and Mr. Pardoe had had one which had not been reported yet. The two cases which had just been narrated completed the total number. He thought his own case was a congenital one, even though the patient was now 51 years of age. The microscopical section showed three distinct areas of muscular tissue, and there was normal mucous membrane. The patient had had some symptoms referable to the bladder for the greater part of his life. Other authors described cases in which there were three distinct layers of muscular tissue, and they also had classed them as congenital. Whether that was so must be left for future investigation.

Mr. PAUL B. ROTH desired to mention a third case of the kind, one which was under the care of Sir Frederic Eve at the London Hospital, in 1909. He thought that was certainly congenital, because the right kidney was lying in the right iliac fossa.

The PRESIDENT asked if Mr. Berry would explain the difference in the relationship of the ureter to the cysts, supposing them both to be congenital. The ureter in Mr. Berry's case went into the sacculus, and in the other case passed behind it.

Mr. BERRY, in reply, said he was much interested in hearing of Mr. Joly's case. It was, however, not quite of the same kind as his own, as the opening of the ureter was in the bladder, not in the pouch itself. Other cases had been recorded in which the ureter opened into the sac, but in these it was unusually close to the main opening into the bladder. The ureteric orifices could easily be drawn into the pouch as the latter developed; but in his case the orifice of the ureter was so far from the mouth of the pouch that he did not think it could have been merely drawn into it. He expressed his gratitude to those who had mentioned other cases. Another point in which Mr. Joly's case differed from his own was that the orifice of the pouch was small.

The Inguinal Method of Radical Cure for Femoral Hernia.

By C. H. FAGGE, M.S.

MULTIPLICITY of operative methods for the cure of any surgical lesion indicates either that a similar degree of success may be obtained in different ways, or that thinking surgeons who follow up the after-results of their cases become dissatisfied with the want of success which has resulted from that method, which up to then they had regarded as the best, and so are led to originate a new procedure or to modify their former operation. I think we shall be of one mind in agreeing that the latter is unfortunately the true explanation of the many operations described as radical cures for femoral hernia. Lotheissen[1] writes how he—dissatisfied with the usual methods, such as those of Fabricius,

---

[1] *Centralbl. f. Chirurgie*, 1898, xxv, p. 548.

Bassini, and Kocher, because they only produced a fibrous barrier in place of a muscular one, and therefore favour recurrence—was led, in May, 1897, when dealing with an inguinal hernia by the usual route, to devise a plan for the cure of an associated femoral hernia. Instead of suturing the conjoint tendon to Poupart's ligament he brought the former further back, and anchored it with interrupted sutures to Cooper's ligament so as to raise a muscular wall between the femoral canal and the parietal peritoneum. (I need scarcely mention that by Cooper's ligament is meant the upper thickened margin of the fascia covering the pectineus which extends outwards from the posterior edge of Gimbernat's ligament along the ilio-pectineal line, though I notice that one American writer speaks of the deep crural arch as Cooper's ligament.) Lotheissen goes on to relate his satisfactory experience of this method in subsequent cases. Apparently quite independently Parry was led to employ the same method, for in a paper[1] read before the Surgical Section of the British Medical Association in 1901 he finds fault with methods which approach the sac from the thigh—e.g., Bassini's, Watson Cheyne's, and Lockwood's operations—in that an efficient support cannot be provided by the approximation of structures outside Poupart's ligament, and details an operation which he had planned some four years before, and which differs from Lotheissen's only in exposing the sac from below and drawing it up above Poupart's ligament by a suture passed through the fundus, after the manner described by Macewen for inguinal hernia. The sutures bringing the conjoint tendon into apposition with Cooper's ligament are also tied below. Parry quotes a series of fifteen successful cases of this operation for femoral hernia, of which five were strangulated, but gives no evidence of the permanency of their cure. Again, T. E. Gordon[2] describes a very similar method, and says that Tuffier, in 1896, recommended the inguinal route. Yet while all these observers are searching about for a method which may truly be described as radical, Thomson and Miles, in the third edition of their text-book, state that "opinion is gaining ground that no attempt should be made to close the femoral canal."

Some four years ago I came across two recurrences after I had dealt with reducible femoral herniæ by Kocher's operation, and in these and all subsequent operations for femoral herniæ, whether reducible or strangulated, I have employed Lotheissen's method. Of the thirty-one who have now been operated upon long enough to draw any conclusion as to the permanency of the cure nine were strangulated, ten irreducible, and twelve reducible. Two of the strangulated cases died. I have examined eighteen of the twenty-nine who recovered during the last few weeks, and found three recurrences: all these were operated upon in 1910. The first was a strangulated hernia in a feeble old woman, aged 67. The second was a strangulated hernia, with symptoms of one week's duration, in a man the subject of chronic bronchitis, and in releasing the bowel after dividing Gimbernat's ligament from above it gave way at the site of constriction, and was sutured. The patient recovered, but it was necessary to drain the wound. The third recurrence occurred in a man operated upon under spinal anæsthesia because of glycosuria.

---

[1] *Brit. Med. Journ.*, 1901, ii, p. 1136.

[2] *Brit. Med. Journ.*, 1900, i, p. 1338.

As Kammerer remarks,[1] late recurrences of femoral hernia are common, and often occur after two years if the crural canal has been closed. Even without this precaution recurrence in my thirty-second case only took place five years after Kocher's operation, so that I do not of course claim the remaining fifteen as cures, but several women have gone through pregnancy satisfactorily, and one patient who was six months pregnant at the time of my operation has borne two children since. In another case I subsequently removed the appendix, and found the femoral region quite sound from the abdominal aspect.

So many of the younger surgeons now use this method that it is unnecessary for me to give exact details of the process, and those to whom it is unknown I would refer either to Lotheissen's original paper (loc. supra cit.) or to Mr. Rowlands's description in Jacobson and Rowlands's "Operative Surgery,"[2] but having either myself, or under the supervision of others, made use of it thirty-three times, I should like to make a few practical remarks about details which appear to me to be of first importance. Owing to the presence of the cord it is undoubtedly more difficult in the male than in the female, but it is relatively of greater value in strangulated than in other cases, owing to the ease with which the imprisoned coils can be manipulated in full view, and carefully examined before they are returned. Had the old method been adopted in my case, in which the bowel was unavoidably torn, I cannot think that the result would have been so good, and the occurrence of this complication might even have been overlooked. Gimbernat's ligament may, when necessary, be divided outside the sac from above, and an abnormal obturator artery can be seen and avoided. The sac can usually be dragged bodily upwards out of the femoral canal after it has been opened and its contents returned. In freeing this the deep epigastric vein has to be defined and avoided. It was not until my thirty-second case that I injured this vessel, and was compelled to ligature it after some annoying hæmorrhage. After ligaturing the sac and allowing the stump to retract upwards the divided edges of the parietal abdominal fasciæ (transversalis and iliac) may with advantage be brought together over the sac by interrupted catgut sutures. Three or four interrupted sutures are necessary for the approximation of the conjoint tendon and Cooper's ligament after free definition of the latter ; in passing these sutures lies the chief difficulty of the operation. The outermost must be passed through Cooper's ligament just behind the inner border of the femoral vein, which requires full and careful retraction. It will be found easier to pass these mattress sutures first through Cooper's ligament, and to tie them above rather than below as advised by Parry. A half-inch cutting needle is used, while the peritoneum and bladder are protected with flat retractors. I have seen others use Symonds' fish-hook needles for this purpose as recommended by Rowlands, but myself regard them as both unnecessary and likely to increase rather than reduce the momentary difficulty. I have been accustomed to use iodine catgut throughout. I have not seen any evidence of compression of the femoral vein by these sutures, but one of my cases, otherwise successful, has developed a saphena varix.

---

[1] *Annals of Surg.*, Lond., 1904, xxxix, p. 982.

[2] "Operative Surgery," 5th ed., ii, p. 97.

DISCUSSION.

The PRESIDENT said he did not himself remember, but did Mr. Fagge know what was the percentage of recurrences after some of the older operations for the treatment of femoral herniæ? In the old days, when operations were done only on strangulated cases, it used to be said that recurrence was rare after the simple operation then practised. It would be interesting to know the proportion of successes in the various methods of operation.

Mr. FAGGE, in reply to the President, said there were three recurrences in the eighteen cases. He believed that he had seen it stated that recurrences after operations from below varied between 10 per cent. and 20 per cent., but he imagined this was too low an estimate, as he had three failures in the first two years that he was assistant surgeon at Guy's Hospital and practised Kocher's method.

Surgical Section.

May 9, 1911.

Mr. Rickman J. Godlee, President of the Section, in the Chair.

Demonstration of Cases of Cleft Palate.

Before the debate, reported below, a demonstration of cases of cleft palate took place. A very large and striking array of patients was shown, about sixty in number.

Cases were shown by Mr. R. W. Murray (Liverpool), Mr. James Berry, Mr. Arbuthnot Lane, Mr. C. H. Fagge, Mr. T. H. Kellock, Mr. Bilton Pollard, Mr. W. G. Spencer, and Mr. G. E. Waugh.

Of these by far the larger number had been operated upon by Langenbeck's method, as a rule at the age of 2 or 3 years or more. The age of these patients now ranged from 5 to 30 years.

In a few of the cases exhibited the operation had been done soon after birth by the "turn-over flap" method. These patients were all quite young children at the time of exhibition.

The Treatment of Cleft Palate.

An Address Introductory to a Discussion on the Subject.

By W. Arbuthnot Lane, M.S.

I am much indebted to you for the honour you have done me in asking me to open the discussion on cleft palate and its treatment. I fear, however, that I have nothing to add to what I have been saying for the last fifteen or sixteen years.

When I was working in the dissecting room on the factors that determine the changes in the form of the skeleton, consequent on a specialization of the functions of the individual, I was particularly interested in the study of the forces that determine the development of the bones of the face. This led me into serious conflict with many of those gentlemen who devote themselves to the study of the diseases of the throat and nose, especially in regard to the causation and treatment of adenoids. This arose through the demonstration of the mechanical influence exerted by the free passage of air through the nasopharynx, and its effect on the development of the bones surrounding this cavity. After much controversy of a more or less acrimonious nature on their part, these gentlemen absorbed the principles involved into their treatment, and breathing exercises have even become a portion of the routine of the education of the Board School child.

I merely refer to this, as the factors which affect the development of the nasopharynx have an important bearing on the principles involved in the treatment of cleft palate. I recognized that during the persistence of such a degree of cleft of the palate as entails the absence of the pressure normally exerted by the air in its passage through the nasopharynx the factors that determined the normal development of the nasopharynx and surrounding bones were in abeyance, and that those structures did not respond as they should to normal evolutionary factors. At the time I first devoted my attention to the study of cleft palate, and of its treatment, there was as far as I could learn no difference of opinion among surgeons all over the world as to its treatment. The cleft in the infant was deemed inoperable, and while it was not considered wise to operate under 3 years of age, the time of election was between 4 and 6 years.

What struck me as extraordinary at that time were two facts. The most conspicuous was that surgeons were satisfied to operate on but a very small proportion of the cases of bad cleft palate, since only a fraction of the number of such cases survived to the age at which the operation they recommended was performed, namely, 4 to 6 years. They did not seem to realize that the closing of the cleft in the hard palate was an operation of urgency in severe cases, and that it was a condition that called for immediate interference in order to save the life of the child. I was also impressed by the fact that the operations which they performed with so little success at the so-called period of election with instruments that amazed one by their clumsiness and multiplicity were quite unsuited for such delicate procedure as the closing of clefts in infancy. Even if the patient survives, many of the operations which are easily performed in infancy are impossible later in life, since the presence of the teeth deprives the surgeon of about two-thirds of the breadth of the flap by means of which the aperture can be closed. I therefore determined to operate as soon after birth as possible in order to save many of the lives of those infants with extensive cleft palates who would otherwise succumb if not operated on. This procedure was fully justified by the results, since the closure of the cleft was followed by an immediate and progressive improvement in the constitution of the child.

It was obvious that the operative methods and the instruments then in use would be of no service for such a delicate operative procedure. I therefore devised the flap method, which I have employed throughout. I first attempted to reduce the instruments then in use to some reasonable proportions. They were, however, hopelessly inadequate for the purpose required of them. Early in 1896, Messrs. Down Bros. made for me a needle-holder with minute needles, which I have used ever since. This instrument has remained unchanged in its original form. They also made gags for me which have been equally useful and successful. By means of these implements I have operated on a very large number of cases during the last sixteen or seventeen years, probably on many more than any surgeon has ever operated on, or will operate on in future, since such an opportunity will in all probability never arise again. In England the opposition to any change in the surgical creed was so intense that I had the whole field to myself till quite recently. My opponents were satisfied to stand aside and criticize, and did not take the trouble to render themselves familiar with facts. Consequently I was inundated with material, and operated on as many cases as I wished to. The number was only limited by the fact that even in a children's hospital the proportion of infants that can be admitted into a ward is limited, and the number of cots in a general hospital is small. While the surgeons here were satisfied to criticize or merely abuse the procedures I advocated, those abroad after a considerable time took steps to operate earlier in life, using various methods to effect closure of the cleft.

Some of our surgeons have recently begun to realize that their beliefs on the subject of cleft palate and its treatment were not based on quite such a secure foundation as they had believed, and they have gradually reduced the age at which they operate on cases of cleft palate. The best evidence I have of this is that the supply of infants with cleft palates sent up to the Hospital for Sick Children under my care has diminished enormously within the last year, while at the same time the simpler forms present themselves in a smaller proportion than heretofore. I also gather from many of my more progressive friends that they operate freely upon them with great success. The mortality of the operation is comparatively small considering the desperate condition in which some of these children are when admitted, and the slight hold on life which many of them have. Mr. Barrington Ward, our Medical Superintendent at the Hospital for Sick Children, tells me that the mortality does not exceed 5·9 per cent.

It is perfectly obvious that the more serious the case the earlier in life it is operated on the better. The urgency varies directly with the severity of the deformity, and in severe cases every hour which elapses between the birth of the child and the operation is of considerable importance. The earliest I have done has been within seven hours of birth. In the first few days of life these infants are at their best, and appear to show very much less evidence of shock than they do later. Besides at this period the procedure is a very simple one, and improvement follows rapidly. Changes in the form of parts such as the premaxilla, &c., take place with extraordinary rapidity during the first week of life as compared with the changes which ensue afterwards. The effect of closure of the cleft on the general condition of the child is also most striking.

Putting the matter as briefly as possible, early operation saves a large number of lives which would otherwise be lost. Many of the clefts present in those cases can only be closed before the gums are encroached on by the teeth. The sooner the nose is separated off from the mouth the earlier the nasopharynx is exposed to the influence of the mechanical factors which normally determine the developments of this passage, and of the structures which surround it.

Fifty-six Cases of Cleft Palate treated by Operation, with Special Reference to the various Methods of Operation, and the After-results.

By JOHAN ULRICH, M.D. (Copenhagen).

IN communicating, at the invitation of the Surgical Section of the Royal Society of Medicine, some of my results of operations for cleft palate, I must first express my regret at the difficulties imposed by having to address you in a language foreign to me and at the fact that it has been impossible for me to demonstrate my patients here.

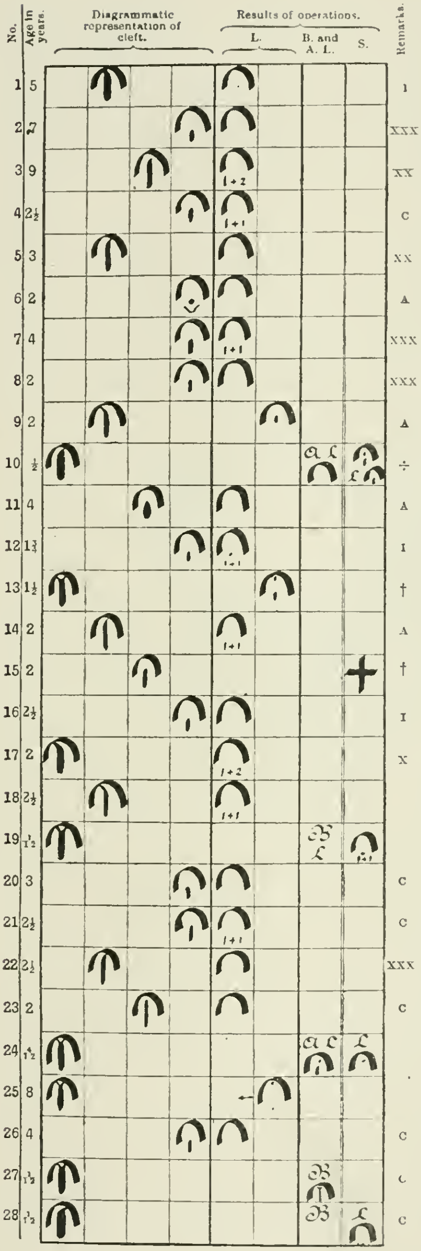
Since 1903, when I began these operations, I have had 77 cases, of which 21 have not been operated on, partly because of the presence of other mortal diseases—encephalocele, heart disease, and others—or because the operation has been postponed. In a few cases I have refrained from operating on account of the breadth of the cleft.

The 56 cases, which have been operated on, I have noted on the diagram (*see* p. 175). The first two columns of the diagram give the numbers and ages of the patients ; the next two columns show the complete clefts, the first the broader (as a rule about 2 cm.) and the second the narrower clefts ; the next two columns show the incomplete clefts, broader and narrower. In the next two columns are shown the operations by Langenbeck's method. The former of these gives the operations where healing is attained absolutely or practically with a hole only passable for a fine Bowman's probe, or a little cleft in the uvula not worth an operation (the numbers indicate how many operations I had to do to secure healing, while the absence of a number indicates that only one operation was done) ; in the latter are given the cases in which complete healing has not yet been attained. Some of these cases belong, perhaps, rather to the healed class, as the holes are quite insignificant. In the next column are some cases operated on by Arbuthnot Lane's and Brophy's methods, or by a modification of the latter operation, which I have tried but have almost abandoned. The last column but one contains some supplementary operations with remarks appended. The sign (†) indicates the cases of patients who have died after the operation. The last column refers to the speech, so that three crosses (xxx) indicate normal or nearly normal speech, and one or two crosses show the cases in which the results were not so satisfactory. Of course, the estimate of the quality of speech is personal. The records are rather defective; many patients have not yet learnt to speak; many are living in the country (marked C), whose capacities I cannot test; others, again, have been lost sight of (marked A).

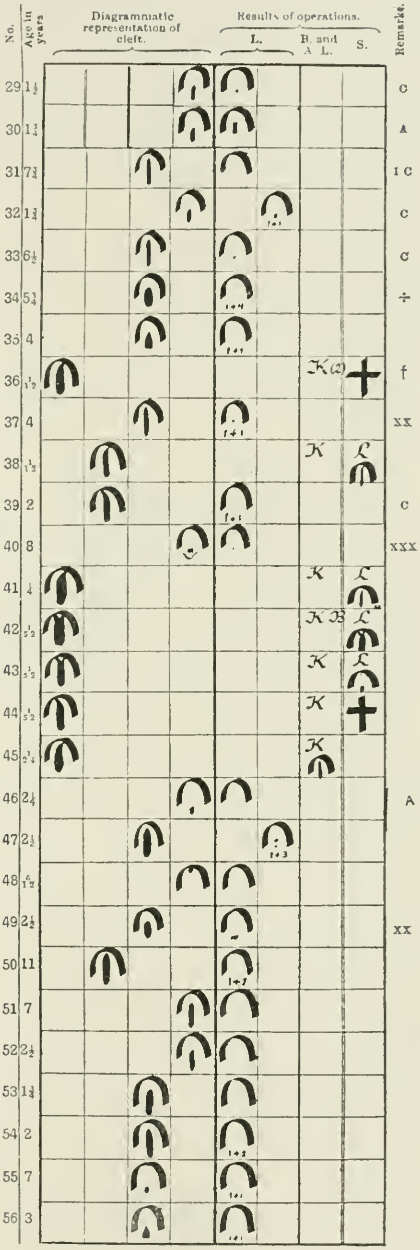
I may here mention that in Denmark, State support is afforded in two different ways for the treatment of cleft palate. First, by paying a dentist in Jutland and another in Copenhagen for making artificial palates for a certain number of patients; and, secondly, by receiving patients operated on or patients with protheses for instruction in speech. It is now arranged that if a patient comes to the institute with a cleft palate he may get a prothesis even for the smallest cleft in the uvula, because financial provision is made only for protheses, not for fees for surgeons and operations. It is impossible to imagine a more stupid arrangement. The explanation is that the matter has been arranged by a smart dentist in Jutland in connexion with some Members of our House of Commons. It will, however, certainly soon be altered, and our medical director, who is a surgeon himself, is at this time interesting himself in bringing about a better arrangement—namely, that a surgeon shall direct the treatment of all cases and decide as to which cases shall be operated on and which shall have artificial palates, and be sent on to the dentists.

As you will see in the diagram, the patients, except those operated on by Brophy's or Arbuthnot Lane's methods, have been operated on between the ages of 2, or a little below, and 11 years. Of course, for functional results it is best to operate early, but according to my experience it is much easier to effect a cure in some older children than at the age of 2 years or below, and I think that I shall in future prefer to operate a little later—for example, at the age of 3 years.

In reference to the technique, I use Geffer's gag (*see* p. 177), depressing the tongue and passing a string through it which I fix to the gag. In some cases of small children I have used Arbuthnot Lane's gag, and have found it very practical. For the freshening I employ besides different knives, Trélat's lance coudée, especially for the anterior angle. For loosening the muco-periosteal flaps I mostly use Trélat's rugines, one for the right side, another for the left, and one, more bent, for the anterior angle. I find them more handy and more powerful than the English rugines that I have used. These I find more suitable for completing the loosening. For the detaching of the nasal mucous membrane from the hard palate I generally use Mr. James Berry's scissors, but I find them a little too big—it is not always easy to



No.	Age in years	Results of operations. L.	B. and A. L.	S.	Remarks.
1	5				I
2	7				XXX
3	9	1+2			XX
4	2½	1+1			C
5	3				XX
6	2				A
7	4	1+1			XXX
8	2				XXX
9	2				A
10	½		A L	L	÷
11	4				A
12	1¾	1+1			I
13	1½				†
14	2	1+1			A
15	2			✝	†
16	2½				I
17	2	1+2			X
18	2½	1+1			
19	1/12		B L	1+1	
20	3				C
21	2½	1+1			C
22	2½				XXX
23	2				C
24	4/12		A L	L	
25	8	←			
26	4				C
27	1/12		B		C
28	1/12		B	L	C



No.	Age in years	Results of operations. L.	B. and A. L.	S.	Remarks.
29	1½				C
30	1¾				A
31	7¾				I C
32	1¾	1+1			C
33	6½				C
34	5¾	1+4			÷
35	4	1+1			
36	1/12		K (2)	✝	†
37	4	1+1			XX
38	1/12		K	L	
39	2	1+1			C
40	8				XXX
41	¼		K	L	
42	1/52		K B	L	
43	1/52		K	L	
44	1/52		K	✝	
45	3/26		K		
46	2¼				A
47	2½	1+3			
48	6/12				
49	2½				XX
50	11	1+7			
51	7				
52	2½				
53	1¾				
54	2	1+2			
55	7	1+1			
56	3	1+1			

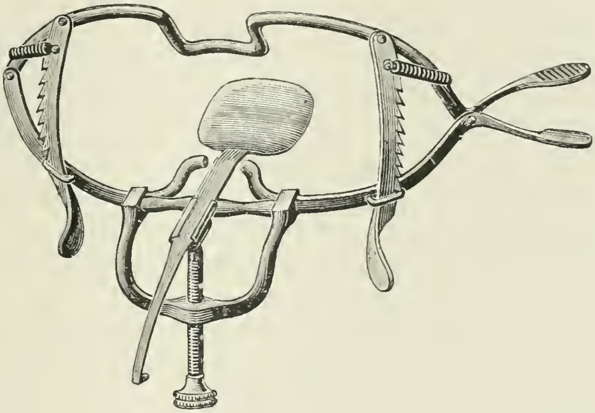
The diagrams show the palate seen from below, with a rough representation of the extent and situation of the cleft. K, clamp employed; B, Brophy's operation; L, Langenbeck's operation; A L, Arbuthnot Lane's operation; XXX, good speech; XX, fair speech; X, poor speech: C, in the country, cannot be traced; A, no address; I, imbecile; ÷, bad result; †, death.

manage them with small children. For the sutures I use metal wire (aluminium-bronze) and Trélat's needle. It is passed through the right border of the cleft, threaded with the metallic wire, drawn back, turned 180 degrees, then passed through the left border and the string released. The technique is very easy and the wire is generally well placed. For the uvula I use silk, small Hagedorn's needles, and a little needle-holder constructed by Doyen.

As to the results of the operations, you will see that in the 44 cases operated on by Langenbeck's method I have effected healing in 37; in 22 in one operation, in 13 after two operations, in 3 after three, and in 1 after five operations. The cases which have required two or three operations have in part been those where the closure of the anterior angle of the cleft has been postponed, and in part those where a little hole has remained, generally between the hard and the soft palate. A few cases have been more difficult on account of a bad constitution or of throat diseases with virulent microbes. Some of these cases are still not healed. No. 9 has disappeared. No. 13 died at home from pneumonia in no way related to the operation. No. 32 was very difficult to operate on, always becoming asphyctic during the operation as soon as the mouth was opened sufficiently for operating purposes. In No. 47 there were four operations; there still remain two small holes in a very thin palate; it is possible that the tissue is so bad that a complete cure will be difficult to effect. In Nos. 12 and 25 the holes are so small that they belong rather to the healed cases, and are not worth any operation. One patient, No. 16, died three weeks after the operation from pneumonia with empyema, certainly resulting from the operation. I have since learnt that the child had bronchitis before the operation.

As you see, I have operated on two of my patients, Nos. 10 and 24, both bad cases, by Arbuthnot Lane's method. The operation is more difficult than Langenbeck's operation, but not so difficult as is commonly supposed. In the first case (No. 10, a child aged 6 months) I obtained healing *per primam* and thought the operation would prove quite successful. The patient left the hospital with a closed palate, but when I saw the child two years later I found a little hole in the middle of the palate and a cleft 3 cm. long at the back. I tried to close these defects by an operation by Langenbeck's method, but I have not cured the posterior cleft, and when I saw the child recently the appearance of the palate was the same, and the soft palate was very thin and cicatricial. I fear that this patient, even after a more successful re-operation, will speak badly. In the second case (No. 24, a child aged 6 months) the immediate result was closure with a hole at the front and an oblique cleft at the back. The last was treated by a Langenbeck's operation, and when I saw the patient recently there remained only a narrow hole, but the palate, though well shaped, was too stiff. The patient has still not learnt to speak, so I cannot say anything about the functional result.

I come now to my most troublesome cases, in which I have tried Brophy's method. Without doubt it is most rational for those cases where the cleft is broad and the upper jaws are wide apart, to bring them together if possible, and if the operation cannot absolutely be recommended, it is not because it is irrational but rather owing to technical difficulties, the danger to the children, and last, but not least, because the reunion of the cleft is so difficult in newly born babies. I have tried this method in ten cases. The first (No. 19) was that of a child aged 1 month; the cleft very broad, about 1½ cm., was reduced by one-half, and seventeen days later the wires still remaining in situ, I did a Langenbeck's operation with partial healing. One month later another operation was done and the palate is now quite healed. Later I closed the cleft in the processus alveolaris by a Bardeleben's operation, and remedied the harelip. The second case (No. 27) was in a child aged 1 month with a cleft of 2 cm. The patient was so ill after the operation that I dared not continue, and the closure has been postponed. In the third case (No. 28, a child aged 1 month, with a cleft of 2 cm.) the effect of the operation was not sufficient, and the closure of the palate was first tried



Geffer's gag with tongue-depressor.

three years later. The cleft was then only ½ cm. broad. I cured the cleft in one operation and two small re-operations for a hole in the anterior angle and a little hole more to the back.

For squeezing the two jaws together better and more successfully I tried a clamp with two branches furnished with teeth, which should be screwed together with a key. The instrument looks quite brutal and is more like an instrument of torture, but when applied under narcosis it seems to me to be no worse than the wires and plates in Brophy's operation; it works more successfully, can easily be removed and applied again, and one can squeeze the jaws together as much or as little as one likes. Though I have found the instrument to work well in a technical sense, I must confess that the results have not been satisfactory. I have used it in 7 cases, and in 2 (Nos. 36 and 44) death has resulted. In the first of these fatal cases (No. 36)—a child a month old, with a cleft of about 2 cm.—it worked at first well, and the cleft was reduced to ½ cm., but the child becoming infected from a salt water injection it was removed. The treatment was tried again nine months later (certainly an error of judgment). The child again contracted an acute infection and died the next day. In the second (No. 44), which was a similar case, the cleft was reduced from 2 cm. to ½ cm., but the child died six days later from a septic phlebitis in the cranial veins. In three cases (Nos. 38, 41, and 42) a closure of the palate was attempted without success. In one of them (No. 42), which I saw recently, the cleft was still so broad that I did not operate, and the child will certainly have to have an artificial palate. In only two cases have I had any success by the squeezing. In one of them (No. 43), which I operated on recently, two years after the squeezing, there still remains a little cleft in the soft palate, but this I feel certain of being able to cure. The other (No. 45) has only been squeezed to diminish the cleft, which was very large. When I saw the child recently the cleft was ½ cm. to 1 cm., and I hope to get it closed in the near future.

Of course, the operations have been undertaken only in the worst cases, but the results are not good. As to the mortality, it is, in my cases treated with Brophy's and similar methods, 2 out of 10 (i.e., 20 per cent.); but in the first case it was certainly imprudent to try the method again, when the child was aged 10 months. And one must remember that the mortality of such children certainly is relatively high. The failure of the early operations is, in my opinion, due essentially to the fact that the healing is much more difficult to obtain at a very early age. The tissues have certainly a more fœtal character, are more fragile, and the squeezing itself will also diminish the vitality of the tissues. The last two cases (Nos. 43 and 45) seem to prove that a prudent squeezing can help to diminish a large cleft, so that the operation later will be easier and the result better.

But another question is, Shall we in any case operate on such very broad clefts? I am convinced that in most of these cases, even when healed after any method of operation, the patients will speak rather badly. On the other hand, I have seen several adult patients with very broad clefts and prothesis who have spoken very well, almost excellently, and we have all heard of patients with healed cleft palates speaking so badly that the palate has had to be cut through again to apply a prothesis. Of course, the inconveniences due to the prothesis treatment are many and great. The prothesis has to be changed every two or three years in children; it demands a great deal of care and much trouble, and the patient and the relatives lose much time in visiting the dentist. I am therefore convinced that the treatment by operation has its limits equally with that by prothesis. Where the surgeon will place the limit is, of course, a matter of taste, but looking at my 56 cases, I believe that about 10 would have done as well without any operation. Of course I would rather operate on a child from the country or on one from the lower classes than on one in better circumstances and from a large city.

Another inconvenience in the prothesis treatment is that the artificial palate certainly spoils the teeth, and especially when it is not kept clean the teeth will often be destroyed by caries, but, on the other hand, the teeth in these patients are, as is well known, often very bad, and, especially after Arbuthnot Lane's and Brophy's operations, they are often almost ruined. The front teeth are also generally very defective in cases of complete cleft palate, and the top lip is frequently depressed or crooked, and therefore it will often be advisable to give the patients, even those well operated on, a prothesis in substitution for the defective teeth and to regulate the exterior.

My position is roughly as follows: In the worst cases I operate if necessary on the processus alveolaris by the unilateral method with a Duplay's operation, by the double method after Bardeleben, and prefer to operate as early as possible. I operate subsequently for harelip at the age of from 4 to 6 months. The cleft in the palate will then, as a rule, gradually diminish; but if it is, at the age of from 2 to 3 years, still very broad, I abandon the patient to the prothesis treatment. Of course, I prefer to operate on patients right out in the country rather than on others.

I think there are cases where careful pressure of the jaws in the newly born child will make the operation easier later. In the other cases, where the cleft is narrower, I operate if necessary on the processus also as soon as possible; on the harelip at the age of from 4 to 6 months, and for the cleft palate I prefer Langenbeck's operation at the age of about 2 or 3 years, with a preference for the latter age.

DISCUSSION.

Mr. Douglas Drew thought Mr. Lane was to be congratulated on bringing his cases forward. No doubt he had influenced many surgeons greatly in regard to the operation on cleft palate, in that many now operated at a much earlier age than formerly. He (Mr. Drew) had operated as young as 7 months old, but he had never performed Mr. Lane's operation. That was first because it always appeared to him to be a dangerous operation in so young a child, and secondly, because a large amount of cicatricial tissue must be formed as a result of the operation, resulting in much more limitation of the palate than with the ordinary Langenbeck's operation. Where the movement of the palate was limited, he thought that must influence the speech in after-life. The most important point about the operation was the condition of the speech, which must depend on the mobility of the palate. He had examined nearly all the cases brought that afternoon in regard to mobility of palate and voice, and the cases operated upon by Langenbeck's method appeared to him to present a better appearance and better mobility than the others. With regard to the period at which the operation should be done, he used to do it at 3 years of age, but during the last three years he had done it earlier, as early as 7 months. He had only performed Brophy's operation once, and was much disappointed with it, though that might have been owing to his deficient skill. On drawing the jaw together with the wire, he found that the palate process was rotated, so that it became almost vertical, and so was much more difficult to repair. With regard to the old question as to whether the hare-lip or the cleft palate should be operated upon first, he did not doubt that by operating on the hare-lip the cleft palate was much reduced in breadth, and was more easy to repair. He exhibited a gag which fitted on the alveolar arch, was very narrow, and did not overlap the palate. It facilitated the operation. He considered Mr. Berry's needle was the most convenient which has been devised. It could be introduced from one side to the other without difficulty. Whatever the method of operation, it must be admitted that in most of the cases the resulting speech, when the child grew up, was very disappointing. In the majority of children seen that afternoon, it could not be said that the speech was good, though of a few it might be said that one would hardly have known that they had had cleft palate.

Mr. C. H. Fagge said he had operated on 57 cases of cleft palate, of which 38 were infants under 1 year of age at the time of their first operation, and he proposed to confine his remarks to these early operations, as it was evidently with this object that the Secretary had asked him to speak at this stage of the debate. Of these 38 cleft palates, in 17 there was an associated single hare-lip and in 10 a double hare-lip—i.e., the cleft was complete from back to front, and the premaxilla was projecting forwards. He would be glad to know whether the advocates of late operations met with anything like a similar proportion of these difficult cases in their series, for it seemed to him that the question of how and when to deal with an incomplete cleft was of less importance, and presented far fewer difficulties, than were met with in treating a case which was open from back to front. One of the 38 died during the palate operation, and two died in hospital, but the remote mortality was much more alarming, for an effort to trace these patients by correspondence for this meeting showed that 14 others had died from various causes, of which 6 were directly or indirectly due to subsequent operations for hare-lip.

With regard to his views as to the age of selection he had, following the excellent lead of Mr. Arbuthnot Lane, been gradually led to operating earlier and earlier, and usually took the matter in hand as soon as the child had begun to thrive on spoon feeding, at about 1 month; with regard to the question of method, he had always been able to close an incomplete cleft by Langenbeck's method, but from his own experience he did not doubt that the only way to close a wide cleft associated with a single or double hare-lip was by Lane's flap method; he had uniformly failed when using Langenbeck's plan for these extreme clefts, though perhaps it was a better method for dealing with small clefts involving the soft palate only. In his earlier operations he had first united the whole palate and sutured the lip at a subsequent date, but in all recent cases he had done the whole operation at one sitting; yet each case usually required more than one subsequent operation to close recurrent openings in the palate. He had eight of his cases there that evening, of whom six had been operated upon under

1 year of age. He had written to all of some 60 cases on which he had operated, and he had presented to the meeting all the patients who were available, one coming up from Penzance for that purpose; no selection had been made, and he was willing to abide by the judgment of others on this series.

Unfortunately, he had no opportunity that afternoon of comparing his results from the cosmetic and speech point of view, as well as in the matter of breathing through the nose, with those of Mr. Berry or of others who advocated late operations, but he had to-day been much struck by the improvement in the appearance of the palate, and the conformation of the whole upper jaw in three of his cases which had undergone early operations, and which he had not seen for five years; so marked a degree of alteration could scarcely be expected if the nasal cavity were allowed to remain functionless, and therefore undeveloped, owing to its free communication with the mouth during a period so important to natural growth as the first two years.

The mortality from malnutrition among babies suffering from bad cleft palates was very high, and in fact he had never seen a baby, either at Guy's or the Evelina Hospital, who had arrived at 2 years with a double cleft palate and hare-lip; from this it seemed a logical deduction that early operation was a life-saving proceeding; as late operators avoided a large percentage of these—the most difficult cases—it was unfair to compare their results with those of advocates of early operation when, as in his series, about two-thirds were of this extreme form.

Mr. JAMES BERRY proposed to limit his remarks mainly to facts; he would not go into the question of the physiology of the nasal passages, or the cause of the nasal intonation in cleft palate speech. The number of patients on whom he had operated for cleft palate was 144, of whom 57 had complete cleft of the whole palate associated with hare-lip; 28 had a cleft of the soft palate only, and the remaining 55 a cleft involving the hard palate to a greater or less degree. The latter class included the kind of cleft palate which, in his opinion, was often the most difficult to cure—i.e., that which stopped just short of the incisor teeth and had a broad rounded end. In opposition to Mr. Fagge, he thought that complete clefts were more easy to close than clefts of the type just referred to, of which he had shown more than one example that day. He had never refused to operate on any cleft palate, however wide, unless the patient were (1) over 16 years of age (he had done a few beyond that age), or (2) in such very bad health that no operation whatever was justifiable; or (3) one in whom one or more operations had been done by other surgeons without success; he had operated, however, on a good many of the last class. Mr. Lane said there were many cases of cleft palate which if not operated upon in infancy could never be closed afterwards. He (Mr. Berry) had never seen such a case, and he would be grateful to Mr. Lane or anybody else who would show him a case at the age of 2 or 3 years in which the cleft was too wide to be closed by a Langenbeck operation. With very few exceptions he had been able to effect closure in all his cases, although not always by a single operation. As regards the soft palate, in his last 100 cases there were only 2 in which he had not obtained union after a single operation.

With regard to the mortality, in his 144 cases he had had no death at all. With the help of the sisters of his wards, and the dressers and house surgeons, he had been in the habit of following up his cases after they left the hospital, and he had seen or heard of nearly all of them. There were three who had died many years afterward. One of them died from heart disease, having had two attacks of rheumatic fever before his operation. One died recently, ten years after the operation, from an operation for acute appendicitis performed in the country. The third died some little time after the operation from a severe burn. With regard to the question of the early flap operation being a "life-saving" one, that was a very difficult matter to settle without very careful statistics, which hitherto had never been produced. His strong belief was that the early operation was the very reverse of life-saving. When he saw a young child with a wide cleft palate and much emaciated, he usually instructed the nurse or mother to feed it properly and carefully until it was better nourished, and then he operated upon it at his own time. If those children were nursed properly he did not find that many of them died. He would be glad to have from those who advocated very early operation some definite statistics as to how many they had done, and how many of the children were alive, not when they left the hospital, but say one year afterwards. Such information had not yet been produced, although it had been asked for in the medical Press more than once. With regard to complications, in only two of his patients had such occurred. One was a secondary hæmorrhage which occurred on the tenth day, and which stopped spontaneously and immediately; the child was present that evening. The other was a case of empyema, which occurred about a month after the operation. The child was operated upon by another surgeon, and he (Mr. Berry) had not seen it since. But it occurred some years ago, and he knew the child was alive and well a few days ago.

With regard to age, he had operated at all ages between 4 months and 26 years. The patient operated on at the latter age was one of the fifteen whom he showed that day. The one done at 4 months he did not think it worth while to show; it was merely a narrow cleft in the soft palate, the kind of case in which anyone could get a good result. The period of choice for the operation he regarded as about 2 years of age, though there were many cases with narrow clefts which could be advantageously operated upon earlier, some even in the first year of life. It was impossible to mention any age that was suitable for all cleft palate operations; but the difficult ones should be done at about 2 years of age. He had done the Langenbeck operation of median suture in every case, never the so-called flap operation, because from what he had seen and heard of the results of the latter operation it did not seem to him to be a justifiable proceeding. In that matter he was in direct opposition to Mr. Lane. He quite agreed with Mr. Fagge, that in the young infant the double cleft palate often presented a very wide cleft. He showed drawings of the palate of one of his own cases, one of the fifteen patients he exhibited that afternoon when he first saw it in early infancy, and again just before operation. There was a very wide double cleft, but in such a case if the lip were closed in infancy, as was done in that case, the anterior part of the cleft, and to a large extent the whole of the cleft, would become in a year or two much narrower, as the second drawing showed. This spontaneous narrowing of the cleft seemed to him the principal reason why the operation on the palate should be deferred for a year or two. The operation would then be much easier to perform and give a more satisfactory result. With regard to the so-called flap operation, a point which had not been alluded to by any of its advocates was the occurrence of atrophy of the flap. When one turned a large flap of mucous membrane across a broad cleft, it tended to atrophy, and he knew that that happened sometimes, even in the best of hands. But it was not known how often this atrophy took place. He exhibited another drawing which would interest the meeting, because it was one of the few cases upon which he had refused to operate, operations having already been done three times, at a London children's hospital and at a large general hospital in London. The interesting point was that the front part of the cleft had been "closed by a flap operation," as was borne out by a letter which the surgical registrar of the latter hospital had kindly sent him. He saw the child a few years later, at the request of Dr. Gundlach, of Clapton, and was asked to advise as to some further operative proceeding. There was a very large hole in the anterior part, in which one's thumb could be placed. The mother assured him that the hole had come since the operation and was still enlarging. On the other side of the palate was an area of the same size in which the flap had atrophied to the thickness of tissue paper. The soft palate was represented by a huge triangular gap; on account of the extensive destruction of tissue, it was impossible to recommend any further operation. That was not the only case of the kind he had seen after the flap operation, and he would like to know how often this atrophy occurred.

With regard to Brophy's operation, the theory of it seemed to be good, but he had never performed it. The risk to life appeared to be too great. He knew of a surgeon who did the operation on four patients, and they all died. He knew of another surgeon who did one, and it was followed by necrosis of the jaw. If it were done with the idea that the child might die, it must be remembered also that it might live, but with terrible destruction of the superior maxilla from necrosis, though he did not think that often occurred. Dr. Ulrich said that out of ten cases in which he had performed the operation, two died, and he had now abandoned it. He believed Dr. Ulrich's series of ten Brophy cases was the largest consecutive series of that operation which had ever been brought before an English medical society. He was very sorry that Mr. Edmund Owen was prevented from being present, as he was the chief advocate in this country of Brophy's operation, and he had hoped that he would have been able to show a long series of successful Brophy operation cases.

Another point to which he wished to allude was the necessity for speech training after all cleft palate operations, whatever operation was selected, and however it was done. Of the patients he showed that night, only one or two had had any lessons in speaking from a professional teacher; none of the others had had any training whatever, except such as their mothers had given them, and some of the mothers who had been there that day belonged to a somewhat humble station in life, and were not capable of taking much care of their children. That was one of the great troubles which had to be contended with in the treatment of cleft palate in hospital practice in London. He had not been able to show the meeting any of his private cases; these were the patients who could afford to have a governess, and the results as regards speech were, as a rule, better than in hospital cases.

Another point which, he thought, had not received sufficient attention, was the importance of regulating the teeth after operation. When the anterior part of the jaws was narrow and contracted, when the incisor teeth were irregular or deficient, articulation was less likely to be perfect. But if the surgeon would work hand-in-hand with the dentist, as he usually could in the case of private patients, then the results of cleft palate operations, with all due deference to Mr. Lane and other distinguished authorities, were not at all bad, even when they were done in the third year of life, within that period when, according to Mr. Lane's well-known dictum, the operation "should not be permitted."

In conclusion, he wished to demonstrate the speech of a patient, who was operated upon at a period which he (Mr. Berry) was able to choose for himself. It was a case of complete cleft of the whole palate associated with hare-lip. He operated upon this patient's hare-lip at the age of 2 months, and on the palate at the age of 2 years and 1 month, the very period at which it was said by some the operation should not be done. The boy had had no special training, but it would be seen that his speech was practically normal.

[The boy was then asked by various Fellows to pronounce aloud such phrases as "British Constitution."]

This patient was one of Mr. Berry's very earliest cleft palate cases, the third he ever operated upon; if he had been one of the latest, it might have been said that the excellent result was the outcome of long experience. The operation was done on the ordinary principles of Langenbeck's method, and in the ordinary way.

Mr. G. E. Waugh remarked that the discussion so far had turned largely on the mere mechanics of the problem concerned in closing a cleft—i.e., a certain deformity was given, and a certain limited amount of tissue provided for bridging across that cleft. But, so far, no surgeon had dealt thoroughly with the topic of what purposes it was hoped to achieve by effecting the closure of the cleft. The last case which Mr. Berry showed provided a text for remarks upon that aspect of the problem. It was one of an interesting series of cases with structural alterations produced as the result of surgery, upon which he thought any surgeon might congratulate himself. Yet that patient seemed to be the only one amongst the whole of those shown—probably nearly 100 in number—who was able to come and speak to his fellow beings, and who was not obviously betrayed by his speech as being the victim of cleft palate. It was further interesting that among these patients of Mr. Berry's, this was one who had suffered from the grossest type of defect; whereas in those who had had slighter defects the fact that they had had cleft palates was still obvious when they essayed to speak. He himself brought three cases to illustrate that point. He showed a patient, aged 35, who in 1876 had had two operations by Langenbeck's method, by the late Sir Thomas Smith, for cleft palate involving the soft palate only. Neither operation was successful, but he had two small pieces of soft palate elevated against the posterior pharyngeal wall, and freely moving during speech. He also showed a boy aged 17 who had had three operations of the Langenbeck type done by various surgeons before he was 8 years old. He then had a flap operation done upon him, and he still had a cleft involving two-thirds of the hard palate and all the soft palate. He also showed a patient aged 6 who had had a complete cleft of both hard and soft palates, with the premaxilla elevated on the nasal septum. He now had complete closure of the whole defect with a freely mobile soft palate, achieved by Mr. Lane's flap operation when he was 7 weeks old. But if those patients were engaged in conversation, it would be difficult to award the palm of excellence to any one of them for speaking better than another. Here, then, were three distinct types of cases in which speech defect did not seem proportioned to the deformity present in the palate itself. That opened up the question whether surgery was really able to modify the speech of the sufferer from the deformity. The evidence that the cleft in the palate was responsible for the speech defect was very largely an accepted tradition, but had not been subject to criticism. Surgeons saw all varieties and grades of defect of speech which might be associated with slight palate defects, but might also be associated with gross palate defects; there was no proportion between the two. Neither were the after-results to be correlated with the extent of the deformity. There might be left behind a residuum of grave speech defect after a most successful operation, and in spite of the most careful training. In other cases there might be left after operation a not very mobile soft palate, and yet the patient could speak comparatively well. There was also the type of case to be explained in which, without defect of either structure or function in the hard or soft palate, the child spoke as if it had no roof to its mouth, and no mobile palate. That provided one with the key to the discrepancies of speech which followed not only on the operative results achieved by closing the palate, but also from the variations in the degree of speech defect associated with the different deformities.

While it had been assumed hastily that the cause of the speech defect was residual in the structural alteration of the palate, in all probability there were two associated lesions to consider—one in the function of the speech centres, and one in the structure of the palate—and they were not two interdependent lesions. Therefore, it ought not to go forth as the opinion of that meeting that surgical operations on cleft palate could hold out much hope to the victims of that condition that they would be able to disguise their deformity in after-life. Among the cases shown that day, with the exception of Mr. Berry's solitary brilliant case, he could not find a patient who was not very obviously a palate sufferer. The problem of dealing with cleft palate became, possibly, an eugenic one. He had reviewed the notes of 80 cases, and it was surprising in how many there was a history of cleft palate in the family, on one side or on both. If it were assumed, as a result of a consensus of opinion, that surgery would enable a cleft palate sufferer to cover his deficiency of speech and allow him to enter any profession, it would be wrong. Seeing that it tended to become a familial defect, it should be a warning that the stock was not one likely to result, if perpetuated, in happiness for the individual who had to bear the brunt of that lesion. That was particularly hard upon some of the patients, as many of them were highly developed intellectually. Nearly all the subjects of the condition he had seen were above the standard for their age at school, and were noted for their facility and aptness in learning. Hence, their disappointment at being so handicapped would be all the keener. He thought surgeons should contradict a wrong impression as to the benefits which surgery could confer, and should claim only that patients could be enabled to breathe through the normal passages, and allow swallowing to be carried on in a fairly normal manner, as a result of surgical interference with the deformity.

Mr. F. F. BURGHARD said his practical interest in the subject was centred on the two reasons upon which Mr. Lane founded his justification for these early operations. He came there that day hoping to be convinced that the after-results of these early operations were greatly superior to anything done by Langenbeck's or any other method, for it must be admitted, as Mr. Waugh had said, that Langenbeck's or any other method was faulty in some degree. Therefore, he had hoped to see something in the way of results that day to justify the claim to superior functional excellence. After the collection of cases they had just seen, after both types of operation, he did not think anyone would venture to maintain that again. The other point which interested him was that concerning life-saving. There he would join heartily with Mr. Berry in asking for more information. If it could be proved that operation at a very early age was in any way life-saving, that would be very greatly in its favour. He asked Mr. Lane to give some information as to the presumed premature decease of those children. He had never operated on a child under 12 months old, though at the Paddington Green Children's Hospital they came at all ages, from a few days old, to have the cleft palate treated. He kept such cases on his list until he considered they were ready for operation—at any age between 1 and 3 years. The average age at which he operated was 1½ years, and he did not find that the children died in the period intervening between application and operation. Some of them went to other hospitals—generally Great Ormond Street Children's Hospital, where they were operated upon by Mr. Lane—but it was rare indeed to find, on writing for the child to come in for operation, that death had occurred in the meantime. He had inquired into the matter carefully, because it was one to which he had devoted considerable attention, and he appealed to Mr. Lane and Mr. Fagge for the data upon which they founded their statements. He cordially endorsed what Mr. Berry said about the feasibility of closing certain complete clefts in the palate at any age, and he would be glad if Mr. Fagge would send him some cases with a cleft which he (Mr. Fagge) said could not be closed at any time except in early infancy. He had never found insurmountable difficulty, and he thought it was a matter of technique, and that anyone who had had considerable experience in those operations could always close the clefts ; it might be difficult at first, but when one got used to it, it could be managed. He had never done Mr. Lane's operation. On a few occasions he had done the flap operation introduced by Mr. Davies-Colley, upon which Mr. Lane's operation was founded. He (Mr. Burghard) was always displeased with it, because of the unsightly appearance which resulted, and that led him to try to perfect himself in Langenbeck's operation. In Brophy's operation he had had no success. He had a case in which he tried to do it, but no sooner had he got the palate processes wired up than the child stopped breathing, and he had to undo them. In three other cases the operation did not come off owing to an inability to get the palate processes together without very great violence.

Mr. T. H. Kellock considered that it was not quite right to discuss the treatment of cleft palate as if the cases were all alike. They ought to be divided into two classes at least, if not more. A cleft of the soft palate, perhaps extending a little into the hard, was a different thing to deal with from one which came through the whole of the hard palate and the alveolar margin. He was a strong advocate for leaving the more serious of those cases to a later age than Mr. Lane advocated. But clefts of the soft palate in which the two sides lay fairly well together must be looked upon as different from those where there was much difficulty in bringing the parts together. Cleft of the soft palate in a baby was often one of the simplest things to close; the sides could be stitched across and lay in apposition without tension. The result was, as a rule, excellent, both immediate and with regard to the subsequent voice. So one ought not to compare the voice in those children where the soft palate was the only part operated upon with that where there had been a cleft of the hard palate with hare-lip, which was more important still, because he thought in later life hare-lip had much to do with the defect of speech.

He would like to support what Mr. Berry and Mr. Burghard had said, that the advocates of early operation had no right to say that the mortality in cleft palate cases which were not operated upon was greater than amongst ordinary children in that class. There were no figures available to show it. To say that these children died if they were not operated upon early was going too far. It was known that many of them died if they were operated upon; the figures proved that. With regard to the operation for cleft palate, the question was what would be the result to the child, its speech and its power of swallowing. Looking at the children who were exhibited that day, he had not been favourably impressed by the results of the flap operation; the majority of the cases shown were babies still, so that one could not judge of their speech. One would like to see them at 6, 8, or 10 years of age. His objection to the result of the flap operation was that there was much scar tissue, that the palate was crooked, and when the children spoke there was a drawing up on one side. In some the palate was stretched like a bow, so that it was not good functionally. He asked the members to compare that with the result of a successful Langenbeck's operation. By that he meant to include any operation of complete suture of the hard and soft palate in the middle line. Langenbeck's operation, if successful, could produce an almost ideal result; it was the fault or misfortune of the surgeon if it did not. But such an operation was almost impossible in the case of a young baby, because there was not enough tissue to bring together to meet in the middle line. When that was possible was any time after a year or 2 years of age. He agreed that if the cleft were left alone for a year or 2 years it became by that time much smaller than it was in the baby. In babies one often saw a large gap between the premaxilla and the maxilla, but how often was that seen at the age of 2 years? For the reasons he had given, he was in favour of deferring the operation to a later age, when such an operation as he had mentioned could be done. One of the finest testimonials he had ever had as to the result of a cleft palate operation was from the father of a child shown that day. As he was leaving the room he said, "He is in the choir at Woking now." That child had a cleft of the soft palate and part of the hard which had been closed by median suture.

Much had been said about particular instruments for cleft palate operations. To look at the surgical instrument catalogues one would think there was nothing so important for this operation as the instruments. But the fact was that any particular surgeon did the operation best with the instrument he liked best. An instrument which to one surgeon appeared clumsy might be that which another surgeon found best for his purpose. It was a matter of individual skill.

Mr. Berry had laid great stress on the subsequent training of the children. He (Mr. Kellock) would do the same, but would also attach much importance to the care of the child before the operation, for much of the success of an operation seemed to depend on how the child was managed immediately before the operation. Failure—and he supposed all surgeons had failures—was often due to the great change of life and feeding which occurred at the date of the operation. He thought the child's food, habits and surroundings should be the same for some time before the operation as it was intended they should be after it, so that when it recovered from the anæsthetic it would continue the same kind of life as that to which it had been accustomed before the operation.

Mr. L. E. Barrington Ward said: My contribution to this subject is based almost entirely on an analysis and examination of the cases operated on by Mr. Arbuthnot Lane in the Hospital for Sick Children, Great Ormond Street, during the past two years. In every instance the cleft was repaired at as early an age as possible. In this period 369 operations were performed by him for the relief of cleft palate, with a mortality of 5·9 per cent. This figure includes cases where a second or third operation was necessary: the number of new cases operated on for the first time being 172. Of these, one patient was but 10 hours old, 14 were up to 1 week, 43 to 1 month, 66 to 6 months, and 28 were over 1 year of age. Thus it can truthfully be described as an early operation. No case was refused because of the extent of the deformity, but five were discharged unrelieved on account of such varied conditions as bronchitis (2), convulsions, hydrocephalus, and a skin complaint. Nor was any case refused operation on the ground of malnutrition, for the majority of babies afflicted with severe deformity suffer from marasmus, and operation was undertaken with the idea of remedying their disability, and enabling them to take proper nourishment.

Distinction in any particular line brings in its train certain disadvantages. From the statistical point of view at any rate, not the least of these is the natural tendency for the worst cases to gravitate into the hands of the most competent. A very large number of these cases have been sent to Mr. Arbuthnot Lane, and the majority of them have been examples of extreme malformation. Thus in 53 per cent. there were complete clefts of both lip and palate; in 36 per cent. the hard palate, and in 11 per cent. the soft palate only was involved.

In comparing the final results of any two methods it is necessary to take into account the degree of the previous deformity. It is hardly necessary to add that the method of operation was by muco-periosteal flaps—a method which Mr. Lane has made so peculiarly his own. In the majority of instances so wide was the gap that simple paring and suturing even by the most competent hands could not have effected a cure.

I have endeavoured without success to find (in the published writings of the advocates of the so-called late operation) the data on which to determine the age at which in their opinion operation should be performed. It is generally given at about 3 (or 4) years of age, and the only reason brought forward is that it is safer to operate at that age than earlier. On the same analogy it might with equal truth be said that operation should be postponed till puberty or later, for then the risk of operative measures is still further decreased.

Any other objections against the early operation are found on examination to be really levelled against the more modern method of closing the cleft with muco-periosteal flaps, in defence of the old-fashioned procedure of simple paring and median suture. Thus it is claimed, without any proof, that the speech is better after median suture because the palate receives less damage. This we would be ready to believe if the operation consisted only in simple paring of the edges of the cleft, and their approximation by sutures, but an integral part of the operation, except in the simplest of cases, is the free incision of the lateral part of the palate, and sometimes fracture of the hamular process also. The watchword of the median suture operator is "no tension," and to ensure this, according to a widely read modern text-book on surgery, it is necessary to divide the levator palati, and to snip across both anterior and posterior faucial pillars containing the palato-glossus and palato-pharyngeus muscles respectively. It is hardly reasonable to urge that a more natural palate is the result of this interference.

The question of tension never enters into the flap operation. The treatment of cleft palates by bridging the gap with reflected flaps is merely an extension of the general surgical principle of overlapping which has come into recognition for dealing with abnormal openings anywhere in the body. Thus in the repair of a large ventral or umbilical hernia few people would dream of establishing continuity of the body wall by simply paring and suturing together the edges.

In all cases by operating early before the teeth erupt it is possible to get material from the under and outer surface of the alveolus, and from the palatal margins to provide a thick, loosely hung partition between mouth and nose. The advocates of the waiting policy will, I think, admit that there are a certain number of clefts of a very wide and complete nature that cannot be cured at an early age, at least except by a flap or by a direct attack on the bones. Of the latter class of operation I have no experience, and do not intend to speak. They state, however, that if the treatment of these wide clefts is delayed until their unhappy possessors are 3 or 4 years of age, the clefts will then be found to have become appreciably smaller. It is not difficult to see how this idea has arisen. There can be no doubt that a large number of infants so grievously handicapped do not survive the numerous trials of early life, and do not live to fall into the hands of the late operator. Not only are they as a rule below the average in constitutional vigour, but in addition they start life at a grave disadvantage with their feeding and breathing apparatus abnormal, and they succumb readily to gastro-intestinal and pulmonary disorders.

It has appeared to me that a surgeon who prefers to wait some years before operating takes a very limited view of the question. He looks at the actual cleft as a thing by itself, and neglects the attendant circumstances. His attention is so firmly fixed on the cleft, that he fails to note the nasopharyngeal catarrh, the rhinitis, the ulceration of the septum, the Eustachian catarrh with the attendant risk of middle ear and mastoid suppuration, the increasing oral sepsis, which are often present in the older cases, and which are a constant menace to the health if not the life of the individual. Careful attention may prevent these complications from occurring, but the average hospital mother is not very intelligent; her surroundings are not very conducive to cleanliness; nor if she wished could she give the necessary time to the task of feeding and cleaning the baby properly.

The more certain way of avoiding these troubles is by repairing the cleft at the earliest possible date, to enable the infant to breathe in the normal manner, to confine its food to the proper channel, and to improve its nutrition and resistance to all forms of infection.

Any improvement that does occur in children with extensive clefts who survive has been shown by Mr. Lane to be due to approximation of the sides of the alveolar arch because of the mal-development of the nasal fossæ from lack of function. A high Gothic-arched palate develops at the expense of the nose, and the edges of the cleft approach each other more nearly. Two wrongs, however, do not make a right, and this is one of the most potent arguments in favour of early operation. At birth the nose of a cleft palate baby has the same potential growth as the nose of the normal baby, and it is only when the power of healthy breathing is denied that the nasal fossæ fail to develop pari passu with the rest of the face, and that the normal dome-shaped palate becomes peaked.

Those surgeons who are in favour of delaying operation give as their chief object in closing a cleft palate a meritorious desire to render the speech more perfect than it otherwise could be. They state that deaths from inanition in the early days do not occur. On the other side it is held, and I think rightly, that the restoration of the mouth and nose to the normal is a life-saving measure. It is not possible for me to prove this with figures: statistics must come from the side of those who wait, and have an opportunity of watching.

The question of speech is a difficult one, and more factors enter into it than the purely mechanical. It has to be explained why an individual with, say, a small cleft of the soft palate will speak with a pronounced nasal voice, while one with a much more extensive cleft may have almost normal intonation. The flexibility and movement of the soft palate are of importance, but the shape of the palate is in even more definite relationship.

In some classes of the insane, who tend to have a highly arched degenerate palate, the nasal type of voice is not infrequently met with.

It is interesting to note also, in passing, that the mother of two children on whom Mr. Lane has operated, and who are shown at this meeting, has the character of voice associated with a cleft palate, and on examination she is found to have a complete but very highly vaulted roof to the mouth. In several instances I have found that the mothers of cleft palate children have abnormally high palatal arches themselves. Yet another factor in the causation of the peculiar speech, and one which is rarely mentioned, is the tongue.

If the child with a completely cleft palate has learnt to talk at all, the tongue will have developed its fine co-ordination and combination of movements necessary for speech. If, then, operation is performed, and the cleft is bridged across, the tongue finds itself in a much more restricted space, and has to adapt itself, which it frequently fails to do, to its new conditions. Earlier operation, at a time when the patient is still an infant, permits the tongue to become accommodated to its surroundings, and the development of its movements in connexion with speech takes place naturally. The shortness of the palate and the feeble musculature are congenital, and can be benefited but little by any operation. The best opportunity for the development of the muscles is provided when they are caused to functionate at as early a date as possible.

The most serious charge brought against early operation is the contention that the mortality is high. During the past two years, at the Great Ormond Street Hospital, out of 369 operations performed by Mr. Lane 22 resulted fatally. Of these 369 operations, 144 have been first operations on infants of 1 year of age and under, and of these 124 were under 6 months of age. Of the 144, 18 died, being a mortality of 12·5 per cent. I have already stated that no selection whatever was made. All cases, whose viability appeared assured, were submitted to operation, and many of them were on the downward path with malnutrition. Extent of deformity was not considered to be a contraindication, and in 53 per cent. of the cases the deformity was extreme.

In the Registrar-General's returns, the deaths of infants under 1 year of age over England and Wales in 1901 were at the rate of 10·9 per cent. It is evident that the operation involves a mortality of 1·6 per cent. over the normal death-rate. If the probable high mortality of severe cases unoperated on is taken into account, the death-rate cannot be considered excessive.

As to the causes of death, bronchitis and broncho-pneumonia involved the death of 11, whilst, in four, operations failed to relieve the marasmus, and death occurred four to nine days after operation. In three, the notes do not state the cause.

The operation itself is entirely devoid of hæmorrhage or shock: the infants sleep peacefully, and very rarely cry: they take feeds readily in one or two hours: they are not troubled with anæsthetic vomiting: they do not often require stimulants, and the minority regurgitate food through the nose after operation. The baby makes an excellent patient, and the comparison of its comfort with that of a child aged 3 or 4, operated on by the older method, with perhaps a tape passed round the stripped muco-periosteum, as suggested by C. H. Mayo, is in striking contrast. I have seen it stated that operation should be performed between the ages of 2 and 3, when the patient can be more easily kept under control. I venture to disagree with this entirely. I have asked sisters and nurses of long experience, who have told me that the child of 2 causes infinitely more trouble in nursing than the baby of a few hours or days old. The opinion of the parents as to the result of the operation is one well worthy to be considered. I have been at particular pains to ascertain this, and I know of no more grateful group of parents than those who have had their babies changed at an early date from unpleasant-looking monstrosities to respectable-looking members of Society. It is right, I think, to lay considerable stress on the feelings of the parents in the matter, for the mothers are, as a rule, particularly sensitive about their offspring's deformities, and always think that they are in some way to blame for the condition.

If, then, it is possible to cut short with safety two or three years of continuous worry, and two or three years of arduous and sometimes unsuccessful nursing, operation in earliest infancy is not only expedient, but demanded.

I have endeavoured to show as shortly as possible that, on broad humanitarian grounds alone, as a life-saving measure, early operation in cleft palate is justifiable. It is none the less desirable from the æsthetic standpoint also. Correct development of the face depends on the correct and symmetrical development of the bones, and this in turn depends on their being subjected, at as early an age as possible, to the strain of their proper function. An examination of Mr. Lane's cases shown to-day will, I feel sure, prove that the æsthetic demands have been fully met. The verdict upon any operation must be given by the results of actual practice, and I submit upon a review of these results it is abundantly proved, practically as well as theoretically, that the repair of a cleft palate should be undertaken at as early a period of life as possible.

Mr. W. G. Spencer desired to support some of the contentions which Mr. Lane brought forward, especially as the skilful operators on older cases of cleft palate who had previously spoken had not tried Mr. Lane's method, and therefore he did not think they were in a position to speak quite so dogmatically as if they had done so. His reason for following Mr. Arbuthnot Lane's practice had been that the babies brought to the hospital had been suffering from malnutrition and oral sepsis. After a short time in hospital their complications had disappeared, but in the absence of careful feeding on leaving the hospital relapse would have been inevitable. Surely the death-rate among the cases not operated upon early must be the most important consideration. Among fourteen cases in which Mr. Lane's method was followed three required the crushing inwards of the upper jaws after Brophy's method. The worst case was admitted with the upper jaws wide apart, suffering from thrush and suppuration in the submaxillary glands of each side. The palate and lips were closed at separate operations. The patient could not be exhibited, having but recently recovered from an attack of scarlet fever. A second patient necessitating Brophy's modification was exhibited. He was now aged 10; his age at the first operation was 3 weeks. He had a complete and wide cleft through the lip and palate. Also the upper lip was so defective that lateral slits were required at the angles of the mouth. He had now a hole through the hard and another through the soft palate, but fluids did not regurgitate. The line of teeth was irregular. Phonation was defective. The third patient requiring Brophy's modification, also exhibited, was now aged 7. His age at the first operation was 3 months. He had a single hare-lip and complete cleft palate. The jaws only required squeezing together, not fixing by wire. There were now two holes through the palate which admitted a probe. There was no regurgitation of fluid. The lines of teeth were not good. The child began to talk at 3 years. The phonation was defective.

Mr. Spencer's small experience, however, did not accord with Mr. Lane's, that there was a marked absence of mortality after the closure of the palate. Three patients out of the fourteen died after leaving hospital, although when sent out they were in a good state.

Case I.—Boy; age at operation, 24 days. Single hare-lip and complete cleft palate. Lane's operation. Result: Complete union of lip and palate. Death from pneumonia fourteen days after discharge.

Case II.—Boy; age at operation, 1 month. Double hare-lip and complete cleft palate. Lane's operation. Healed except for small gap in palate through which there was some regurgitation. Death from pneumonia three years after discharge.

Case III.—Girl; age at operation, 10 weeks. Double hare-lip and cleft palate. Lane's operation. Complete union of lip and palate. Death from pneumonia three weeks after discharge.

Mr. Lane had not met with a death from hæmorrhage. The only death among the fourteen was the last case, who died of recurrent hæmorrhage.

Boy: admitted when 10 days old for marasmus and thrush. A month later he appeared a thriving baby. Age at operation, 5 weeks. Complete cleft palate, lip and alveolar border normal. Closure by Lane's method, easy and satisfactory, with very little oozing, and at the end all hæmorrhage had ceased. Seven hours later the child's mouth was found filled with blood. The house-surgeon could not see any bleeding point and painted on adrenalin. Two hours later the mouth again filled with blood and the child died.

Two other cases were exhibited, both with good articulation. One, aged 3½, was operated upon at 4 months for a cleft in the hard palate and lip, not in the soft palate. The other, aged 5, was operated upon at 2½ years for a cleft in the soft palate.

Mr. LANE, in reply, remarked that he did not think it would be of any use for him to say any more on the matter. Those who had been in the habit of doing the old operation would continue to do it, while he believed that the younger men were taking up the new operation. As to what the result would be he had no doubt whatever. There was only one point on which he would say a few words, because he knew something about it—namely, the question of mortality. At the early period when he did the operation he had about 100 children applying for admission to the hospital, and the Resident Medical Officer, when writing to six of them at a time, would calculate on getting only four or five to come up, as he knew that in all human probability one or two out of those six had died in the interval since application was made for admission. He wished some of the old resident medical officers from the Children's Hospital, Great Ormond Street, were present, as they could state those facts much more clearly. The mortality among severe cases of cleft plate was enormous. An endeavour had been made to trace the mortality; some speakers seemed to have had no difficulty in tracing the absence of mortality. He must leave the matter in the hands of surgeons, who must decide for themselves as to the best methods of treatment of these cases.

Surgical Section.

June 13, 1911.

Mr. RICKMAN J. GODLEE, President of the Section, in the Chair.

On the Division of the Posterior Spinal Nerve-roots: (I) for Pain, (II) for Visceral Crises, (III) for Spasm.

An Address Introductory to a Discussion on the Subject.

By ERNEST W. HEY GROVES, M.S.

SINCE Abbe, in 1888, first divided the posterior spinal nerve-roots for the relief of intractable pain, the number of cases recorded of this operation have been remarkably few, but in quite recent years Foerster has given a new stimulus to the subject by his suggestion that the scope of the operation may be extended to the treatment of such diverse conditions as visceral crises and spastic contractures. With the exception of isolated cases reported by Bennett, Horsley, Head, and Morton Prince, there has been practically nothing written on this subject in England. And yet, from both the physiological and the clinical standpoints, it is one of great interest and importance. For, on the one hand, it demonstrates, and to some extent elucidates, the complexity of the problems of pain, whilst on the other hand, it offers a cure for certain disorders which are otherwise almost intractable. Strangely enough the division of the posterior spinal roots has given the least satisfactory results in those very cases where we should have expected it to have been the most efficient—i.e., in the relief of pain—whereas in symptoms with which it is much more remotely connected it has never failed to yield some measure of success. I have to report four cases of posterior root resection, and I will preface my remarks on each subdivision of the subject by a short description of the personally observed cases which illustrate it.

SECTION I.—FOR PAIN.

Case I.—F. H., aged 50, used to be the secretary of an insurance company before his illness. As he has always been of very methodical habits, and has kept a diary, the history of the onset of his symptoms is exceptionally precise and reliable.

History: January, 1896—first noticed the loss of sensation in the left foot. February, 1896—first had pain in the left foot. September, 1897—the left foot swelled suddenly. November 1897—first noticed lameness; occasional pain noted in the right leg and foot. March 1898—swelling of the left knee. May, 1898, till August of the same year, was an in-patient at Queen's Square Hospital for Paralysis, under Dr. Ormerod. There, for the first time, he had difficulty in micturition. For the next ten years the pain continued without much alleviation, occurring in severe paroxsysms once or twice a week. It has always been most severe in the left leg from above the knee to the extremity of the foot, but the right leg has also been the seat of a good deal of pain. Since 1898 he has only been able to walk with the help of crutches, because of the pronounced ataxia and the deformity of the left foot and knee.

Condition prior to operation (August, 1909): He is a heavily built man, well nourished, and perfectly healthy except in respect to the nervous system. Left leg: The knee is much swollen, measuring 22½ in. in circumference (as compared with the right, which is 15 in.); there is much bony thickening and free fluid with several large loose bits of bone; an extreme valgoid position is present amounting to about 30°, whilst there is nearly 15° of lateral mobility. The left foot is also in the condition typical of a Charcot's joint, being flattened, everted, and deformed. The pain which is present in both legs is much worse in the left, feels as if in the bones, is liable to severe exacerbations about every third day, which last about seven to eight hours, generally at night, and is more relieved by antipyrine (30 gr.) than by any of the other innumerable things which he has tried. The sensibility to tactile, painful and thermal stimuli is greatly impaired in both legs. This impairment of sensation in the left leg extends almost to the groin, in the right to the knee. The loss of sensation amounts to a dulling of perception, with great delay, but not absolute anæsthesia.

Laminectomy (August 16, 1909): The ninth dorsal spine was first removed in order to allow the turning up of a large osteoplastic flap containing the tenth dorsal to the first lumbar laminæ inclusive. After opening the dura it was found necessary to take away the second lumbar lamina as well, in order to gain sufficient view. About 1 cm. was resected from each of the following posterior nerve-roots on the left side—viz., the L3, 4, 5, S1, 2, 3. The resection was done as close as possible to the junction of the roots to the cord. For three days after the operation he had very severe pain, chiefly in the left foot, which required $\frac{1}{4}$ gr. to $\frac{1}{2}$ gr. morphia, to be repeated twice a day. The bladder had to be emptied by catheter from this time onwards until he was able to get up.

Excision of the left knee-joint (August 30, 1909): The disorganized joint was excised, many large, loose pieces of bone removed, and the femur and tibia united by means of a mortice joint. The operation, which lasted one and half hours, was borne well, *without any anæsthetic whatever*, either local or general. After it was over he was a little pale and complained of feeling tired, but otherwise he had no sensation or discomfort from the operation. The wound healed by first intention with a good, firm, fibrous union between the bones. Osseous union was prevented by the strong involuntary contractions which were constantly taking place in the muscles of the leg.

Recovery from the operation was uneventful except for an unfortunate attack of suppurative parotitis on the left side, which required an incision. He returned home on October 21, 1909. He had a small pressure sore on the left heel which was somewhat slow in healing. On two occasions the actual cautery was applied to this without causing him any discomfort.

Present condition (February, 1911): I have asked the patient to give his own opinion as to his present condition, and the benefits he has derived from operative interference. He writes: "The pain in the left leg has certainly been much less severe than previously. There has, however, been pain, though more of the gnawing than of the shooting description. I suffer enough, though, still to make life a doubtful blessing, and without antipyrine and heroin I hardly think I could go on with it. I do not think I would undergo the operation again, but if I had to do so would have it done *on both legs.* It has not affected my locomotion at all. If the pain I experience in my right leg were modified as it has been in the left, it would not then be unbearable. I only wish that the nerve operation could have been as successful as that of straightening the leg was."

From an objective point of view, the left leg has lost sensation completely, right up to the fold of the groin and round the iliac crest (fig. 1). Tactile, painful, and thermal sensations are all quite absent over the whole of this area, but that of tactile sensation is not quite so extensive as of the other two forms, by about 2 in. The limb is well nourished, but he has had on more than one occasion some superficial ulceration between the toes, which has healed readily under ordinary treatment. He wears a leather splint for the knee.

Case II.—R. S., aged 39, a married woman with four children.

History: In 1907 she had a severe attack of puerperal pyæmia, which necessitated several operations for the opening of abscesses, the largest of which was over the left iliac crest, and was associated with partial necrosis of that bone. In the beginning of 1909 she had phlebitis in the left leg, with a large indolent ulcer over the shin. This was the beginning of the pain, which has continued ever since. By the autumn of the same year the pain had become so unbearable that she begged to have the limb amputated.

First operation (September 1, 1909): Laminectomy. Division of posterior spinal nerve-roots. The laminæ of the last two dorsal and the first lumbar vertebræ were removed, and the roots of the fourth and fifth lumbar, and the first, second, and third sacral roots on the left side divided. She had a good deal of pain in the back and left leg for about a fortnight after this operation, but then was much easier for a month. However, as very little loss of sensation could be detected in the left leg it was feared that the division must have been incomplete. This was confirmed by a speedy return of the pain in all its former intensity.

Second operation (October 8, 1909): The lumbar enlargement of the cord was exposed by opening the former scar, cutting through very dense cicatricial tissue. This gave a much more bloodless field of exposure, and it was found that in every case some of the nerve filaments had been left to each divided root. These were now completely resected. After this the leg was absolutely anæsthetic to within a hand's breadth of the groin. No painful, tactile, or thermal impression could be felt in any part of this area, nor had she any idea of the position of the limb. On first attempting to walk the leg was thrown about in the most wildly ataxic manner, but this slowly improved in time.

After-history: For some few weeks she had relief from pain, but it then began to be as bad as ever. The pain nearly always began at night and continued for many hours, making her roll about in agony and threaten to take her life. She said she felt as though the pain was driving her out of her mind, and this fear proved to be well founded, and she had to be removed to the Fishponds Asylum, where she remained from April 22 to October 3, 1910. For about a month of this time she was wildly maniacal, and it was necessary to place her in the padded cell. Her mental condition slowly improved, but the pain in the leg never wholly left her. On returning home she became much worse, and I admitted her again to the hospital for observation in January, 1911, but again she had to be taken to the asylum, where she now is. During both attacks of insanity the pain in the leg is better, then after she has been discharged from the asylum—where she gets good food and fresh air—and goes to a poor home, where she has every encouragement to ill-health, the pain begins again.

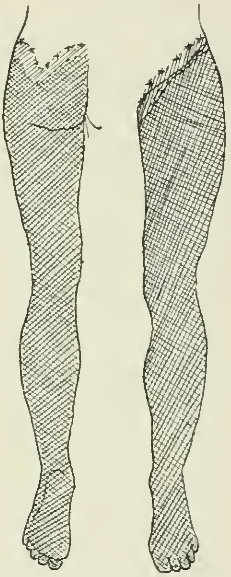


Fig. 1.

Leg from Case I showing areas of anæsthesia and analgesia. The latter is the larger, and is delimited by x x x.

Judging from a priori arguments we should certainly have supposed that in the section of the posterior spinal roots we have a certain means of curing peripheral pain, and yet we have the very remarkable phenomenon of complete and lasting analgesia associated with a continuance of a subjective feeling of pain. And it is easy to satisfy oneself that this result is the general rule and not the exception in similar instances recorded in the literature.

Among the sixteen cases collected by Jacoby [10] in 1907 there are only six in which the pain is said to have been completely cured. In six others there is said to have been great relief, and of the remainder there is a significant silence about the after results.

Knapp [11] has recorded two cases: (1) A man, aged 40, alcoholic; in 1890 the leg was amputated after some accident; then during the next five years he had several excisions of the stump and of the bulbous nerve-ends for pain, and finally in 1896 the posterior roots of the sacral plexus were divided, but he died of shock. (2) A man, aged 25; amputation in 1898 of the right arm below the elbow on account of a severe crush: great pain in the stump required four operations for the removal of bulbous nerve-ends. The nerves were injected with osmic acid, the limb was amputated higher up, but with no avail. In 1899 he was nervous. hysterical and generally wretched. In 1902 the C6, 7, 8, and D1 right posterior roots were resected. He was free from pain for some months and then became as bad as ever. Two years later he developed spastic paraplegia and at that time and up to 1907 the original pain was as severe as ever, although the cord was re-exposed and the division of the roots assured.

Chavannaz [5] relates the case of a man aged 68 who had severe intercostal neuralgia on the left side from the axilla to the mamma, which persisted in spite of excision of the corresponding intercostal nerves. The posterior roots D1—4 on the left side were resected, but the result was only some amelioration but not cure.

Hildebrand [9] gives an account of three cases: (1) Woman, aged 48; luetic neuritis of arm, which was relieved by no drugs; the roots of seventh and eighth cervical nerves were cut (February 23, 1910); relief for a time, but the pain soon returned. A second operation on April 16, 1910, divided the sixth cervical and first dorsal, but sudden death occurred at the end of the operation from heart failure. (2) Man, aged 62. October, 1909—Pain in right hip, thigh and leg with some muscular wasting; arterio-sclerosis, œdema, and cyanosis of feet. Operation, June 1, 1910: Laminectomy of L1—5. Resection of right posterior roots. Pain temporarily relieved, but by July 15, 1910, the pain had recurred as bad as ever in the right leg. (3) Woman; operated on seven years before, for breast cancer. Metastatic growth in left brachial plexus. Severe pain in arm. Operation July 16, 1910: Laminectomy with resection of C5—8 D1—2, good recovery. Patient declared she still had pain. This was considered due to the fact that she still craved for morphia.

These are all the cases of this kind that I have been able to collect from the literature, and they are enough to show pretty clearly what the general results have been. Some analysis of the details of the twenty-four cases included in Jacoby's list and those I have added may serve to emphasize the points of importance.

Sex.—Seventeen were males and six females; in one the sex is not mentioned.

Age.—In nineteen cases the age is given and the average is 41. The oldest was 68 and the youngest 15.

Exciting Cause.—This is mentioned in ten cases: Tabes, one; cancer of the brachial plexus, two; pyæmia, one; trauma, four; syphilitic neuritis, one; athetoid paralysis, one.

Region Affected.—Arm, fifteen; leg, six; intercostal areas, three.

Immediate Result.—Death occurred in, four; recovery in, twenty.

Late Results.—Complete cure of the pain, six; moderate or partial relief, six; failure to relieve the pain, three; not mentioned, three; died within a few weeks (the two cancer cases), two.

Nervous and Mental Sequelæ.—Moderate degree of Brown-Sequard paralysis, one; spastic paraplegia (occurring some years after an operation on the brachial roots), one; morphia habit (acquired before the operation), three; insanity or other neurosis, three.

The point of the greatest interest in these cases is the failure of the resection of the posterior nerve-roots to cure the pain in the corresponding areas, in about three-quarters of their number. A certain number of these cases may be accounted for by supposing either that not enough of the roots had been divided, or that the division had been incomplete. But probably the majority are not susceptible of this explanation, and those I have related are among such, for in both these there was the most absolute loss of every kind of sensation, so that one patient could have his knee-joint resected without an anæsthetic, and yet pain is still referred to the same area. It is easy to understand that the relief from pain would not be immediate, or that it might for a time be increased, as it usually is, but when the ascending tracts of the cord have had time to completely degenerate, it is hard to understand where the pain comes from. In the case of facial neuralgia, the removal of the sensory ganglion or the division of the root of the fifth cranial nerve proximal to that ganglion causes immediate and lasting freedom from pain. Why is the result different when the neuralgia is in the area supplied by the spinal nerves? In some cases, such as the second I have related, the pain is evidently of psychical origin, and in her case it alternates with absolute insanity.

From a practical point of view it is clear that we must give a very guarded prognosis as to the results likely to be attained by resection of the posterior roots for pain. But it is certainly a more rational and more hopeful proceeding than that of amputating a limb, for the former may cure, it often relieves and it does not mutilate, whilst the latter never cures, although it involves the sacrifice of a member.

SECTION II.—FOR VISCERAL CRISES.

Case III.—O. A., a labourer, aged 34. Symptoms: Pain, emaciation and vomiting.

History: Had always been healthy until two years ago, when he first began to suffer from vomiting and loss of flesh. For the last few months he has had daily lavage of the stomach, which has given him some relief. I saw him with Dr. Grier, of Mevagissey, and we diagnosed his condition as being due to some form of pyloric stenosis. I admitted him to the Bristol General Hospital on August 1, 1910.

Condition: The most notable thing about him was his great emaciation; he seemed to be a walking skeleton, weighing only 85 lb. He vomited several times a day, and had a great deal of epigastric pain. The stomach was dilated and displaced downwards to one finger's breadth from the navel, and splashing sounds could always be elicited over the organ.

First operation (August 2, 1910): Gastro-enterostomy. The stomach was found to be dilated and prolapsed so that the pylorus appeared to be kinked. There was no evidence of ulcer or new growth. The usual posterior gastro-enterostomy was performed. He derived no benefit from this proceeding, but was sick as before. About a fortnight after he complained of severe shooting pains in his legs, which suggested the idea that he might have tabes. Further examination by Dr. Carey Coombes confirmed this. There was complete absence of knee-jerks, Argyll-Robertson pupils and Romberg's sign. The pain which we had mistaken for indigestion was in reality a girdle pain, due to the tabes.

Second operation (August 26, 1910): Laminectomy; resection of the posterior dorsal roots. Through a median incision the laminæ of the fifth to eighth dorsal vertebræ were removed and the seventh to the tenth dorsal posterior roots resected on both sides. (*See* fig. 2.)

After-history: For a few days he suffered an exacerbation of his pain, but this quickly disappeared and he was free from vomiting except on one or two occasions shortly after the operation. He took his food well, but although on full diet he never gained flesh. This was largely due to the fact that he had several large loose actions of the bowels daily, which no kind of medication had any effect in arresting. He died quite suddenly on September 21, although at this time he was well enough to get up each day and go out of doors.

Autopsy: This was performed by Professor Emrys Roberts. Nothing abnormal was found, apart from the condition of the nervous system and the extreme wasting. The gastro-enterostomy junction was firmly united and the laminectomy wound was soundly healed.

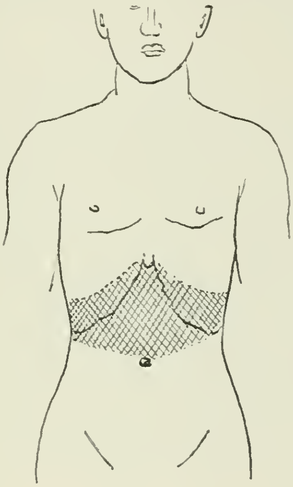


Fig. 2.

The shaded area shows the loss of sensation in Case III after the operation.

Structural changes in the cord: I am greatly indebted to Dr. Rendle Short, the senior demonstrator in physiology, for a most careful and exhaustive examination of the cord. He could not find any evidence of tabes in the lumbar region. The dorsal and cervical regions show the degeneration of the posterior columns which results from the resection of the posterior nerve-roots in the dorsal region. Many of the cells of the anterior horns of the grey matter in the dorsal region show marked chromatolysis.

Note on the Degenerations and Cell Changes resulting from Section of the Posterior Nerve-roots in Man, by A. Rendle Short, F.R.C.S.

The material for this investigation was obtained from a patient in whom Mr. Hey Groves had four weeks before death divided the posterior nerve-roots from the seventh to the tenth dorsal on both sides for paroxysmal abdominal pain diagnosed as gastric crises of tabes dorsalis. The patient died of asthenia, weakened by an attack of diarrhœa.

Unfortunately the brain and spinal cord had been put into unsuitable hardening materials before they reached me, and had moreover been allowed to become dry for a few hours, which spoiled the extreme periphery of the white matter of the spinal cord. A more serious drawback was that I was unable to obtain any good results with either Marchi's or Donaggio's differential stains for degenerated white fibres, as the material was too hard for Müller's fluid to penetrate.

Degenerated Tracts.—For the reason stated, negative results were obtained with Marchi's method and Donaggio's three hæmatoxylin methods. Nor did the Pal-Weigert stain bring out any degenerated fibres anywhere, presumably because the nerve-roots were divided too recently. There was, however, a perfectly definite wedge-shaped degeneration in the middle of the posterior columns in the cervical region shown by Van Gieson's stain, and also by aniline blue-black and by toluidin-blue (fig. 3). The base of the wedge abutted upon the posterior surface of the cord, and the apex did not quite reach the grey commissure. This area of course corresponds to the distribution of the long ascending fibres of the column of Goll, cut off from their trophic centres in the posterior root ganglia by the operation. As might have been expected, no descending degenerations could be traced. Sections of the various regions of the spinal cord, medulla, pons and isthmus, stained by the Pal-Weigert method for any old-staining degenerations, showed nothing abnormal. This discounts the suggestion that the original complaint was advanced tabes dorsalis, although it is still possible that early tabes was present without recognizable changes in the posterior columns.

Cell Changes.—A large number of sections from the cervical region, far above the divided nerve-roots, and also from the lower dorsal region, in the midst of the divided nerve-roots, were stained by the toluidine-blue modification of Nissl's method, and examined for cell changes. A few sections were also examined from the lumbo-sacral region. These presented no abnormalities. In the cervical region, the nerve-cells of both the posterior and the anterior horns were normal in appearance and staining reactions. Nissl granules were abundant in all the cells. I only found one cell in a large number of sections, which was perhaps somewhat deficient in granules. In the dorsal region, however, opposite the severed posterior nerve-roots, there were marked changes affecting more than half the nerve-cells, both of Clarke's column and of the anterior horn. In Clarke's column nearly all the cells were affected, to a varying degree. The nucleus was usually still present, but in some cells appeared to be lost. There were not many cells totally devoid of Nissl granules, but in most of the cells the granules formed a narrow fringe at the edge of the cell, often at one pole only. In the cells of the anterior horn the changes were less universal, but quite as marked in many cases. Less than half the nerve-cells were normal. In a good many there was an excessive deposit of fat, and only a few peripheral granules remained. In quite a large number the changes were extreme; the granules were totally absent, and the nucleus very excentric or entirely lost. These changes, especially those in the anterior horn cells, are interesting and remarkable, and I am not aware that they have yet been described in man. They are, of course, well known to take place in any nerve-cell when the axon coming from it has been cut, and therefore division of the *anterior* nerve-roots would undoubtedly have led to a similar disappearance of the Nissl granules and excentricity or loss of the nucleus. But in this patient the anterior nerve-roots were not damaged, only the posterior being divided. This was perfectly obvious even in the specimen after removal from the body. It is probable that when all the posterior nerve-roots issuing from neighbouring spinal segments are cut, the number of nervous messages received by the anterior horn cells is reduced very much. The

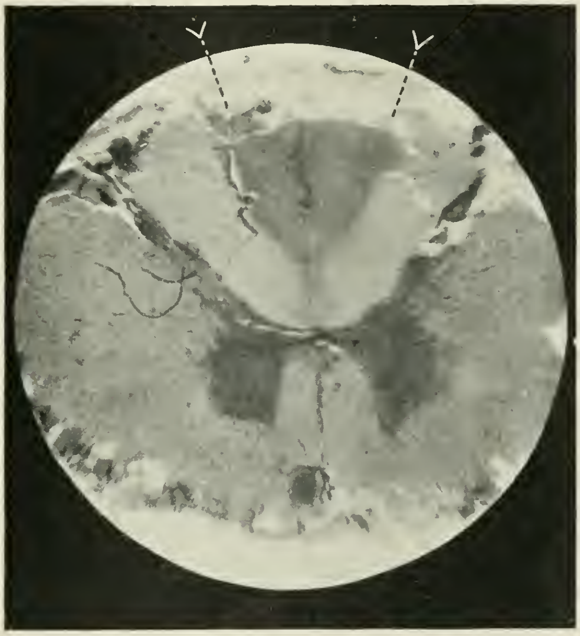


Fig. 3.

Section of cervical region of cord in Case III, showing area of degeneration in posterior columns, v . . . v.

changes, summed up under the name of chromatolysis, are the expression in the nerve-cell of the atrophy of disuse. It will be noticed that some normal cells still remain in the affected regions. The chromatolysis in the cells of Clarke's column is no doubt due to the same cause. It is interesting to notice that there was no degeneration in the fibres of the dorsal cerebellar tract, arising from Clarke's column. It is well known that loss of the Nissl granules is not a nerve-cell's death warrant, but recovery is probably impossible when the nucleus also is lost. The marks of degeneration in the anterior horn cells are accompanied by definite functional symptoms in the muscles they supply. In the particular case in question, where the lower dorsal roots were divided, it would have been difficult to demonstrate this. When the posterior nerve-roots from a limb are cut in an animal, there is nearly always a marked disinclination to use the corresponding limb, although there is not actual paralysis present. Of course, the limb is anæsthetic; there is also a loss of muscular tone in the limb. This was well seen in several of Mr. Hey Groves's cases, and indeed a principal indication for the operation of severing the nerve-roots is to reduce excessive tone, in spastic cases. The reduction of spasm and the chromatolysis are both the evidences of diminished activity on the part of the nerve-cells.

Some very interesting observations were published so long ago as 1898 by Warrington [14], of Liverpool. He divided various posterior nerve-roots in cats, and also in one case at least in a monkey, and was able to demonstrate exactly the same changes in the anterior horn cells which are described here as occurring in man. In his cats, as in this patient, the nerve-cells in the anterior horn at the level of the injury were not all affected. Some of them were normal, others showed some dissipation of the Nissl granules, and others, again, had lost all their granules and sometimes even the nucleus itself. The cats showed the usual symptoms described by many observers, that is to say, they hopped about on three legs, preferring not to use the anæsthetic limb. When this limb was used, its movements were very ataxic. The reflexes were lost, and the muscles were flaccid. These observations have been confirmed in Germany by Brauenig [3].

The idea that the gastric crises of tabes might be cured by division of the posterior nerve-roots of the spinal segments governing the stomach is due to Professor Otfrid Foerster, of Breslau, who with Professor Küttner [7] carried out the first operation of this kind in 1908. His conception was that the visceral crises were due to an exalted reflex action, and that in order to inhibit this it would be necessary to break the nervous arc upon which it depended. As Foerster's original paper is readily accessible, I will refer to it for the fuller discussion of the theoretical aspect of the question and content myself with giving a brief abstract of his case.

E. G., aged 47, an architect, had had syphilis for which he had been treated in 1905-08. In 1902 the first attacks of vomiting began and continued until 1908. Each vomiting attack lasted for about a fortnight and was followed by an interval of three weeks' freedom. But towards the latter part of the time named he had only a few days' freedom from vomiting each month. Intense pain accompanied the sickness, and he brought up about 1½ litres of bilious fluid every twenty-four hours. He had wasted to a skeleton, and had yielded to the morphia habit until he was taking ¾ grm. daily; Argyll-Robertson pupils, lightning pains, great hyperæsthesia and distension of the abdomen, and paræsthesia in the hands and feet. Every kind of medicinal and electrical treatment had been tried without result. First operation (December 31, 1908): Laminectomy of the fifth to the tenth dorsal vertebræ, dura not opened. The pain and vomiting were much worse after this stage. Second operation (January 1, 1909): Resection of the sixth to the tenth posterior dorsal roots on both sides. After this the pain and vomiting absolutely ceased. The patient could eat anything and rapidly gained weight, increasing by about 4 lb. each week. The patient died nine months after the operation, of phthisis, and during this time he had no return of the gastric crises. (I am indebted to Professor Foerster's kindness in sending me the last-named fact by a private communication, which also mentioned several details about the two following cases of Foerster and Küttner.)

Foerster and Küttner's second case was a man who had become greatly weakened by the morphia habit. He stopped breathing during the operation, and artificial respiration had to be performed. He died a few hours after.

Foerster and Küttner's third case was a man who had had two previous operations performed on the stomach, including a gastro-enterostomy. The sixth to the eleventh dorsal roots were divided on both sides and he has completely recovered from the gastric crises, so that at the present time, one and a half years after the operation, he is in good health.

Bruns's and Sauerbruch's [4] case: Patient was a mason who for several years had had gastric and intestinal crises of increasing severity, so that before the operation he often had fæcal vomiting. The seventh to the ninth dorsal roots were divided on both sides. The result was excellent at first, but six months later he suffered a relapse, the stomach and rectal crises being as bad as ever. This was attributed to an alcoholic excess, and he subsequently recovered, so that he is now able to resume his work as a mason. (Private information from Professor Foerster.)

Moskowicz's [12] first case: A man, aged 46, who for three years had had gastric crises. The resection of the posterior roots was followed by a cure which lasted for six weeks, the date of the report.

Moskowicz's second case: This case died two weeks after the operation from sepsis of the wound.

Thomas's and Hall's [13] case: A man, aged 32. Gastric crises of eight years' duration; weight had dropped to 100 lb. He had the typical symptoms of tabes. In 1905 he underwent a laparotomy by which a few gastric adhesions were found and freed, but this caused no relief. On March 3, 1910, the seventh, eighth and tenth dorsal roots were cut. On two occasions after the operation he vomited, but he had no further pain or gastric crises. The relief had lasted till July of the same year, but he had not gained in weight, nor had he broken himself of the morphia habit.

Hahn has had one very successful case, and Gulicki several, but I have not been able to obtain the reports of these.

Of the eight cases here collected, the notes of several are too meagre to gain much information from, being merely given in congress reports. But I think that there is enough evidence to prove that the operation is the most radical means of arresting the severe gastric crises of tabes that we have at our disposal. It is too soon yet to say whether the cure is going to be a lasting one. Foerster's third case has remained well for one and a half years, whilst Bruns's case had a severe relapse six months after the operation, but has since again recovered. Two of the eight cases died as the result of the operation, one of shock and one of sepsis. My case died four weeks after the operation from inanition and diarrhœa, but whether this was because of the operation or in spite of it, it is impossible to say. In all these cases the condition has been allowed to go on for several years (two to eight), and the patients have become extremely ill from the combined effects of pain, emaciation, and morphia. Such patients are in the worst possible condition to recover from a severe operation, and the actual results obtained as regards mortality are better than might have been expected. It is quite clear, however, that if the value of the operation is recognized, cases will be sent to the surgeon much earlier, and in this event there is no reason to regard the operation as a particularly dangerous one. No case ought to be allowed to go for more than twelve months, with gastric crises of increasing severity, which resist the physician's art, without offering him the chance of cure afforded by this radical procedure. Obviously the patient ought not to be allowed to have morphia or any of the allied drugs, and the operation should, in my opinion, be considered as a much less dangerous alternative than the morphia habit.

Before leaving this part of the subject, it is of interest to refer to two operative procedures of a nature closely allied to that of Foerster. The one is described by Cotte [6]. He had a patient who for five to six years had had very severe gastric crises with other signs of tabes. On January 28, 1906, the abdomen was opened and the stomach found to be normal in size and appearance. The solar nerve plexus was freed from the cœliac axis and stretched. All pain and vomiting were completely relieved after this operation, and the relief had lasted until the time of the report, three months later. It is difficult to believe, however, that a proceeding of this kind can possibly be of more than temporary alleviation, but it is worth bearing in mind for those cases where the abdomen has been opened on account of intractable vomiting and where no gross disease of the stomach is found. The other is contained in a suggestion by Franke [8] during the German Surgical Congress when Foerster's operation was under discussion. He states that it is possible to sever the posterior dorsal nerve-roots by avulsion of the intercostal nerves without opening the vertebral canal. In the absence of detailed information it is impossible to express an opinion as to the feasibility of this idea, but it would certainly be an excellent substitute for Foerster's operation in those cases of extreme debility where the latter would be dangerous, if it could be carried out efficiently.

SECTION III.—FOR SPASM.

Case IV.—L. R., girl, aged 16. She was sent to me by Dr. Toye, of Bideford.

History: She has had spastic hemiplegia since birth, the upper limb having been always more affected than the lower. Fig. 4 is from a photograph taken at the age of 5, and shows well the contraction of the hand and arm.

Condition, May, 1910: She is a well-nourished and very intelligent girl, perfectly healthy except for the paretic condition. The right arm is never used, but held at the side with the forearm strongly pronated and the hand flexed. There is, however, no muscular wasting. All active movements are clumsy and uncertain, whilst passive movements provoke a strong and painful spasm. This is particularly the case with the muscles of the hand, and any attempt to straighten the fingers or wrist causes them to be tightly folded back so that the tips of the fingers touch the front of the forearm (fig. 4). The movements of the right leg are much less interfered with, but there is a spastic contraction of the calf muscles which makes her walk on the toe in a position of talipes equinus.

First operation: In view of the fact that the principal disability of the arm was due to the over-action of the muscles supplied by the median nerve, this nerve was exposed at the bend of the elbow, and forcibly stretched. The right tendo Achillis was divided to relieve the spastic equinus. A certain amount of improvement followed these operations, especially in the leg, the walking being greatly facilitated. The relaxation of the arm spasm was so temporary, however, that within two months the hand was as bad as ever.

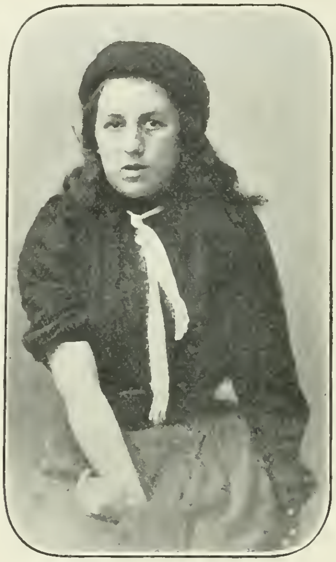


Fig. 4.

Case IV, before operations. Hyper-pronation of forearm and hyper-flexion of fingers.

Second operation (December 20, 1910): Posterior root resection. The area over the cervical spine was thoroughly infiltrated with a large quantity of a solution of adrenalin chloride (1 in 50,000). This was repeated at intervals until the actual opening of the dura, and was of the utmost value in securing a bloodless field of operation. It not only greatly shortened the operation, but made the observation of the roots, when exposed, very easy. Only the right halves of the fifth, sixth, and seventh cervical vertebræ were removed, and through this it was quite easy to resect four roots. I did this for what I took to be the fifth to the eighth posterior cervical roots on the right side, but as subsequent observation showed, I must have cut C6, 7, 8, and D1. On the following day the hand was absolutely flaccid, and afforded the most convincing demonstration of the influence of posterior root resection in abolishing spasm. About a week later, although there was no return of spasm the muscles had regained their tone, and it was evident that the long-continued contraction of the forearm muscles had caused a shortening of these, relative to the length of the bones. Either the wrist or the fingers could be extended separately but not at the same time.

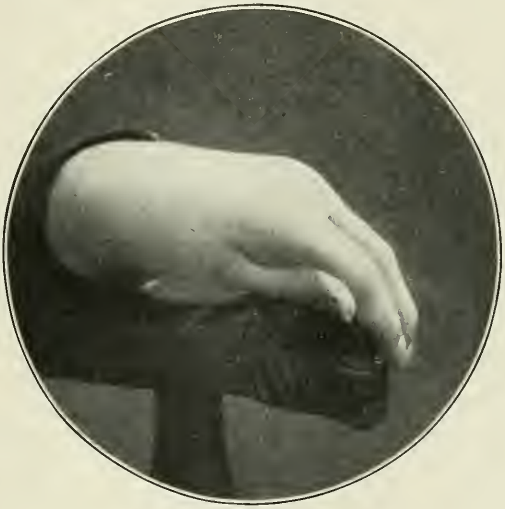


Fig. 5.

The hand in Case IV after the operation.

Third operation (January 3, 1911): A portion of both radius and ulna, measuring $\frac{3}{4}$ in., was removed, after cutting with a Gigli's saw. The bones were brought together over medullary pegs without any other sutures.

Present condition : The forearm is kept on a splint in a position of slight dorsiflexion of the wrist, and is taken down three times a day for massage and movements. She has already acquired some use in the fingers, being able feebly to grasp a pen. The whole hand and fingers can be fully extended, but the thumb is still strongly adducted, owing to the shortening of the intrinsic muscles (fig. 5). Movements at the elbow and shoulder can be executed within a small range, which is slowly increasing. If her present rate of improvement continues I hope that within three months she will be able to leave off the splint and use the hand for simple things, such as dressing herself and feeding. Sensation is unimpaired, except over an area extending along the ulnar side of the forearm including the little and ring fingers, and reaching above the elbow for 2 in. The protopathic loss is slightly greater than the epicritic, the margins of the areas for the two forms of sensation being about 1 in. apart above the elbow and ½ in. in the forearm (fig. 6). I saw this patient again in April of this year. The absence of spasm was maintained, but the return of motor power was disappointingly little. The deformity is cured, but the hand still remains almost useless.

Case V.—F. P., single woman, aged 33.

Past history: She is said to have had a stroke when 15 months old, and ever since she has been weak in the left arm and leg. For some years past she has been an inmate of the workhouse, and she was admitted to the Cossham Hospital on March 6, 1911.

Present condition: She is a well-nourished and intelligent woman, with no disease except the spastic paralysis of the left arm and leg. There is slight wasting and loss of mobility on the left side of the face. Left arm: There is some general paresis affecting chiefly the muscles below the elbow; this is accompanied by spasm with coarse athetoid movements of the hand and fingers. Left leg: There is slight muscular wasting below the knee, the foot is held in a position of extreme equinus, and on attempting to correct this, very marked clonic contractions are set up which are very painful and make her cry; on attempting to walk she holds the foot in extreme plantar flexion, and is often thrown to the ground by the onset of the clonus; it is this liability to be thrown down that makes her unable to earn her living as a domestic servant. The left knee-jerk is much increased. Babinski's sign is absent. The neuro-muscular system is perfectly normal on the right side, except for a slight increase in the knee-jerk. The whole of the left side of the trunk and limbs presents a marked anæsthesia and analgesia.

Operation (March 13, 1911): Laminectomy of the eleventh and twelfth D. and the first and second L. Resection of the L2 and 4, S1 and 3 posterior nerve-roots. The blood-pressure only fell from 140 mm. to 128 mm. after the nerves had been divided, and this was not apparent at the time of the division, but only when the wound was being sewn up.

After-history: She made a good recovery, except for some pain in the wound, and a slight attack of cystitis which followed temporary retention. On March 16 the note is: "The muscular rigidity of the left leg is greatly improved, the foot can be dorsiflexed without causing any clonus, and the knee-jerk is absent." But unfortunately this improvement was only of temporary duration, and on April 25 the liability to clonus had returned. She had at that date a good deal of ataxia in the movements of the leg, which made her feel that the operation had left her worse off than before. This case, though a failure from the patient's point of view, is very interesting and instructive from that of physiology. Professor Foerster gives the L5, S1, S2, as being the segments of the cord concerned in the control of the plantar flexors of the foot; and of these three I only divided the middle one—viz., the S1. This accounts, I think, for the very short duration of the relief of the spasm. The patient refused, however, to have anything further done.

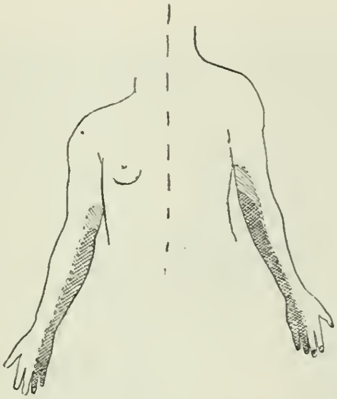


Fig. 6.

Showing areas of loss of tactile and pain sense in Case IV after the operation. The area of loss of pain is the larger.

Case VI.—J. C., a labourer, aged 48.

Past history: He had been in the Army and had served abroad in Egypt. Had had a sore on the penis, but does not know whether it was followed by any other signs of syphilis. Was quite well until three years ago, when he began to feel weakness in the legs, and pain. Both these symptoms became steadily worse, and for the last year he has been an inmate of the workhouse infirmary. Admitted to the Cossham

Hospital on March 9, 1911, when his condition was as follows: He is thin and weak, but there is no organic visceral disease. The right eye has a cataract of three years' formation; the left eye has good vision, but the pupil is small, reacting to light, but not to accommodation. All the muscles of the trunk and limbs are flabby and wasted. The left arm is very rigid, being held with the elbow flexed at a right angle. The legs are kept flexed at the hip and knee-joints and strongly adducted, so that he has to wear pads between the knees and ankles to prevent these parts becoming rubbed into sores (fig. 7). Any attempt at extension or abduction of the legs causes him the most intense pain. He finds that sitting in a chair is the most endurable attitude, but during the past year the pain has been so severe as to allow him very little sleep.

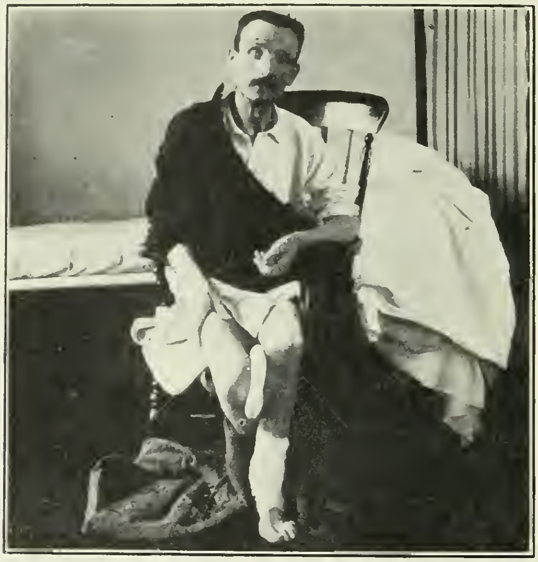


Fig. 7.

Case VI, before operation, wearing a pad between the adducted knees.

Operation (March 11, 1911): Laminectomy of the D11 and 12, L1 and 2 vertebræ; resection of the L1, 3, 5, S1 and 2 (left), L1 and 4, S1 and 2 (right), posterior nerve-roots. At the beginning of the operation the blood-pressure was 108 mm., and at the end it was 102 mm.; and it was observed that the division of the nerves caused no more drop in the blood-pressure than any other stage in the operation.

After-history: The pain and spasm have completely disappeared from both legs from immediately after the operation until the present date (June 6). The patient has been able to sleep quietly and comfortably, and he expresses himself as greatly pleased with the relief he has been afforded. On April 23, 1911, a tenotomy was done of both the adductors and the hamstrings on each leg, after which the limbs could be placed in a fully extended and abducted position (fig. 8). It was then noticed for the first time that the right hip-joint was dislocated, but of the origin of this we have no knowledge. He can stand with crutches, but we have not yet been able to get him to walk. The wasting and paresis do not give much grounds for hope in this respect.

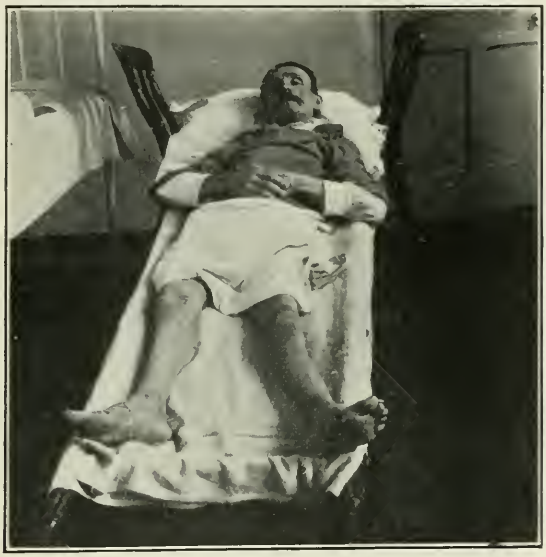


Fig. 8.

Case VI, after operations, legs lying fully extended and abducted.

I do not propose to summarize the cases published of this operation when done for the relief of spasm, because Professor Foerster is dealing fully with this and with other general aspects of the question. There are, however, several points of interest about the subject suggested by the cases related that I will briefly touch upon.

The Theory upon which the Operation is based.—Hitherto, as far as I am aware, there has been no definite proof as to the manner in which the division of the posterior roots diminishes the spasm emitted from the motor nerves of the same segment. Warrington's experimental work, however, supplies this, and Dr. Short has demonstrated in my third case that there does occur in the anterior horn cells and in those of Clarke's column a definite chromatolysis. This change only affects a certain number of the motor cells, and, according to Warrington, some of these recover their normal structure in time. We have here, therefore, a definite anatomical proof of the lessening of the motor activity in these segments where the posterior roots have been resected. In the light of this fact it may be urged that some atrophy and loss of muscular power must result from the operation. But this is shown not to be the case by such examples of the operation afforded by my first two patients, in whom all the posterior roots connected with the leg had been divided, and in whom at intervals of more than one year there is neither loss of power nor bulk in the muscles.

The best Method of Laminectomy.—In my first case I adopted the method of an osteoplastic flap, but I think that it has no advantages which compensate for the great risk of the ill-nourished flap sloughing, and I should never attempt it again. Taylor's suggestion of a hemi-laminectomy is an excellent one when applied to the cervical region. The bifid cervical spines make it quite easy of performance—the oozing surface is reduced to half the area of the usual laminectomy, and the important neck muscles are only interfered with on one side. This method gives, moreover, ample room for dealing with the posterior roots of both sides (fig. 9). In the lumbar region most of these arguments do not apply. The parts are so deeply placed, that one wants all the possible lateral space, and the medianly placed muscle tendons are of not much importance.

The Question of the Two-stage Operation.—On the Continent the operation has usually been performed in two stages—the removal of the laminæ first, and the opening of the dura with the resection of the nerve-roots second. I think that this is not necessary, and that its drawbacks greatly outweigh its advantages. It is designed to minimize the shock which is supposed to be especially great in this operation. This idea arises largely from a theoretical conception that division of a number of sensory roots must cause a grave degree of shock. But when tested by observations on the blood-pressure taken at frequent intervals throughout the operation, it is seen that no more effect is caused by the division of the nerve-roots than by any other stage in the proceeding. In the last two cases recorded above there was no difference between the blood-pressure taken just before and just after the division of the roots; and at the end of the operation it was only a few millimetres less than at the beginning. In those cases where much shock occurs during the course of a laminectomy it is probably due to the too free bleeding and to the long time taken in arresting it. If, however, a large quantity (2 oz. to 3 oz.) of a 1 in 50,000 solution of adrenalin be freely injected into all the subcutaneous and muscular tissues, as suggested by Braun [2], the operation becomes an almost bloodless one, and since I have adopted this plan (in the last three cases) I have not had to apply a single pair of pressure forceps or ligature to a bleeding point. If, then, the operation can be made quick and bloodless, and if the division of the nerve-roots is not productive of

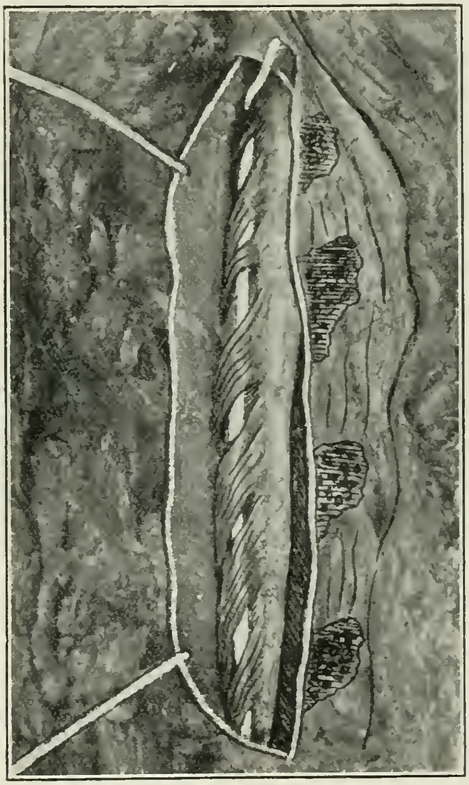


FIG. 9.

Exposure of the brachial roots through a hemilaminectomy of the fifth, sixth and seventh cervical and first dorsal vertebræ. The highest root shown is the fifth cervical and the lowest the second dorsal. A piece of white string underlies the posterior roots.

shock, there is left no reason for adopting the two-stage method of attack. The disadvantages of the double operation are, moreover, very great. There is a much increased danger of sepsis, to which a considerable proportion of the recorded deaths have been due. After the first stage there is all the pain of an ordinary laminectomy, whilst the painful spasms or crises are much exacerbated. The risks of the anæsthetic are doubled, and the chances of healing by first intention are diminished.

The Approach to the Lumbo-sacral Roots.—It has usually been advised in dealing with the lumbar and sacral roots, to approach them at their point of exit from the dural cavity. But this necessitates the removal of all the lumbar laminæ. These, on account of their size, their depth, and the thickness of the muscles covering them, are the most difficult to remove. It seems to me that it is much simpler and easier to attack the lower roots at the point where they all lie close together at their origin from the spinal cord. This necessitates a smaller removal of laminæ, because the roots lie crowded close together instead of far apart. It is usually quite sufficient to take away the laminæ of the eleventh and twelfth dorsal and that of the first lumbar vertebræ to fully expose the roots of all the lumbar and sacral nerves. The incision is placed higher up the back in a situation where it is in less danger of being fouled by urine, or infected from a bedsore. The differentiation between the anterior and the posterior roots is much easier at the level of the cord than at the exit from the dura. The only disadvantage of the high attack of the roots is the increased difficulty of identifying the various nerves. This may be overcome by observing that the third sacral root is the last root of any size to be given off from the conus medullaris. This never need be cut for the relief of spasm of the leg muscles, because it is associated with the buttocks and perineum only. The root above it, viz., the second sacral, is the first to be divided, and then each alternate root is removed [fig. 10]. So long as a good half of the afferent roots in the lumbar and sacral regions are divided, the object of the operation is fulfilled, irrespective of the exact nerves cut. In certain cases, such as my fifth, where the main complaint is due to spasm of a single group of muscles, it would be better, I think, to divide all the posterior roots concerned with that group, rather than to take alternate nerves. It was because I failed to do this that the result in the case I refer to was so poor.

The Relation of Orthopædic Operations to that of Nerve-root Division.—Professor Foerster has so clearly indicated the general rules for the selection of cases of spasm suitable for the operation that I will only refer to one point. It has been suggested by some, especially by Biesalski [1], that the nerve operation should not be done until every possible attempt has been made by tenotomies and other orthopædic

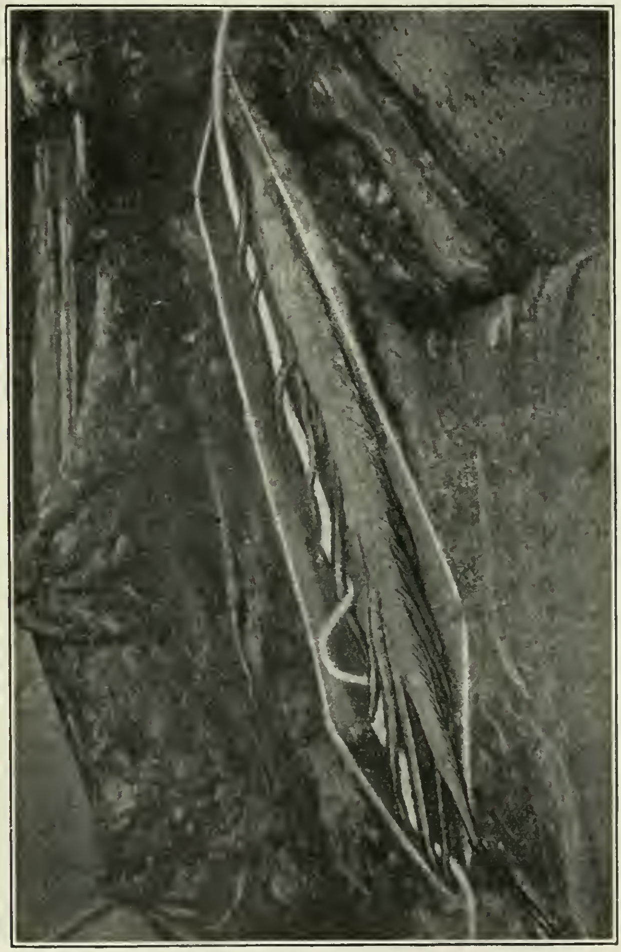


Fig. 10.

Exposure of lumbo-sacral roots by laminectomy of eleventh and twefth dorsal and first and second lumbar vertebræ. The highest root shown is the first lumbar and the lowest the fourth sacral. A piece of white string underlies the roots, and the conus medullaris is held up by a black thread.

operations to correct the deformities. There are, of course, some slight cases where a simple tenotomy will give great relief, but when there is that excessive spasm which ties up a whole limb in a complicated deformity, it seems to me to be useless to expect any good at all from orthopædic operations whilst this spasm remains unabated. Both weight extension and splint treatment are very badly tolerated by patients in whom there is active and painful spasm, whereas if this spasm be first abolished by the nerve operation, the ideal conditions of muscular flaccidity and freedom from pain are provided which are so necessary for the correction of deformities.

I would briefly summarize my conclusions as follows:—

(1) For the relief of pain the operation of posterior nerve-root resection is a somewhat uncertain proceeding. Cases which present very great pain without any organic disease are often of a highly neurotic temperament, and their pain seems to have an important psychical element, which is not relieved by the operation.

(2) For the relief of visceral crises, the operation holds out the only prospect of radical cure which we know of at present. Only those cases of this kind are suitable in which all other means have failed to give relief, but, on the other hand, it is important not to wait until the patient has become debilitated by emaciation and morphia.

(3) In cases of spasm associated with lesions of the pyramidal tract, the operation has its largest field of usefulness. Spasm is, in all these cases, definitely relieved, and if the pyramidal tract is not totally severed, a great improvement of motility results.

(4) The operation causes definite chromatolysis of some of the motor cells in the anterior horn of the segments involved, and this is probably associated with the lessening of motor spasm.

(5) By the use of adrenalin injections as a preliminary to the operation the procedure becomes an almost bloodless and speedy one.

(6) There is no special shock associated with the division of the posterior nerve-roots.

(7) It is better to perform the whole operation in one stage.

(8) In dealing with the brachial roots it is best to do a hemilaminectomy.

(9) The lumbo-sacral roots are best approached at their junction with the cord, rather than at their point of exit from the dura.

In conclusion, I would express my most sincere thanks to Professor Foerster for the inspiration of his work and writings, and especially for his kindness in coming over to London to explain his views, and to demonstrate the brilliant results which have been achieved by his initiative. I have also to thank most cordially Dr. Short for his elucidation of the pathological part of my paper, and Dr. Jackson Taylor for the photographs and slides of the cases, which were under his care as my house surgeon.

REFERENCES.

[1] BIESALSKI. *Münch. med. Wochenschr.*, 1910, lvii, p. 1634.
[2] BRAUN. *Deutsch. Zeitschr. f. Chir.*, Leipz., 1910, cvii, p. 561.
[3] BRAUENIG. *Archiv. f. Anat. u. Phys.* (Phys. Abth.), 1903, p. 251.
[4] BRUNS und SAUERBRUCH. *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, Jena, xxi, p. 173.
[5] CHAVANNAZ. *Gaz. hebd. des Sciences méd. de Bordeaux*, 1906, xxvii, p. 369.
[6] COTTE. *Lyon Méd.*, 1906, cvi, p. 777.
[7] FOERSTER und KÜTTNER. *Beitr. z. klin. Chir.*, Tüb., 1909, lxiii, p. 245.
[8] FRANKE. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1910, p. 37.
[9] HILDEBRAND. *Archiv. f. klin. Chir.*, Berl., 1911, xciv, p. 203.
[10] JACOBY. *New York Med. Journ.*, 1907, lxxxvi, p. 192.
[11] KNAPP. *Boston Med. and Surg. Journ.*, 1908, clviii, p. 149.
[12] MOSKOWICZ. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1910, p. 40.
[13] THOMAS and HALL. *Boston Med. and Surg. Journ.*, 1910, clxiii, p. 26.
[14] WARRINGTON. *Journ. of Phys.*, Lond., 1898, xxiii, p. 112 ; 1899, xxiv, p. 464.

Resection of the Posterior Spinal Nerve-roots in the Treatment of Gastric Crises and Spastic Paralysis.

By Professor FOERSTER (Breslau).

FIRST of all I wish to thank you most sincerely for your kind invitation to this meeting of the Surgical Section of the Royal Society of Medicine. I am conscious of the great honour you have bestowed upon me by allowing me to attend your illustrious assembly. I would also express my deepest gratitude to Mr. Groves for his kind words of welcome and for his having so kindly occasioned my taking part in this present discussion, since the scientific topic which is treated to-day has been especially furthered by English workers from the beginning. I need not remind you that Sir Victor Horsley was the first to open the dura of the spinal cord, and Sir W. H. Bennett, of London, was the first to divide posterior spinal nerve-roots, on December 24, 1888. I should like to express emphatically that my own modest researches, too, on this subject have received the greatest stimulus from English workers, especially from the discoveries of Sherrington and Head. I need not mention before this audience how much we are indebted to Sherrington for all our knowledge of the physiology of the posterior spinal nerve-roots, and that the work of Head on the disturbances of sensation in diseases of the viscera represent the most valuable documents of our knowledge of the sensory nerve supply to the internal organs.

(1) Resection of the posterior spinal nerve-roots for the relief of pain has already been carried out in the past in a considerable number of cases. After Bennett, who divided four lumbar and one sacral posterior nerve-roots in a case of sciatica, the operation has been performed by Abbe and Munro in several cases of neuralgia of the brachial plexus, by Sir Victor Horsley in two cases of severe neuralgia of the arm, by Chipault, Demoulin and Monod in five cases of neuralgia of the arm, by Giordano in a case of sciatica, and by Faure in a case of cancer of the uterus. In three cases of cancer of the spinal column Tietze and I performed resection of the posterior roots in order to relieve the fearful pain which radiated from these roots: but the results were only successful for a short time, since, owing to the progress of the disease, other roots were subsequently attacked. In 1900,

Mingazzini suggested resection of the posterior nerve-roots in cases of locomotor ataxia with severe lightning pains, but the operation was not actually performed until recently, when Hildebrand, in 1910, cut two cervical nerve-roots in a patient suffering from tabes: a relapse occurred. Enderlen performed resection of certain thoracic roots for severe girdle pain. Mr. Groves has also to-day reported to you on a case in which the pain remained. Last summer, Gottstein and I cut the fourth and fifth lumbar and first sacral roots in a tabetic patient suffering from a perfectly localized neuralgia of the internal malleolus; for some weeks there was complete absence of pain, but afterwards it recurred as badly as before. I am inclined to believe that if resection of the posterior nerve-roots is to be performed at all in cases of tabes with lightning pains, it will be necessary to remove all the roots of one extremity at once; for we never know with absolute certainty which roots are particularly influenced by the morbid stimuli, and there is considerable overlapping of the regions supplied by the different roots. Thus, in our case of tabes, the resection of three roots was not followed by any sensory disturbance.

(2) When I suggested performing resection of certain posterior thoracic roots in cases of visceral crises I was influenced by a consideration of the severe pain and symptoms of sensory irritation which form the basis of these crises, and of the often enormously increased hyperæsthesia of the skin of the epi-, meso-, and hypogastrium which accompany the crisis. In the manner in which patients are wont to describe and localize the pain in gastric crises, and in the abdominal hyperæsthesia, such crises show a marked resemblance to the sensory symptoms observed in certain organic diseases of the stomach, notably gastric ulcer, as well as diseases of the liver, gall-bladder, and intestine. With these diseases also, marked hyperæsthesia of certain parts of the chest and abdomen occurs very frequently, as has been shown by the classical investigations of Head. I was thus led to the view that the gastric and intestinal crises in tabes were caused by an irritation of the sensory sympathetic fibres of these organs which enter the spinal cord through certain posterior thoracic roots. Supposing this view to be correct, the crises would cease after the resection of these roots.

The operation has now been performed altogether twenty-eight times, as shown by Table I (p. 228). Three cases succumbed to the immediate effects of the operation, two showed no improvement, the crises persisting. In the remaining twenty-three cases the operation was successful. Immediately after the resection of the roots the crises disappeared, the body-weight increased, and the general condition showed a marked improvement: some of the patients who, until then, had been absolutely confined to their bed for some time, have since regained their power to work. In the majority of these cases (fifteen) no relapse has been reported. We must add still the case reported by Mr. Hey Groves to-day. The period which has elapsed since the operation is shown in the table. In several of these cases there has been no recurrence of the crises for many months, and even for over a year, although before the operation a continuous critical state had persisted. In the remainder of the cases (seven) the crises ceased for a while, but subsequently recurred. Yet even in these the critical state has not returned. The crises only occur occasionally, at long intervals; they are of short duration and moderate intensity. Among this latter group of cases are three patients who have even regained the power of earning their living, one of them being an architect and one a mason.

Table I.—Gastric Crises.

28 cases ... ... 25 survived ... ... 3 deaths

23 successful ... ... 2 not benefited

				Interval between operation and publication
No relapse	15 cases	Küttner	(1)	5 months
		Küttner	(1)	1⅓ years
		Bierens de Haan	(1)	7 weeks
		Goetzel	(1)	3 months
		Becker	(1)	3 months
		Guleke	(1)	2 months
		Nonne	(2)	Not stated
		Sänger	(1)	
		Tick	(2)	
		Bornhaupt	(1)	
		Thomas-Hall	(1)	
		Franke	(1)	
		Frankl-Hochwart	(1)	
Relapse	8 cases	Sauerbruch	(1)	
		Tietze-Foerster	(3)	
		Guleke	(3)	
		Mainzer	(1)	

If in a number of cases the result of the operation is not perfect, it is to be attributed to the fact that the system of roots carrying sympathetic nerve-fibres of gastro-intestinal origin is very extensive. By the method which has hitherto been practised of only cutting from the seventh to the ninth or the seventh to tenth thoracic roots the source of the crises has not always been entirely eliminated. Probably the fifth and sixth as well as the eleventh and twelfth thoracic roots are also concerned. It is therefore desirable to remove as many of the nerve-roots as possible, although this increases the gravity of the operation. What is even more important, is in every case to determine exactly the situation of the pain, the extent of the hyperæsthesia of the skin and the site of its maximum intensity, since these data may possibly afford a guide as to which roots are chiefly irritated and concerned in causing the crises. In this connexion a publication by Guleke is of interest: in one patient the crises disappeared for a time after the resection of the seventh to the ninth thoracic roots. They recurred, but again disappeared after the resection of the tenth and eleventh thoracic roots. For such extensive resections Guleke's method would appear to be particularly well suited. While ordinarily the dura is opened and the posterior thoracic roots are resected close to the spinal cord—as is shown in fig. 1—Guleke does not open the dura, but picks up the roots in their course outside the dura (fig. 2). Here also the anterior and posterior roots are enclosed in separate sheaths separable

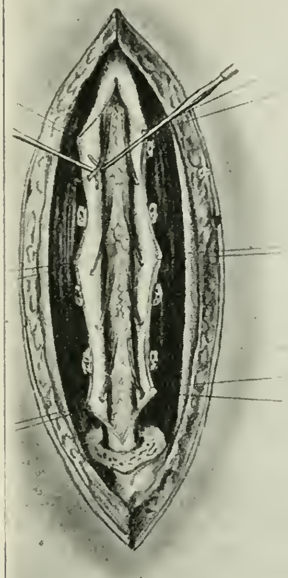


FIG. 1.

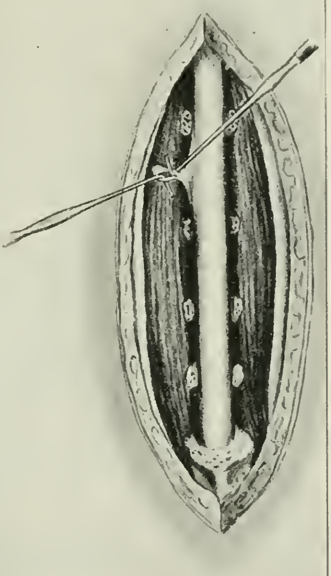


FIG. 2.

from one another with comparative ease. The posterior roots are then cut.

We must, however, bear in mind that gastric crises may possibly arise from irritation of the pneumogastric nerve and perhaps even of the vomiting centre in the medulla. All cases of gastric crises without any pain should be regarded as suspicious and as unsuitable for resection of the posterior nerve-roots.

(3) I will now discuss the value of resection of the posterior spinal nerve-roots in spastic paralysis due to disease of the cortico-spinal path, especially the pyramidial tract. These paths carry two kinds of fibres—firstly, those bearing the motor impulse, injury to which causes paresis or paralysis, and secondly, the inhibitory fibres which check the sensory stimuli entering into the grey matter of the spinal cord by the sensory nerve-roots. When the inhibitory fibres are damaged, the sensory afflux acts unrestrained upon the muscles, thus causing the spastic symptoms. If in such cases the posterior roots—that is to say, the path of afferent sensory impulses—are cut, the excess of sensory impulses is again reduced and the spastic symptoms are diminished or may even disappear

TABLE II.—SPASTIC PARALYSIS.

81 cases ... ... 72 survived ... ... 9 deaths

51 *cases of congenital spastic paraplegia (Little's disease)*—
45 successful; 1 not benefited; 5 deaths

3 *cases of acquired cerebral spastic paraplegia*—
All successful

4 *cases of spinal spastic paralysis of traumatic origin*—
2 successful; 2 not benefited: 1 case, wrong roots divided (11 Th., 12 Th., 1 L.); 1 case, complete interruption of the cortico-spinal tracts

1 *case of spastic paraplegia from compression of the spinal cord (Pott's disease)*—
Successful

2 *cases of syphilitic spastic paraplegia*—
Successful

1 *case of primary spastic paraplegia (?) (Erb-Charcot)*—
Successful

6 *cases of disseminated sclerosis*—
3 deaths; 1 case not benefited; 1 case successful, disease rapidly progressing, death; 1 case successful

12 *cases of spastic paralysis of the arm*—
1 death; 8 cases benefited; 3 cases not successful—complete interruption of the cortico-spinal tracts

completely. Frequently the fibres carrying the motor impulses are *still fairly intact.* In such cases resection of the posterior roots is followed by a complete disappearance of the spasms, and all those voluntary movements which were before so seriously interfered with by the spastic resistance of the muscles are now executed without any difficulty. We may therefore conclude that the operation can only be successful where voluntary excitability of the muscles is still present to a certain extent. Further, this treatment can only cure those spastic conditions of the muscular system which are actually due to the influx of excessive *sensory* stimuli to the grey matter of the cord—i.e., only in true *reflex* spastic contraction of the muscles. On the other hand, resection of the posterior nerve-roots cannot be successful in the muscular contractions of athetosis, chorea, mobile spasms; for this latter class of diseases, which have so often been confused with the former, are due to the direct pathological stimulation of *motor* centres and paths belonging to the cerebrum and mid-brain: they are therefore comparable to the epileptic convulsions which arise from a corresponding irritation of the motor centres of the brain. Resection of the posterior roots cannot eliminate the pathological stimulus of these centres. In all such cases it has therefore proved unsuccessful.

The operation has hitherto been performed in eighty-one cases, as is shown by Table II. Of these, nine died as the result of the operation; seventy-two survived. Fifty-one were cases of congenital spastic paraplegia (Little's disease): almost all these patients have been benefited by the operation, some of them showing a marked improvement. I should like to demonstrate to you in pictures the extent of this improvement in some cases. The patients in question were operated upon by Küttner, a part only of these cases having been published.

Case I.—Boy, aged 7. Severest rigidity of the limbs (fig. 3), unable to make the slightest voluntary movement, to sit, stand, or walk; the patient lay in bed like a log. Preliminary orthopædic treatment, consisting in tenotomy of the Achilles and adductor tendons, had been absolutely of no avail. In consequence the legs were now held in extreme abduction (fig. 4) with the same rigidity as they had previously been held in adduction. The result of the operation of root division was immediate cessation of the spasms and the return of voluntary motility. The boy now moves his legs freely, he even lifts one leg alone without any assistance (fig. 5), he can stand erect with straight knees unaided (fig. 6), and walks quickly and safely on two crutches

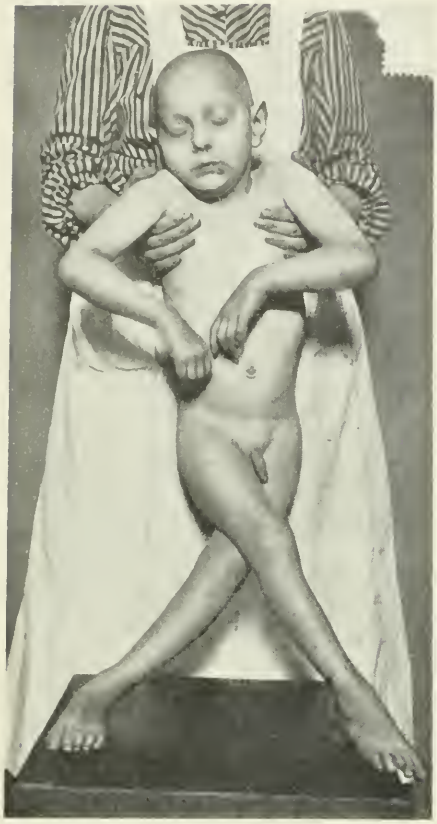


FIG. 3.

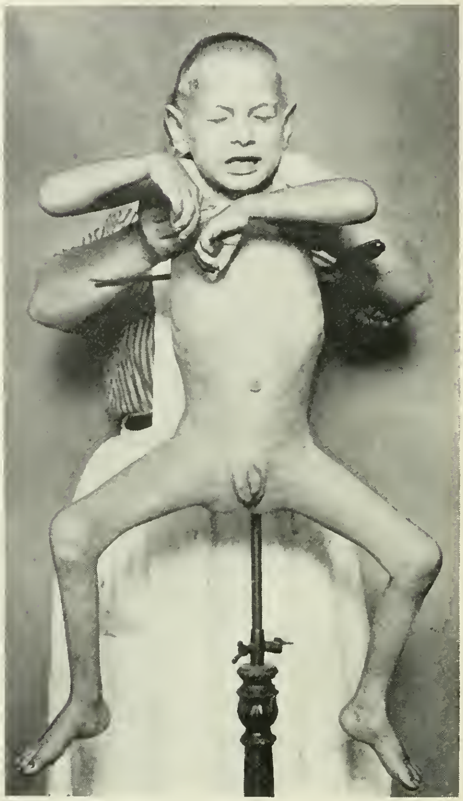


FIG. 4.

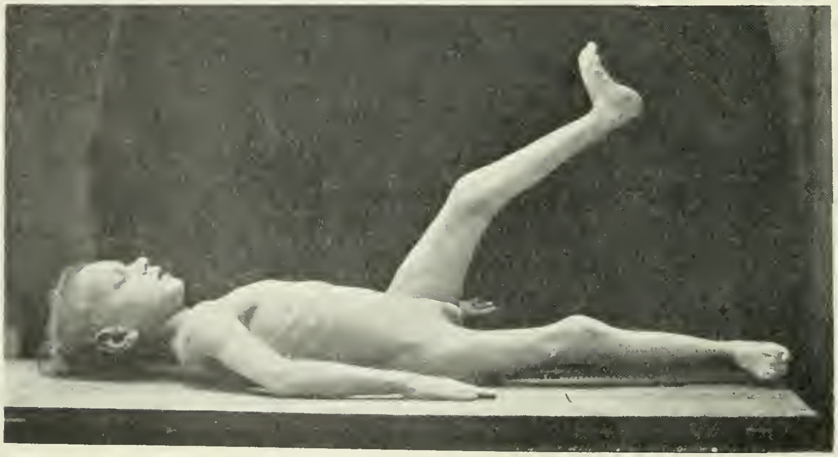


FIG. 5.

without any help. The improvement was maintained under the use of suitable exercises, and is still going on.

Case II.—Boy, aged 11. Severest rigidity; was able to carry out slight voluntary movements of the legs, but could only bend both legs a little at the same time (fig. 8), considerable subsidiary movements being carried out with the arms and head. The patient was quite unable to stand (fig. 7) or to walk, and could not sit up even when assisted, the resistance of the extensors of the pelvis being insurmountable (fig. 9). After the operation the rigidity has quite disappeared; voluntary movements of the legs are carried out freely, the patient being able to bend each leg by itself (fig. 10), to lift it up high without also bending the

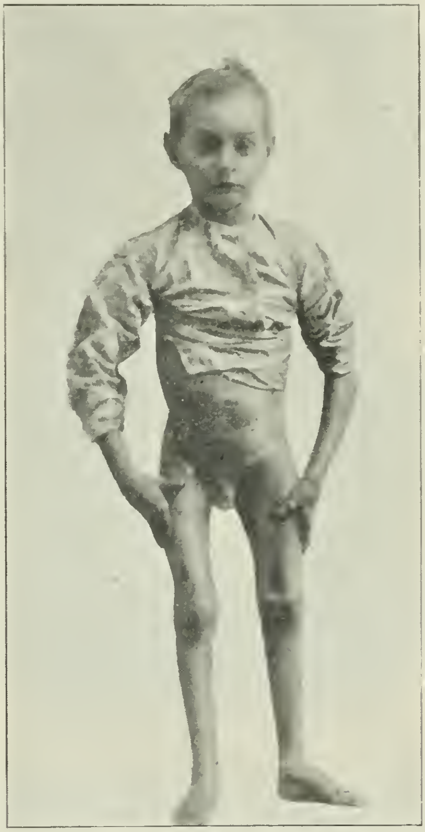


Fig. 6.

knee and ankle-joint (fig. 11); he sits up alone (fig. 12), stands (fig. 13), and walks alone with long strides (fig. 14).

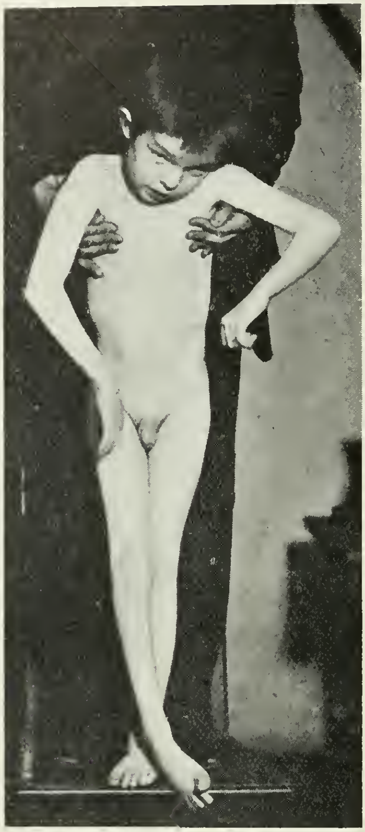


Fig. 7.

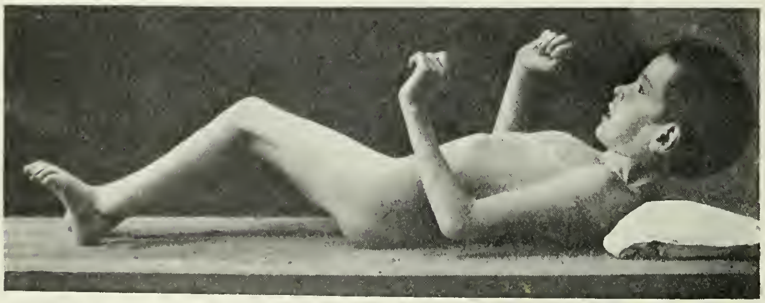


Fig. 8.

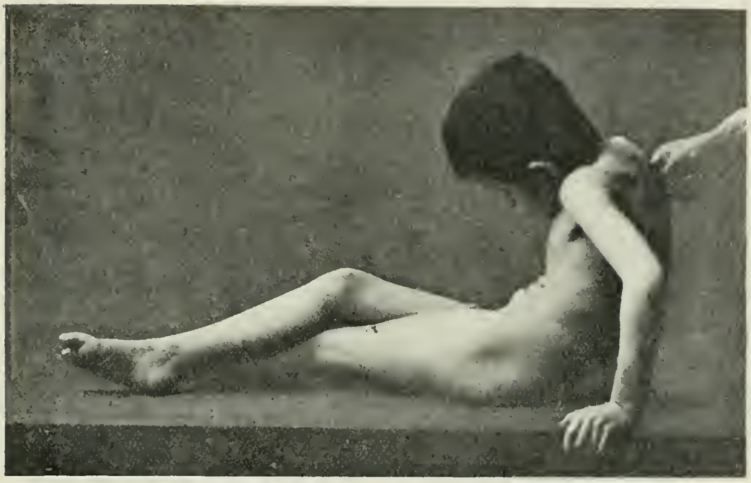


FIG. 9.

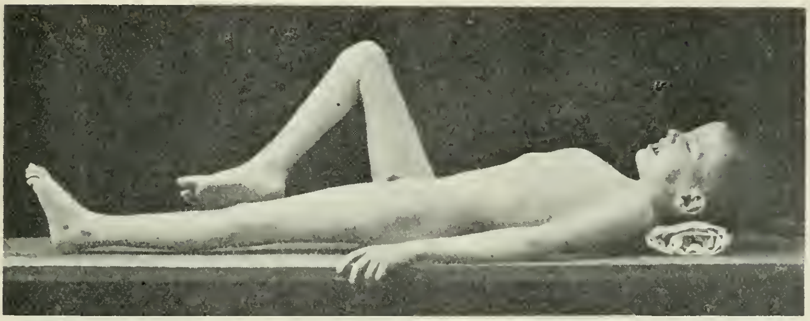


FIG. 10.

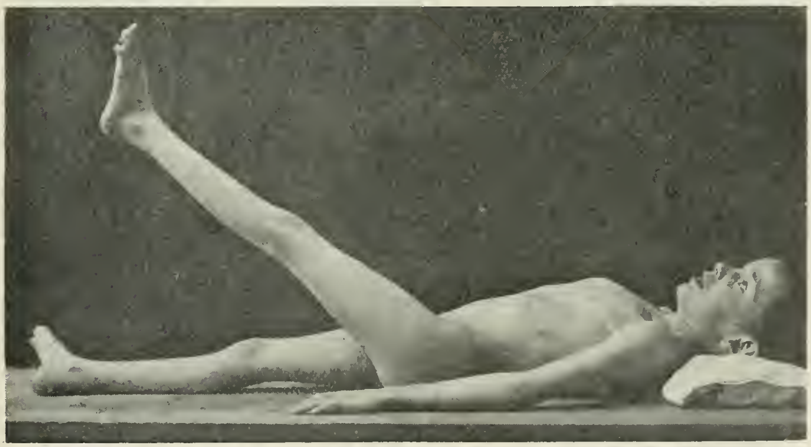


FIG. 11.

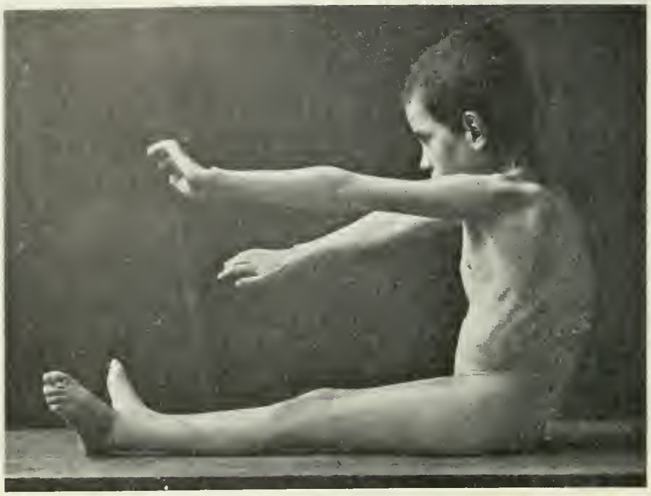


Fig. 12.

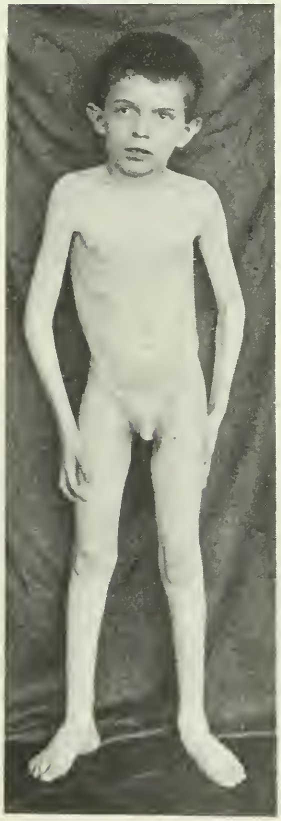


Fig. 13.

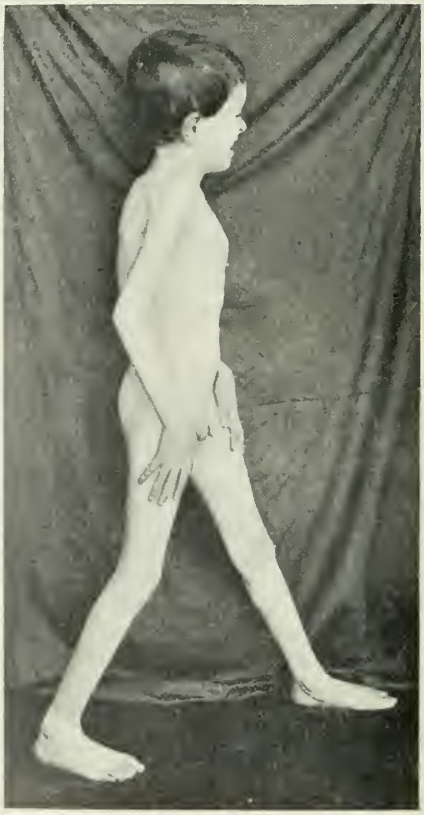


Fig 14.

Case III.—Boy, aged 10, with severe rigidity of the limbs and contraction of the flexors (fig. 15). Motility as in the previous case: the patient was unable to sit, stand (fig. 16), or walk. After the operation the spasticity quite disappeared, and perfectly free motility was

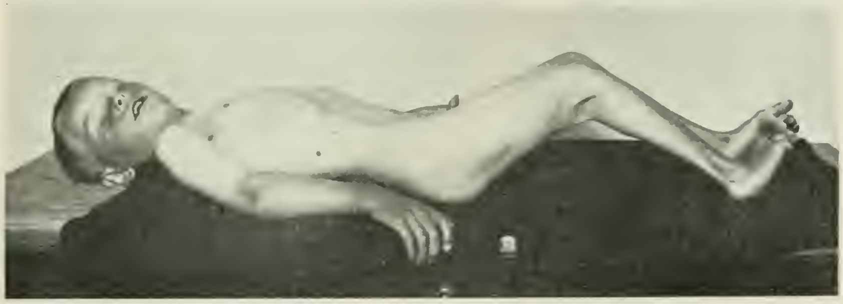


Fig. 15.

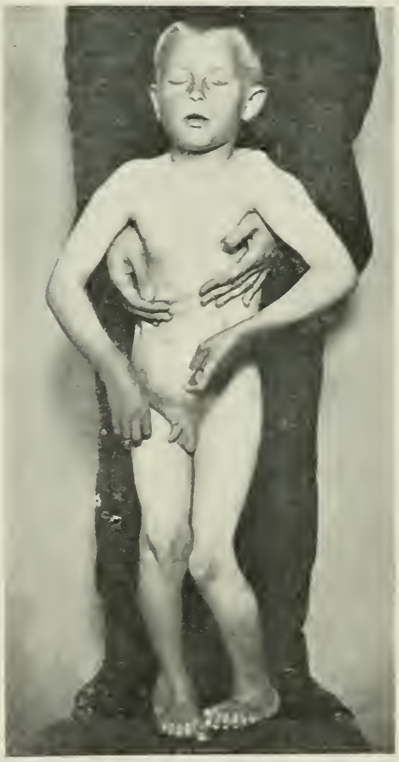


Fig 16.

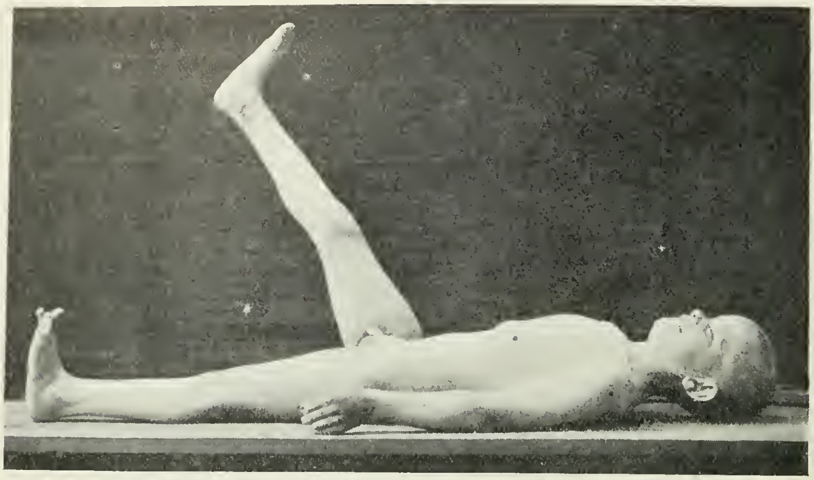


Fig. 17.

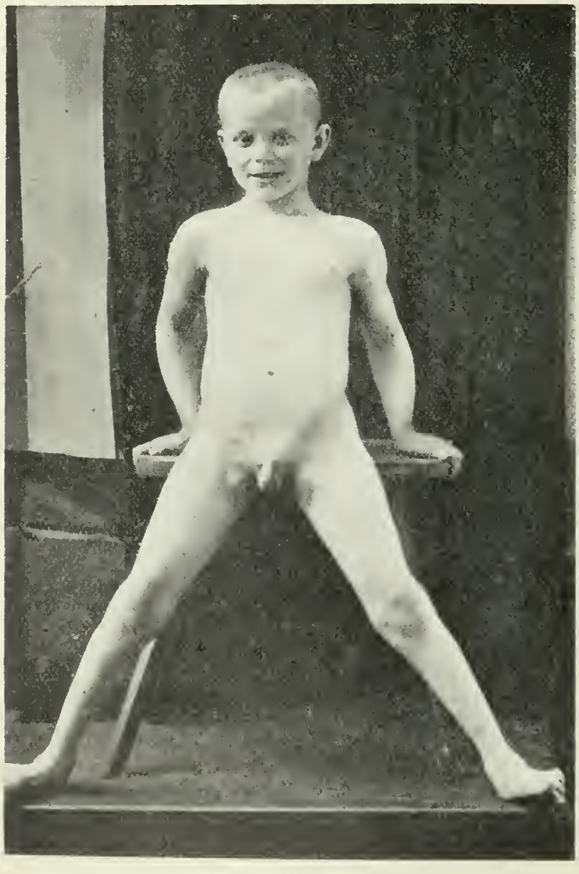


Fig. 18.

obtained, the patient being able to bend each leg independently or to lift it high up (fig. 17), and to abduct the legs widely (fig. 18); the patient stands alone (fig. 19) and he walks alone with long steps (fig. 20), he bends the leg well in walking (fig. 21) and steps well forwards (fig. 22), mounts stairs without fatigue, and is out alone in the open for many hours.

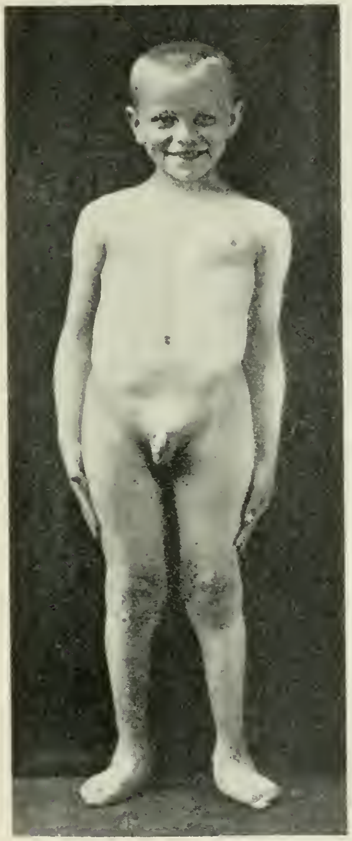


FIG. 19.

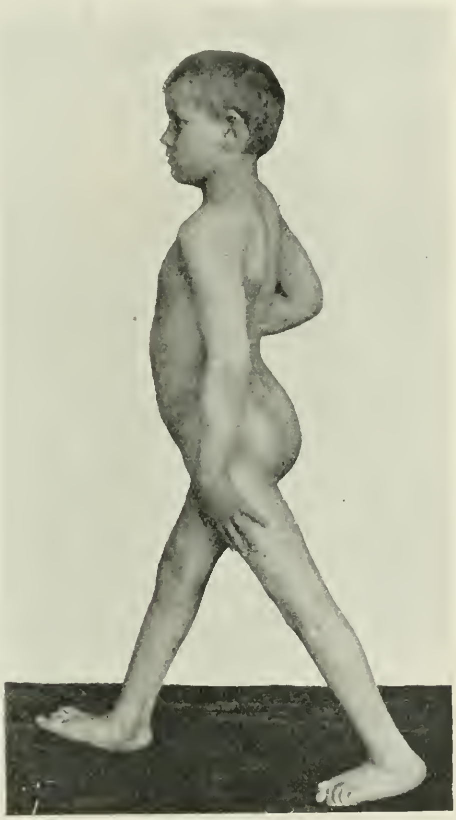


FIG. 20.

Case IV.—Unpublished case. Severest spastic paralysis of all four limbs, figs. 23 and 24 showing how arms and legs were inextricably interlocked, as it were, in a tangle. The patient was totally unable to carry out any voluntary movement, lay in bed huddled up, and in addition suffered from athetosis. Root resection was carried out here both in the lumbo-sacral and the cervical part of the cord. The spasms were greatly improved; the patient can now lift up each leg separately (fig. 25); in standing (fig. 26) and walking he has still to be supported on both sides, since the operation was carried out comparatively recently, but he walks in this way with long steps. The patient voluntarily lifts each arm up high (fig. 27), and can use his hands in eating and drinking. In another similar case of spastic diplegia Küttner and I have performed resection of the cervical as well as the lumbar roots.

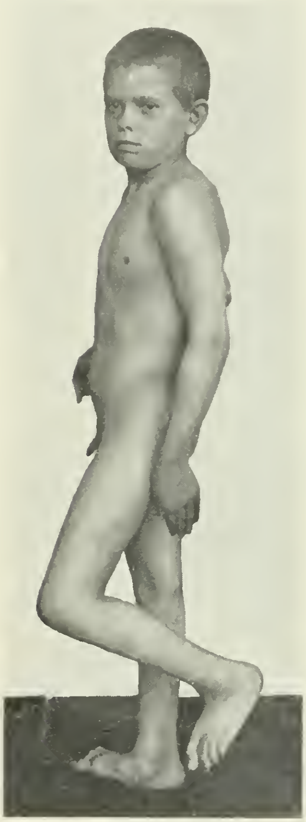


Fig. 21.

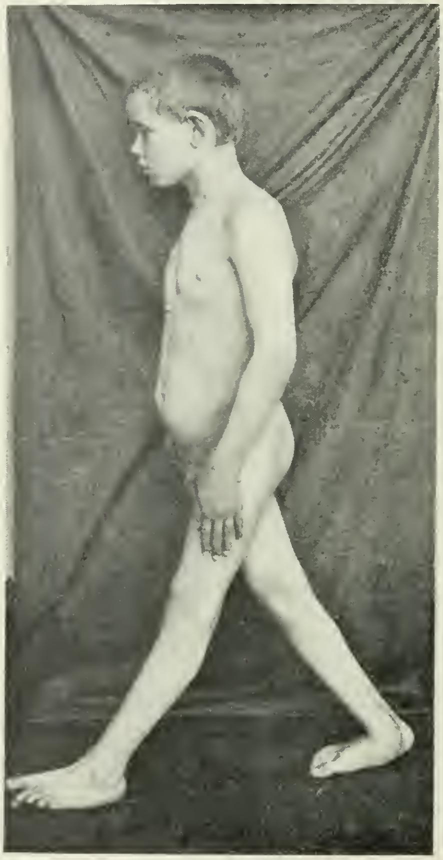


Fig. 22.

On returning to a consideration of our table, we next find the following cases: Three patients with acquired cerebral spastic paralysis, all of which were treated successfully, one of them being a case of

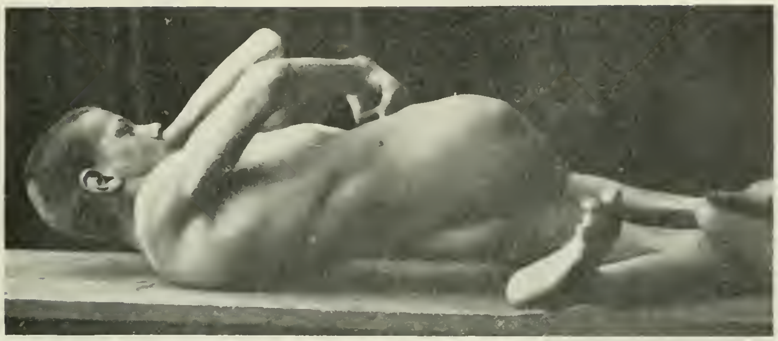


FIG. 23.

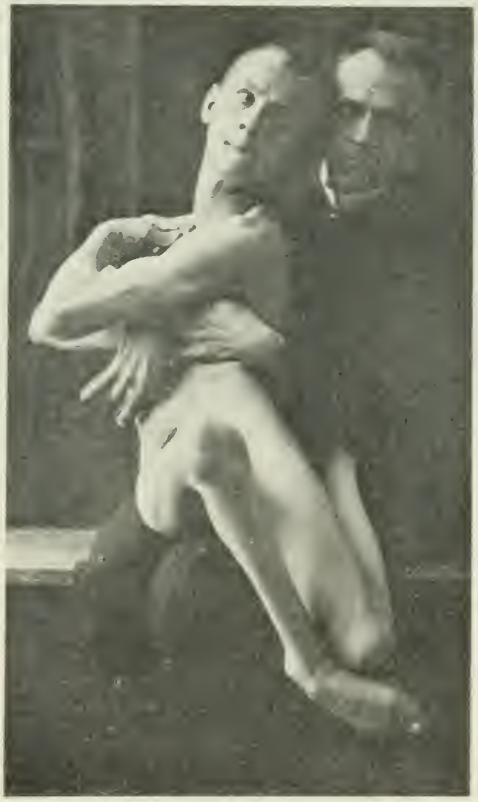


FIG. 24.

hydrocephalus with severe spastic paraplegia. There is also a considerable number of cases of spinal disease with spastic symptoms. Of four cases of spinal spastic paralysis of traumatic origin, two were successful; of the remaining two, which were failures, in one wrong roots were cut, whilst in the other the cortico-spinal fibres appear to have been completely interrupted; in this case the spasms disappeared, but voluntary motility did not return.

In addition there is one case of Pott's disease which was successful, also two cases of syphilitic spastic paraplegia, successful; two cases of primary spastic paraplegia (Erb-Charcot), successful; of six cases of disseminated sclerosis, three died as the result of the operation, one was not benefited, one was at first successful, but subsequently the disease progressed rapidly and the patient died; the sixth was benefited.

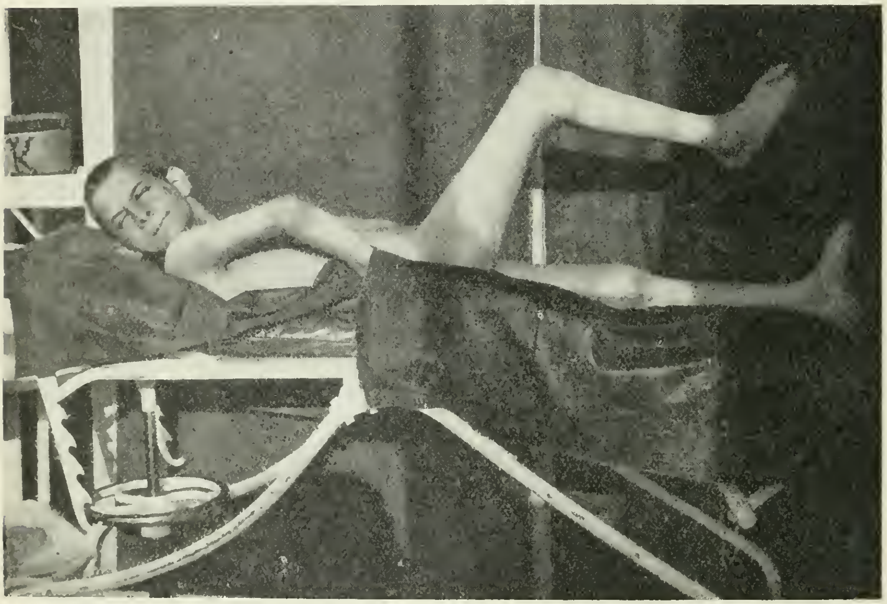


Fig. 25.

Of twelve cases of spastic paralysis of the arm, one died, eight were successful, one of these being the Case IV already demonstrated; the remaining three were not benefited owing to complete interruption of the cortico-spinal path; in these patients the spasms disappeared, but voluntary motility did not return.

These comprise the cases to my knowledge.

There remain still a few points to which I would call your attention. As regards the indications of the operation I must, in the first place, repeat that only lesions with real reflex spasticity as the result of the loss of the cortico-inhibitory fibres are suitable, but not the diseases of the type of chorea, athetosis, mobile spasm and spasmodic torticollis. Lately, Abbe recommended *posterior* spinal nerve-roots division for the relief of athetosis, and quotes a case as proof in which he, in addition to the resection of posterior roots for the relief of pain, also resected four *anterior (!)* roots for the relief of athetosis, as he expressly says in his first publication of the same case in 1896. Often athetosis is

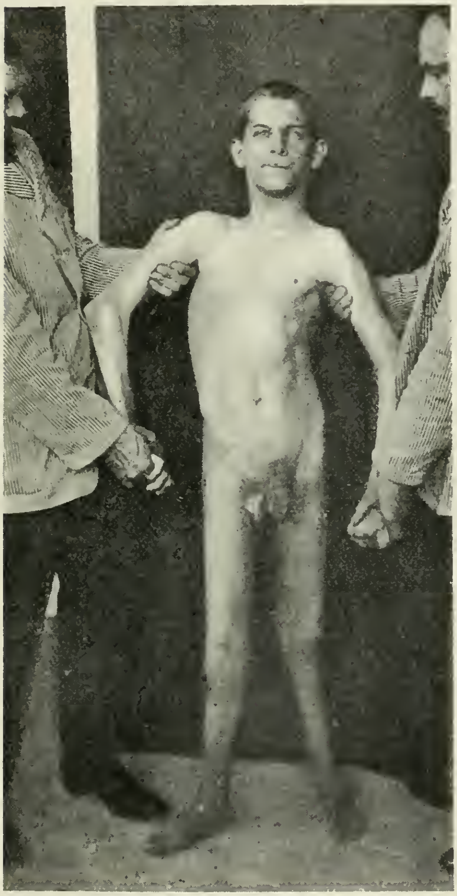


FIG. 26.

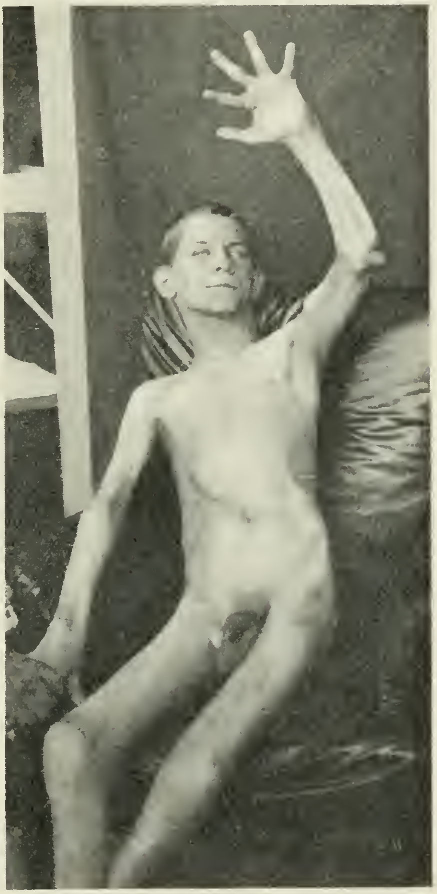


FIG. 27.

combined with real reflex spasticity; in such cases the resection of the posterior roots removes the spasticity, whilst the athetosis does not disappear, or only for a short time. Possibly Parkinson's disease may be suitable.

Secondly, there must be a reasonable prospect of innervating fibres of the pyramidal tract still existing, all cases with slight spasms and considerable paralysis being unsuitable. In cases of severe spasms and total voluntary paralysis there may remain a considerable amount of voluntary excitability only obscured by the spasms and only apparent after their elimination. In such cases the experiment is worth trying. The question whether any voluntary motility remains may be decided by preliminary injection of stovaine, which temporarily removes the spasms. The arm, as a general rule, is less suitable than the leg. Thirdly, the disease must have become practically stationary, whilst progressive diseases like disseminated sclerosis are hardly suitable, very slowly progressing ones like spastic spinal paralysis being perhaps more promising.

The second point to which reference should be made is the after-treatment of the patient. Good results can only be obtained with very careful exercise treatment. It is only by such exercises that voluntary motility is brought up to its highest level, and that, above all, standing and walking are gradually rendered possible. Such exercises should be continued for years, during which continuous progress may be observed. The assistance of orthopædic measures cannot be dispensed with; above all, it is necessary to place the limbs in removable plaster splints in such a position as to correct the previous deformity; the legs in abduction, extension and outward rotation, the knees in extension, the feet in dorsal flexion, the arms in abduction, the forearms in extension, the hands in supination and extension, the fingers in extension, the thumbs in opposition. These positions are required during the whole time immediately following the operation, the limbs being only removed from their plaster cases for the purpose of the exercises, which should be carried out several times a day; later on they should still be kept in the splints at night.

If beside the real spastic contractions there are also organic contractions due to shrivelling of the muscles and tendons, tenotomy or tenoplasty may also have to be performed. As to the technique of the operation, I am fully convinced that Mr. Groves's proposal for the use adrenalin is of the greatest importance, as the bleedings often assume a threatening character. It seems to me also that his method of cutting the roots nearer to the spinal cord simplifies the operation, although it renders it more difficult to distinguish the different roots.

In conclusion, Mr. President and Gentlemen, let me thank you for the kind attention with which you have honoured me and the leniency with which you have regarded my inefficient use of your language.

BIBLIOGRAPHY.

I.—Resection of Posterior Spinal Nerve-roots for Neuralgia.

Abbe. *Ann. New York Med. Soc.*, February, 1889, and *Med. Rec.*, New York, 1889, xxxv, p. 149; "Intradural Section of the Spinal Nerves for Neuralgia," *Boston Med. and Surg. Journ.*, 1896, cxxxv, p. 329; *St. Luke's Hosp. Med. and Surg. Reports*, 1910, ii, pp. 1-20; *Med. Rec.*, 1911, lxxix, p. 377.

Bennett. *Med.-Chir. Trans.*, 1889, lxxii, p. 329; *Lancet*, 1889, i, p. 839.

Chipault. "Nevralgie de la huitième racine postérieure cervicale droite . . ," *Nouv., Iconog. de la Salpêtrière*, 1895, xiv, p. 136; "Du traitement de certaines nevralgies rebelles . . ." Acad. de Méd., 19 janvier, 1897, *Gaz. des Hôp.*, Par., 1897, lxx, p. 71; "Etat actuel de la chirurgie nerveuse," Paris, 1902, pp. 319 *et seq.*

Chipault et Demoulin. "Resection intradurale des racines postérieures," *Trav. de Neur.-Chir. de Chipault*, 1896, i, p. 234.

Faure. "Resection palliative des racines postérieures dans les cancers uterins inopérables," *Trav. de Neur.-Chir. de Chipault*, 1889, iv, p. 238.

Flörcken. *Münch. med. Wochenschr.*, 1910, lvii, p. 441.

Giordano. *Gaz. degli Osp.*, December 9, 1900.

Hildebrand. *Archiv f. klin. Chir.*, Berl., 1911, xciv, p. 203.

Horsley, cited by Head. *Brain*, 1905, xxviii, pp. 81-84.

Mingazzini. "Rendiconti della R. Accademia medica di Roma seduta del 26 Marzo, 1899," referita *Policlinico gior. di med. pratica*, 1899; *Neurol. Centralbl.*, Leipz., 1910, xxix, p. 406.

Monod et Chipault. "Un cas de resection intradurale des racines postérieures," *Trav. de Neur.-Chir. de Chipault*, 1899, iv.

Munro. *Journ. Amer. Med. Assoc.*, Chicago, 1904, xliii, p. 1183.

II.—Resection of Posterior Spinal Nerve-roots for Gastric Crises.

Becker. *Med. Klin.*, Wien, 1911, vii, p. 775.

Bierens de Haan. *Nederl. Tijdschr. voor Geneeskunde*, 1910, tweede helft, No. 3.

Bornhaupt. *St. Petersb. med. Wochenschr.*, 1911, xxxvi, p. 25.

Bruns und Sauerbruch. *Mitt. u. d. Grenzgeb. d. Med. u. Chir.*, Jena, 1909, xxi, p. 173.

Foerster. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 46; *ibid.*, 1911; *Verhandl. d. Gesellsch. deutsch. Nervenärzte*, Leipz., 1911, p. 231; *Verhandl. d. breslauer chir. Gesellsch.*, Sitzung vom 8. Mai, 1911; *Berl. klin. Wochenschr.*, 1911, xlviii, p. 1156; *Therap. der Gegenwart*, Berl., 1911, lii, p. 276.

Foerster und Küttner. *Beitr. z. klin. Chir.*, Tüb., lxiii, p. 245.

Franke. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 37.

v. Frankl-Hochwart. *Verhandl. d. Gesellsch. deutsch. Nervenärzte*, Leipz., 1911, p. 229.

Goetzl. *Wien. klin. Wochenschr.*, 1910, xxiii, p. 777.

Guleke. *Zentralbl. f. Chir.*, 1910, xxxvii, p. 1190; *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1911. Bericht: *Zentralbl. f. Chir.*, Leipz., 1911, xxxviii (Beilage), p. 48.

Küttner. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 41.

MAINZER. *Münch. med. Wochenschr.*, 1911, lviii, p. 989.
NONNE. *Verhandl. d. Gesellsch. deutsch. Nervenärzte*, Leipz., 1911, p. 227.
SANGER. *Ibid.*, p. 229.
THOMAS and HALL. *Boston Med. and Surg. Journ.*, 1910, clxiii, p. 26.
TICK, cited by FLÖRCKEN. *Münch. med. Wochenschr.*, 1910, lvii, p. 1441.
TIETZE. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 38.

III.—RESECTION OF THE POSTERIOR SPINAL NERVE-ROOTS FOR THE RELIEF OF SPASTICITY.

ANSCHUTZ. "Med. Gesellsch. zu Kiel," *Münch. med. Wochenschr.*, 1909, lvi, p. 1676.
BÁRÁNY-BANZI. *Wien. klin. Wochenschr.*, 1911, xxiv, p. 187.
BIESALSKI. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 34; *Münch. med. Wochenschr.*, 1910, lvii, p. 1634.
BORNHAUPT. *St. Petersb. med. Wochenschr.*, 1911, xxxvi, p. 25.
BRODNITZ. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 36.
BYCHOWSKI. *Gazety Lekarskiez*, 1910.
CLAIRMONT. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1911. Bericht: *Zentralbl. f. Chir.*, 1911, xxxviii (Beilage), p. 48.
CLARK and TAYLOR. *New York Med. Journ.*, 1910, xci, p. 215.
CODIVILLA. *Munch. med. Wochenschr.*, 1910, lvii, p. 1438.
DELREZ. *Soc. Méd.-Chir. de Liège*, séance du 4 mai, 1911; *Zentralbl. f. Chir.*, 1911, xxxviii, p. 1200.
FOERSTER. *Zeitschr. f. orthop. Chir.*, 1908, xxii, p. 203; *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, Jena, 1909, xx, p. 493; *Berl. klin. Wochenschr.*, 1910, xlvii, p. 1441; *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, pp. 42 ff.; *Verhandl. d. Gesellsch. deutsch. Nervenärzte*, Leipz., 1911, pp. 146 ff. and 229; *Therap. der Gegenwart*, Berl., 1911, lii, p. 13; *Ergebnisse d. Chir. u. Orthop.*, Berl., 1911, ii, pp. 173 ff.
FRAZIER. *Surg., Gyn., Obstet.*, Chicago, 1910, xi, p. 251.
VAN GEHUCHTEN. *Le Névraxe*, 1910, xi, p. 245.
GÖBELL. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, pp. 34 ff.
GOLDENBERG. *Münch. med. Wochenschr.*, 1911, lviii, p. 988.
GOTTSTEIN. *Berl. klin. Wochenschr.*, 1909, xlvi, p. 784; *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, pp. 29 ff.
GOYANES. *Rev. de méd. y cir. prat. de Madrid*, January 14, 1910 (ref. *Centralbl. f. Chir.*, 1910, No. 14).
GÜMBELL. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1911. Bericht: *Zentralbl. f. Chir.*, 1911, xxxviii (Beilage), p. 48.
HEVESI. *Deutsch. med. Wochenschr.*, 1910, xxxvi, p. 880.
HILDEBRAND. *Archiv f. klin. Chir.*, Berl., 1911, xciv, p. 203.
KLAPP. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 33.
KOTZENBERG. *Zentralbl. f. Chir.*, 1909, xxxvi, p. 501.
KÜTTNER. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, pp. 23 ff. and pp. 40 ff.; *Beitr. f. klin. Chir.*, Tüb., 1910, lxx, p. 393.
MAY, O. *Lancet*, 1911, i, p. 1489.
MOSKOWICZ. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 40.
ROSE. *Semaine méd.*, Par., 1909, xxix, p. 3113.
SCHLESINGER. *Neurol. Centralbl.*, Leipz., 1910, xxix, p. 970.
SCHÜLLER-MOSKOWICZ. *Wien. klin. Wochenschr.*, 1910, xxiii, p. 833.
SPILLER and FRAZIER. *Univ. Pennsyl. Med. Bull.*, Philad., 1909-1910, xxii, p. 314.
TIETZE. "Die Technik der Foersterscher Operation," *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1909, xx, p. 559; *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 38.
WENDEL. *Münch. med. Wochenschr.*, 1910, lvii, p. 273; *Verhandl. d. deutsch. Gesellsch. f. Chir.*, Berl., 1910, p. 31.

DISCUSSION.

The PRESIDENT (Mr. Rickman J. Godlee) tendered the thanks of the Section to Professor Foerster for the interesting paper he had read, and at the same time congratulated him upon the efficient English in which he clothed it, so that there was no difficulty in following it. The appreciation of the Section was not lessened by the reflection that the Professor had come from a city which was only six hours removed from the Russian frontier.

Dr. OTTO MAY showed a boy, aged 5, a typical case of Little's disease. He was a premature child, one of twins, and from birth onwards showed a well-marked spastic contracture, chiefly in the legs. The child came under his care at the Evelina Hospital nine months ago, and after he had watched him for six months he suggested to the mother that the operation suggested by Professor Foerster offered a reasonable prospect of enabling him to walk. He regretted that he had not brought photographs of the boy before operation, but the disease was of moderate severity; he could not himself sit up at all, and the legs were very stiff, and in a position of marked flexion and adduction. He could stand if firmly supported, but the legs assumed the typical scissor position. His intelligence was very poor, though his mother stated that at home he could talk. The operation was performed by Mr. Clogg about six weeks ago, and was done in two stages. It consisted in dividing the roots suggested by Professor Foerster—the second, third, and fifth lumbar and the second sacral on each side. In so choosing, the rule laid down by Professor Foerster was followed, not to divide more than two consecutive roots in any position, so as to avoid producing definite anæsthesia of the legs, and to avoid removing the complete sensory innervation of any group of muscles. If one divided all the afferent fibres corresponding to any particular group of muscles, a marked ataxia was to be expected: whereas if one left one corresponding posterior root, ataxia was mitigated. A drawback was that there was still a path for the afferent impulses which led to the spasm, so one could not expect to get rid of all the spasm, but only to diminish it to a very large extent. The operation on the boy was uneventful, and the immediate result was a considerable diminution of all the contracture, and the limbs, immediately after the operation, seemed flaccid. Since the operation the boy had been kept on a double Thomas's splint, jointed at the hips, so that they could be forcibly abducted, and they had been kept in a position of strong abduction and extension. Since about a fortnight after the operation the limbs had been passively exercised two or three times a day; and for the rest of the time he had been kept on splints. The knee-jerks had disappeared for the present, and he was waiting to see if they would return. The spasticity had largely disappeared from the calf muscles and from the adductors, but the hamstrings still showed some tendency to spasticity. The tendency to the scissor gait had been lost, and he could walk a little if supported. He did not doubt that if Professor Foerster's after-treatment were followed, as carefully as it had been in his cases, the result would be comparable. There was no longer the associated movement which he had before; it had not been possible formerly to lift one leg without its fellow almost immediately following. The legs could now be got out quite straight, but on releasing them they still had a slight tendency to revert to their former posture. The patient still showed increased reflex excitability on pressing over the adductor magnus tendon, but to nothing like so marked an extent as before the operation. He hoped to have an opportunity of showing him again when he had arrived at a further stage of his evolution. Professor Foerster had said the outlook in this case was favourable, so perhaps in time the child would be able to walk as well as those whose striking pictures had been shown to them that evening.

Mr. C. A. Ballance, M.V.O., said he would like, before mentioning the two or three cases which he had had bearing on the present discussion, to say what a great personal pleasure it had been to him to listen to the splendid scientific address of Professor Foerster, and to express the hope that in the future the Surgical Section of the Royal Society of Medicine would learn the great advantage of inviting distinguished confrères from foreign countries from time to time to join in the discussions, thus giving English surgeons at first hand an account of their work in the way in which Professor Foerster had done that day.

The first case in which he divided a posterior root occurred many years ago in a patient under the care of Dr. Risien Russell, a woman aged about 35, who had suffered from herpes in the distribution of the first dorsal root along the inner border of the forearm. When he saw her there were certain scars and marks of the previous herpes, which remained after the attack had subsided. But since the subsidence of the attack she had suffered in the distribution of the first dorsal root from considerable pain, and the area was hyperæsthetic. He divided the first dorsal root on that side, and in doing so he relieved the pain completely. It was at the time that Professor Sherrington was working at the sensory distribution of the posterior roots, and he came several times to examine that patient. Professor Sherrington determined that there was a fourth form of sensation, which he had never before been able to discover. The exact details of that he would not enter into, but in the periphery of the anæsthetic area the patient was unable to distinguish a small burn caused by the contact of a red-hot wire as a burn, but appreciated it only as a tactile sensation. He did not know whether the Professor had made any further experimental investigation on the matter, but at that time he was greatly interested in it, and he remembered his desire to publish an account of that new form of sensation. The result of the operation was completely successful—the hyperæsthesia disappeared, as also the pain, and the patient was cured.

The second case was that of a lady whom he saw in consultation with Sir David Ferrier and Sir Victor Horsley. She had hyperæsthesia of the sole of the right foot corresponding to the distribution of the posterior root of the first sacral nerve. She was not able to get about; the hyperæsthesia was not absolutely constant, but whenever it was present in the sole of the foot it prevented her from walking. At that time she was averse to operation, but later on Sir Victor Horsley divided the posterior root of the first sacral nerve of that side, and he (Mr. Ballance) heard a year afterwards that she was cured. There was at first some difficulty in getting about, because of the anæsthesia of the sole of the foot, but he thought that had been overcome, and all the distressing pain and hyperæsthesia of the sole was got rid of.

The third case was that of a girl, aged 22, whom he saw last year. When the patient was a year old she had tuberculosis of the fourth metacarpal bone of the right hand, and when he saw her there was a considerable scar over the region of the fourth metacarpal bone. For many years she had had pain in that scar, and while that pain lasted she was unable to do what she was accustomed to. She was neurotic, and since 1898—ten years before he saw her—she had suffered from epilepsy. The question was whether that painful scar had anything to do with the epilepsy. Convulsions occurred about four times a year, and for many years she had been under treatment by bromides. A number of physicians had seen her, and two years before he saw her Sir Victor Horsley had divided the dorsal branch of the ulnar nerve which is distributed to the region of the scar over the fourth metacarpal bone. The operation gave some relief at first, but appeared to have resulted subsequently in increased pain; and for eighteen months before he saw her she had been in great distress owing to the pain in the scar, which pain had spread up the arm in the distribution of the eighth cervical root. He divided the eighth cervical root, and there was no difficulty in the operation. Dr. Purves Stewart was present at the operation, and they took the opportunity of stimulating the anterior roots of the seventh and eighth cervical nerves, and also the anterior roots of the first and second dorsal nerves. On stimulation of the anterior root of the eighth cervical nerve the triceps acted very powerfully, the pectorals contracted, there was no flexion of the fingers, and there was extreme hyperpronation of the whole limb. When the first dorsal anterior root was stimulated there was flexion of all the fingers, ulnar flexion of the wrist, the triceps contracted, as did also the rhomboids, and there was no hyperpronation. On stimulating the seventh cervical anterior root there was extension of the elbow, the pectorals contracted, and there was hyperpronation, so below the cervical roots there was no hyperpronation, and there was not any flexion of the fingers until one reached the first dorsal root. The second dorsal anterior root was also exposed and stimulated, and in stimulating that there was only contraction of intercostals. The patient was immensely relieved. Six months after the operation she had had no more fits, and had been able to resume her ordinary occupation. He had not heard of her since, and he regretted, in view of the present discussion, that he had not written to find out her later history.

The fourth case was one which he was unable to complete. It was that of a man who came to St. Thomas's Hospital last winter. He weighed nearly 20 st. He was a railway man, and some years previously he had met with an injury which necessitated amputation in the lower third of the right thigh. During the last two years he had suffered from intolerable pain in the stump. Many drugs had been used, the nerve had been injected with alcohol, the nerve had been excised as far as it was found in the stump, right up to the sacrosciatic foramen; therefore, after watching him for some time and being satisfied that he had great distress and pain, he concluded that the only course was to attempt to divide the posterior roots corresponding to that limb. He determined on division of the posterior roots of the third, fourth and fifth lumbar, and the first and second sacral. In that way he aimed at controlling the sensory distribution of the lower limb without interfering with the sensory distribution of the genital region or the anus. After anæsthetization, he got down through the eight inches of fat to the lumbar spine, and managed to get away the laminæ of the second, third, fourth and fifth lumbar vertebræ. At that stage the patient's condition was so bad that infusion was necessary, and the wound was closed. Great difficulty was experienced in reviving him: indeed, for some time he was thought to be dead. Some weeks afterwards the wound was re-opened with the intention of opening the dura and dividing the posterior roots. But before the dura was reached the man's condition became critical, and so the operation could not be completed; he did not feel justified in proceeding with what might have been a brilliant operation, as there was a prospect of it killing the patient. The man had now left the hospital, and he heard from his doctor that he was still in great pain. Still, there was an absence of justification, in the light of the previous experience, in attempting further operative procedure.

Dr. PURVES STEWART remarked that the position of the physician who ventured into a meeting of surgeons was analogous to that of Daniel in the lions' den; but, whereas Daniel was cast into the lions' den, the physician had attended here of his own free will. He desired to support everything which Mr. Ballance had said about the papers which had been read. His excuse for saying anything was that he had had three cases in which Professor Foerster's operation had been carried out. The first case was one of gastric crises, the patient being a billiard marker, aged 40, who had inveterate vomiting during a number of months, with profound emaciation. That was in August, 1909, at which date his colleague, Mr. William Turner, of the Westminster Hospital, divided the thoracic roots from the seventh to the tenth inclusive on both sides. The result was the production of a band of anæsthesia such as Mr. Groves's case showed, from the xiphisternum to the umbilicus. There was immediate and complete cessation of the gastric crises, he rapidly put on weight, and became a normal member of society. But he subsequently developed another occupation trouble, namely, alcoholic poisoning, which was, perhaps, almost inevitable in a man of his occupation, because part of his remuneration for scoring at billiards was, doubtless, offered in liquid form. He saw him a year afterwards, when he was still free from gastric crises, but was obviously suffering from the other disease. Since then he had not known of him for over a year.

The second case was one of tabetic lightning pains, chiefly in the legs, in the area of the second lumbar roots. It occurred at the West End Hospital for Nervous Diseases, and Mr. Wilfred Trotter divided three roots, of which the central corresponded to the site of maximum pain. He divided the first, second, and third lumbar roots, but the result was practically *nil*, for the pains continued, as one would almost expect.

The third was a case of spastic paraplegia in a veterinary surgeon, who many years ago had a syphilitic softening of his cord, and, as a result, was spastic in his legs, without sensory changes, but with marked bladder trouble. Mr. Trotter divided the first, third, and fifth lumbar roots on the same side, and the second and fourth lumbar and first sacral on the other side. In no case did he divide two successive roots on the same side. The result was that, whereas previously the patient walked by placing one foot just in front of the other, he could now take steps two or three times as long, and the spasticity was diminished. He was so pleased with the result that he desired to come back to have more roots divided, so that he could take longer strides still.

Professor Foerster's operation was a brilliant one, but, like all other good operations, it was suitable only in selected cases, as the Professor himself had pointed out. The cases *par excellence* where this operation was indicated were those of gastric crises. As Professor Foerster had shown in numerous papers, a gastric crisis was a storm of a reflex arc, or of several reflex arcs, and if that arc were cut the storm ceased, as there was no longer a path along which it could travel. With regard to tabetic pains matters were different. Tabes was not simply a disease of the roots, but of the continuation of those roots within the spinal cord. Division of those roots for pains in tabes could not be expected to cure the disease; the central parts of the roots remained and might still cause pain. Moreover, the disease was a progressive one, and one could not expect to cure it by dividing certain sensory roots, any more than epilepsy could be cured by dividing motor roots. With regard to the treatment of ataxia, to employ Professor Foerster's operation for ataxia was unjustifiable; it would tend to increase it. In cases of spastic paralysis, one must be very cautious not to employ the method indiscriminately in all cases of spastic paraplegia, because in every case there were two elements, spasticity and paraplegia, and the operation could only relieve the spasticity. Therefore, if a patient was spastic and paralysed, and if, on anæsthetizing his cord with stovaine as suggested, he had still a considerable degree of voluntary power left, it might be worth while dividing the roots, and he would have that degree of voluntary power of which his pyramidal tracts were capable. But if the patient were idiotic, or if he were too severely paralysed, with the pyramidal tracts almost completely degenerated, the operation should not be done. Athetosis was also a contra-indication, as it meant profound degeneration in the brain centres. And, as the Professor himself pointed out, disseminated sclerosis, or other progressive central disease, was a contra-indication of operative measures.

Mr. D. M. AITKEN remarked that Professor Foerster had laid stress on the after-treatment in the cases under discussion, especially of spastic paraplegia. He had been watching cases of the kind for ten years, since he was house surgeon to Mr. Robert Jones, of Liverpool, who treated such cases on orthopædic lines, in which after-treatment was of great importance. He asked where Professor Foerster would draw the line between cases suitable for purely orthopædic treatment and those where one should at once proceed to the section of the roots. He wished to thank Professor Foerster for the marvellous demonstration he had given of cases which had recovered, though they were so severe. A point to which both Professor Foerster and Dr. Purves Stewart referred was the occurrence of reflex spasm. Those were cases which one treated orthopædically, but in which Professor Foerster divided the roots. The primary source of that spasm was what many would like to know with certainty. Was he right in assuming that that spasm came from the muscles themselves? If so, he would like to know why that should not be more generally overcome by division and full stretching of the spastic muscles? He asked those questions purely out of a desire for information, because he had seen some fifty such cases treated by orthopædic methods, and in many there was quite good recovery, especially in the lower limbs. With regard to Professor Foerster's marvellous fourth case, of which he showed a photograph, the excellent abduction of the thumb was a remarkable feature. Spitzy, of Gratz, had done much in transplantation of nerves in connexion with these cases. As far as he (Mr. Aitken) could make out, Spitzy experienced the same difficulty in overcoming the spasm of the adductors of the thumb as was met with in treating these cases by orthopædic methods. The spasm of fingers and wrist could be much more easily dealt with. He would be glad to know how soon the Professor succeeded in getting these children to use their thumbs.

Dr. HINDS HOWELL said he had at present under his care a tabetic with very severe gastric crises, who had been treated by every conceivable sort of medical method for his gastric crises, and finally, last summer, he decided to have the posterior roots divided. Pain seemed to be the predominant feature of the attacks, the vomiting appearing to be secondary. The pain was somewhat relieved when the patient made himself sick. He had the ordinary abdominal hyperæsthesia found in such cases, and the point of maximum pain was just above the umbilicus. The posterior roots, which were divided by Mr. Warren Low, at the Great Northern Hospital, were the eighth, ninth, and tenth on both sides, not the seventh because of the situation of the pain, and because the vomiting seemed to be incidental to the attack of pain. The man made a good recovery, but the gastric crises were not permanently benefited, though they were improved for six weeks. The seat of his pain was now in the totally anæsthetic area. Possibly sufficient roots had not been divided, but it did not necessarily follow that the gastric crises were simply due to irritation of the root outside the spinal cord. As Dr. Purves Stewart said, there was degeneration going on inside the cord. He did not think the gastric crises could be altogether separated from the lightning pains, as Dr. Purves Stewart had seemed to suggest. The operation was not invariably successful for the lightning pains, and it could not be expected to be always successful in gastric crises. The posterior roots should not be divided in tabes until the question had been considered very carefully indeed and other methods of treatment had failed.

The President desired to ask Professor Foerster whether he would advise, as a routine treatment, division of the posterior roots in Pott's disease where there was much spasm. It was an operation which one had frequently to perform where spasm was a very marked feature, and his experience had been that it almost always subsided. He would feel some hesitation in opening the theca when the field of operation was bathed in tuberculous pus.

Professor Foerster, in reply, said that he would only advise division of the posterior roots in cases of extreme spasticity. Of ten cases operated upon by Küttner, six had been treated for a long time, but without success, by ordinary orthopædic means. It was very difficult to decide immediately if a case less severe was suitable for division of the posterior spinal nerve-roots, and it was right in every case to try ordinary orthopædic treatment first. As to the influence of the division of posterior cervical nerve-roots in cases of spastic paralysis of the arm, he could only say that the spasm of the adductors of the thumb had been removed by division of the posterior roots. Dr. Spitzy, of Gratz, had proposed to do implantation of the median nerve into the radial, which was a very good idea for the relief of the paralysis of the muscles supplied by the radial nerve. In cases of Pott's disease he thought one should only try division of the posterior roots when one could be sure that the disease was not going to progress. If there was not a feeling of certainty that the tuberculosis of the vertebral column was cured, the posterior spinal nerve-roots should not be divided. The only case of the kind reported was one in which they had first tried laminectomy of the cervical region, because the vertebral column was defective in that region; but there was no effect, spastic paraplegia was of the same degree as before. Later, they tried division of the posterior nerves in the lumbar region, and the spasticity completely disappeared afterwards, so that the child walked again.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FOURTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1910-11

THERAPEUTICAL & PHARMACOLOGICAL SECTION

1805-1834
1907

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1911

Therapeutical and Pharmacological Section.

OFFICERS FOR THE SESSION 1910-11.

President—

A. R. Cushny, M.D., F.R.S.

Vice-Presidents—

Sir Lauder Brunton, Bt., M.D, F.R.S.
W. E. Dixon, M.D., F.R.S.
W. T. Parker Douglas, M.B.
J. Gray Duncanson, M.B.
John Ince, M.D.
Sydney Ringer, M.D., F.R.S.
Charles Sangster.
Sir James Sawyer, M.D.

Hon. Secretaries—

H. Charles Cameron, M.D.
H. H. Dale, M.D.
J. Gordon Sharp, M.D. (*Foreign*).

Other Members of Council—

F. A. Bainbridge, M.D.
A. P. Beddard, M.D.
James Calvert, M.D.
Robert Capes.
George S. Haynes, M.D.
Arthur F. Hertz, M.D.
Robert Hutchison, M.D.
James Mackenzie, M.D.
William Murrell, M.D.
Harrington Sainsbury, M.D.
E. I. Spriggs, M.D.
Ralph Stockman, M.D.
Frederick Taylor, M.D.
R. B. Wild, M.D.

Representative on Library Committee—

Herbert S. French, M.D.

Representative on Editorial Committee—

J. Gray Duncanson, M.B.

THERAPEUTICAL AND PHARMACOLOGICAL SECTION.

CONTENTS.

October 18, 1910.

PAGE

Presidential Address. A Plea for the Study of Therapeutics. By A. R. Cushny, M.D., F.R.S. 1

November 15, 1910.

On the Action and Uses of Sulphur and certain of its Compounds as Intestinal Antiseptics. By Robert B. Wild, M.D. 13

The Therapeutic Use of the Digitalis Group. By H. Hume Turnbull, M.D. ... 25

January 17, 1911.

The Rôle of Auto-inoculation in Medicine: A Plea for its Rational Extension. By Clive Riviere, M.D. 39

February 21, 1911.

On Drugs used as Local Anæsthetics in the Mouth. By F. R. Smyth, M.R.C.S., L.D.S. 51

March 21, 1911.

An Inquiry into the Value of Rectal Feeding. By W. Langdon Brown, M.D. .. 63

Reactions of Arteries to certain Drugs. By Douglas Cow, M.D. 77

April 11, 1911.

PAGE

Excitation and Section of the Auriculo-ventricular Bundle. By W. E. Dixon, M.D., F.R.S. 97

Ergot: A Clinical Experimental Study. By J. Gordon Sharp, M.D. 114

May 16, 1911.

An Investigation of the Action of Aconite on the Pulse-rate. By Frederick W. Price, M.D. 151

The Effect of Atropine on the Pulse-rate in Cases under the Influence of Digitalis. By M. D. Silberberg, M.D. 192

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

John Bale, Sons & Danielsson, Ltd., 83-91, Great Titchfield Street, London, W.

Therapeutical and Pharmacological Section.

October 18, 1910.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

A Plea for the Study of Therapeutics.

WHEN the future historian of medicine reaches the nineteenth century, he will undoubtedly be struck by the great change in the attitude of the profession towards therapeutics which occurred in it, though he may find difficulty in understanding what that attitude is in the latter half of the century. In the earlier years medicine was nearer to the medicine of the sixteenth century than to that of the twentieth. In therapeutics the same measures were in force for the most part as prevailed throughout the eighteenth century, and it was still possible to found a new school of therapeutics on purely deductive arguments, just as had been the case for centuries previously. The theory underlying the practice of medicine in those days was, in fact, a purely philosophic conception; and the value of a remedy was determined by the statistics of mortality under its use and by the subjective symptoms. Optimism pervaded therapeutics, and the chief anxiety of the physician seemed to be to persuade the patient to swallow enough of his supposedly healing compounds. There appears indeed to have been too great faith in the potency of remedies to encourage any critical examination of their effects, and no attempt was made to eliminate casual factors, such as the effects of rest or of suggestion.

This faith in the usefulness and necessity of drugs received several rude shocks in the early part of the century. One of these was the success of homœopathy, under which patients recovered without the use of drugs in appreciable quantities. This apparently first awakened the profession to a doubt of the necessity for the continuous and excessive dosing then in vogue; and the introduction of various new methods of physicial examination at about the same time rendered the physician less dependent on the subjective symptoms, and showed that a supposed improvement was often unattended by change in the disease and was purely fictitious.

The therapeutic pendulum had begun to move towards a position of moderation as compared with the heroic measures of the beginning of the century, when its swing received a tremendous acceleration from the rapidly developing school of pathological anatomy. The attitude of the anatomist and of his ally, the pathological histologist, has always been antagonistic to therapeutics. It is difficult to stand by the cadaver and, in the presence of the gross alterations of structure there presented, maintain the efficacy of small quantities of chemicals. And as the anatomical school of pathology gained ground, the practice of therapeutics retreated from the position it had previously occupied. The physician lost faith in his treatment and prescribed drugs, not in the belief that they would counteract the disease, but because he felt that they could not do any harm and would satisfy the demand of the patient for some measure of relief. In the wards the placebo reigned supreme, and large use of expectant treatment was considered that most compatible with the position of a teacher of medicine. The text-books of medicine devoted pages to pathological anatomy and to minute points in diagnosis, while treatment was dismissed in a couple of lines. This nihilistic attitude was of course possible only in the high places of the profession. The ordinary practitioner, facing the urgent necessities of disease, continued in the faith of his fathers, and, uninstructed in clinical therapeutics, fell an easy prey to the new methods of advertising adopted. Curiously enough, the mistrust of known drugs was often accompanied by a childlike credulity in regard to other measures. How often does a florid condemnation of the use of antipyretic drugs end in a eulogy of the cold bath, or a criticism of all medicines lead up to the virtues of the electric battery, high-frequency current, or other panacea of the time!

The violence of this storm now seems to have passed. It is now recognized that pathology is not merely anatomy, and that even the most hopeless lesions must have arisen from, and be dependent on, some abnormal condition, which may be susceptible to treatment; and that while the prospect of curing the fully developed lesion may be very remote, there is hope that clinical means may prove efficient to change the environment so as to prevent further progress of the disease, or at the worst to preserve life and ease pain more perfectly than is possible at present.

In the meantime a new departure had been made in the study of drugs. It became necessary to subject their action on the living tissues to the rigidly objective study which was proving so fruitful in physiology, and the new school of pharmacology was founded in the middle of last century. The atmosphere was not favourable to study of the clinical effects of drugs; in addition, the foundations of the subject had to be laid on more definite observations than were possible in man, and the pharmacologists abandoned the hospitals for the laboratory. The subject was studied without any immediate bearing on its usefulness in medicine and threatened to become completely divorced from practical medicine, as was the physiology of that time. How could it profit the practitioner to know the effects of drugs on the frog's gastrocnemius? In the same spirit the practical engineer probably scoffed at the experiments of Galvani, if he heard of them, little dreaming that they would lead to the electrical industry of to-day. And I am not without hope that the principles of pharmacology laid down by the earlier workers may in time lead to an equal illumination in the practice of medicine. Hitherto the experimental study of the action of medicines has tended rather to dispel the darkness of mediæval therapeutics, which persisted until late in the nineteenth century and still throws fog shadows on practice. Many remedies formerly in esteem have failed to maintain their position under its dry light, and many therapeutic views have proved untenable. On the other hand, the experimental investigation of the action of drugs has in many instances confirmed their use in therapeutics by demonstrating and elucidating their effects under conditions as trustworthy as those of the physical or chemical laboratory. And it has added largely to the resources of the medical man by the introduction of new drugs and new methods, as evidenced by the fact that, of the many valuable drugs introduced in the last half century, only one has been found entering by any other way than through the experimental laboratory.

For nearly fifty years, then, pharmacologists have dwelled apart from the practice of medicine, but to deny their influence on practice or to belittle the usefulness of the study is merely to betray ignorance of the history of medicine. Great general views have been obtained, and in some directions, at any rate, merely the details of the picture have to be filled in. The question may be raised, however, whether the advance of medicine does not demand a more active participation on their part. And my own feeling is that it is time for them to leave the cave of Adullam—or shall I say the tent of Achilles?—and apply the principles learnt in the laboratory more directly to clinical treatment. Far be it from me to suggest that the experimental field is exhausted, or even approaches exhaustion. Rather would I say that practical therapeutics needs to be brought to the higher state of cultivation which has been attained by the experimental science. Here the problems are more complex than in the laboratory, and the opportunity of observation is more limited. But, making every allowance for these difficulties, there is a sad want of accurately observed and recorded results in practical therapeutics, and much discordance in results. The habit of mind which is acquired in the laboratory must be introduced into the clinic if the art is to be brought to the level now reached by the science. Let me illustrate what I mean by an example: the usefulness of ergot in childbirth has been a question in practice for many years, though there is no deficiency in opportunity here, for some practitioners make almost a routine practice of its administration. Yet is it still a moot point whether ergot should be given to arouse and strengthen the pains or whether its usefulness is limited to maintaining the tonic contraction after the expulsion of the child. If a problem like this were put to the experimental investigator, he would probably begin by counting the rate of the pains before and after the drug was administered, noting their duration and other features; and when I began to look into the question, I expected to find that each side would have series of such observations, but that these conflicted with each other, and supported the position of each of the disputants. But I could find in the whole literature no evidence that anyone had ever contemplated such an investigation. Here was a drug, given in thousands of cases each year, the action of which could be investigated with apparatus no more complicated than an ordinary watch, and the action and usefulness of which were in doubt, yet no such investigation stands recorded, the nearest approach to it being the observation that "the pains were stronger and more frequent."

This is merely an example of what is true for a great number of therapeutic problems. No exact records of the effects of drugs in man are available. The physician spends any amount of time and patience in examining the condition of the patient, in making his diagnosis, then writes his prescription, and thereafter limits his inquiry to the most general features, depending largely upon the subjective statements of the patient, and seldom attempting to measure the amount of improvement or to eliminate the other factors than the drug. Yet for progress in medicine we must have accurate measurèments of the changes occurring, whether drugs be used or not. Lord Kelvin says: "When you can measure what you are speaking about and express it in numbers, you know something about it ; when you cannot express it in numbers, your knowledge is of a meagre and unsatisfactory kind." The action of drugs must always be quantitative, not qualitative. We cannot change the nature of the activity of an organ, but only the degree of its activity. And in order to estimate the effects of drugs we must be able to measure the activity of the organ under their effects in terms of its activity before their administration. In the laboratory we pay little attention to observations which are not substantiated by balance and foot-rule ; apparently the observers of the effects of drugs on the sick are content with "knowledge of a meagre and unsatisfactory kind." This is, it seems to me, one of the causes of a slight antagonism which is sometimes observable between the experimental observer and the clinical exponent of therapeutics. The laboratory-trained man expects accurate measurements ; the clinician is unable to give them. He is unable or unwilling to devote the attention necessary to obtain accurate results, yet he is ready to express an opinion on the values of drugs, and is often to be heard expressing doubt as to the usefulness of experimental pharmacology. And the absence of accurate clinical records, such as we can use to substantiate our laboratory findings, is seriously hindering the advance of therapeutics. We of the laboratory do not know what the clinicians want, how our drugs should be modified so as to meet the clinical requirements. Returning to the subject of ergot again, there have been three principles isolated from this drug by Barger, and Dale has shown that all of them differ in action in some degree. Which is the one the obstetrician needs, or are they all equally valuable ? Any one or all of them are offered ; but the laboratory findings do not guide us to say which is the essential, because we have no accurate records of the action of ergot in labour, and we do not know what exact feature the obstetrician desires to elicit. Among the heart drugs, in which I am especially interested—digitalis, squill, and strophanthus—the position is the same. No accurate results of their exhibition are on record. They are generally classed together, and one prefers strophanthus, another digitalis, on grounds which are quite "meagre and unsatisfactory." They are reputed to differ in action to a considerable degree, but it is quite unknown in which respects ; yet they are in constant use in the sick room, and their effects are quite readily measurable. No one has even determined the dose of the ordinary preparations which induce nausea.

The same is true in regard to most other groups : the principles underlying their action are known as far as can be determined experimentally, but the details of their effects in the sick bed have never received accurate examination. It is true that a few researches of this character appear. I may cite as an example the admirable work by Stockman[1] on the salicylates read before this Section two years ago; but these are comparatively rare.

It may be urged in reply that clinical observations cannot be attended with the exactitude attained in the laboratory. Yet of late years, hours and days are devoted to the laborious and prolonged estimation of the effect of vaccines on the opsonic index. No more time and no more labour are required for exact observations in regard to the action of better known and (if I may venture to say so) more reliable remedies than vaccines.

I have lately had the opportunity of watching the action of medicines on the sick through the kindness of my friend Dr. McKenzie, who has been willing to consider the suggestions of an experimental pharmacologist in regard to treatment. I should like to give you an account of some of the problems which we have met, and which we are attempting to solve. I find that the work in the wards can give results quite comparable to the laboratory in general accuracy, and that questions which have long puzzled us there may be solved without any great difficulty. The results come more slowly, for one has to wait for the suitable case for inquiry; but progress is made. As an example of the comparative results obtained in man and animals, I should like to show two tracings on the screen, the results of the action of amyl nitrite on the blood pressure. The one is obtained from a dog with the ordinary recording apparatus and the mercury manometer; the other from a case of angina pectoris, in which the blood pressure was taken during a spasm by means of Martin's arm bandage; the numbers were recorded by an assistant along with the time, and the curve was drawn on squared paper afterwards. You will notice the very close correspondence in the curves and the disappearance of the pain when the pressure falls, and its reappearance as the pressure returns. Very often amyl nitrite is said to be ineffective in angina pectoris, and I am not prepared to deny the impeachment. But one wonders whether the drug was given in sufficient quantities in these cases. You will notice that three capsules were inhaled before the fall set in, and nothing serious happened, though the maximal dose of the British Pharmacopœia was considerably overstepped. This is a case in which the spasms are very frequent, and amyl nitrite relieves for a short time, but not long enough. It is probable enough from the curve that the spasm is of central origin, that it is really a sort of convulsion of the vasomotor centre. And the sudden fall which occurred when we were obliged to give chloroform, and which was not attended with loss of consciousness, but only with some haziness of perception, suggested that we might treat the condition by lessening the abnormal sensitiveness of the vasomotor centre, rather than by drugs acting on the vessels. With this idea bromides were prescribed, and the number of attacks fell next day, and disappeared when the bromide action took full effect. The ordinary treatment of angina pectoris with nitroglycerine, &c., was not adopted here, because the symptoms pointed so definitely to a central disturbance that it seemed more reasonable to begin by reducing this directly.

---

[1] *Proceedings*, 1909, ii, p. 31.

Another patient with vasomotor disturbance presented himself with the statement that he became giddy when he rose from bed, but was quite well as long as he lay down. The heart seemed to be fairly efficient, and the inference was drawn that when he stood up the blood was driven into the abdomen and the brain starved. This was confirmed by a simple experiment: a tight bandage was pinned round the abdomen, and the patient felt no giddiness on rising. This indicated that the weakness was due to the slackness of the abdominal vessels, which offered too little resistance to the blood flow, which poured into the abdomen and left the brain insufficiently supplied; this in turn suggested a low excitability of the vaso-constrictor centre. The indications were a stimulant to this centre, and strychnine was prescribed with good effects. The man is now at work. There has been current for some time a statement that strychnine is a heart remedy, for which we could find absolutely no support in experimental work. Are all the circulatory cases in which strychnine is said to be of value of the same nature as this one I have described? Until more accurate records of the cases in which strychnine is said to "tone up" the heart are available, I think we are justified in regarding it not as a heart but as a vasomotor remedy, and the symptoms are entirely different in nature.

Our chief interest, however, has lain in the group of cardiac tonics —digitalis, strophanthus, squill. The preparations we have used were the tinctures, as it seemed more advisable to investigate the action of forms in universal use rather than "pure principles," which vary as much in character as the Galenical preparations, and, in addition, are rarely seen in practice. Opinions vary very much in regard to the relative usefulness of these, the prevailing belief being that strophanthus acts less on the vessels than digitalis, and that squill acts more on the kidney. The first point to determine was whether the three tinctures were approximately equal in their action on the human heart, as is suggested by the doses in the British Pharmacopœia being the same. I had standardized the three carefully on the frog's heart (for this purpose the minimal dose was taken, which, injected into a lymph sac, sufficed to arrest the heart in one hour), and found them of average quality, the minimal doses per 100 grm. frog being in the case of digitalis 0·8 cc., that of squill 0·6 cc., and that of strophanthus 0·04 cc.; that is, the tincture of strophanthus was about twenty times as strong as that of the digitalis, or fifteen times as strong as that of squill when applied to the frog's heart.

The activity of the several tinctures in man might be measured by the minimal amount per day which was sufficient to improve the symptoms, or by the minimal quantity which induced undesirable effects. As a matter of fact, we took as our index the quantity which kept the patient in the best condition. This was practically in all cases the maximum amount which could be taken without unpleasant symptoms being induced. Almost invariably we found that while improvement could be started after a time by small quantities—e.g., ♏v tincture of digitalis three times a day—much better results followed an increase to ♏xv four times a day.

After this dose of 1 dr. per day of tincture of digitalis there was always the risk of unpleasant symptoms developing, but the amount could often be continued for weeks without any such results, and patients often complained when it was reduced in order to find if the maximum was actually the optimum dose. When the optimum dose of digitalis had been reached, and no further improvement seemed to promise, we stopped the treatment. In a number of cases, relapse to the former condition followed after about a week, and then tincture of strophanthus was begun in the same way and its optimum dose ascertained. Finally, squill was substituted for strophanthus, or a return was made to digitalis. The relative usefulness of these three was thus elicited in each case, and it was found that in most cases each of the three was capable of inducing the same degree of improvement; but the dose required was not the same in each. As I have mentioned, in our typical cases, tincture of digitalis acted best in quantities of 1 dr. per day, but to elicit the same improvement tincture of strophanthus and tincture of squill had to be given in doses of 2 dr. per day. It is to be noted that the British Pharmacopœia dose is the same for all three. Our results show that that of squill and strophanthus should be twice that of digitalis, and we would suggest that the dose of the latter should be the higher limit given by the British Pharmacopœia.

The effects of treatment in our cases were very carefully observed, and I may remark that in every case a period of rest and observation preceded treatment. The subjective symptoms were carefully noted, and in many instances corresponded very closely with the results of physical examination. The condition of the heart before and during treatment was examined by the ordinary physical methods and by Mackenzie's graphic methods, which are well known. The degree of cyanosis was estimated with some success by comparing the colour of the lips with a series of coloured papers varying from red to deep red-blue. Particular attention was paid to the results of a definite amount of exertion on the heart and respiration, the patient being sent up a certain number of steps and examined immediately afterwards.

As regards drawbacks to the use of these remedies, we have noted such in all of them. The first symptoms of which patients complain in our experience arise from the digestive tract. As soon as the optimal dose is exceeded under digitalis, nausea appears, the appetite fails, and slight vomiting may occur, or severe headache is complained of. These all disappear within twenty-four hours if the dose is reduced. In the cases of strophanthus and squill, the patient complained of diarrhœa more often than of nausea, though the latter appeared occasionally. This also tended to appear when the optimum dose was exceeded. And thus the lowest dose of strophanthus and squill which induces these untoward symptoms is also twice that of digitalis. For good or evil, the tincture of digitalis is twice as potent as that of strophanthus or squill.

The nausea and vomiting induced by digitalis has generally been regarded as due to its irritant action on the gastric mucous membrane, but some authorities have been disposed to ascribe it to central action. The fact that strophanthus and squill cause diarrhœa rather than vomiting seems to support the former view, for it is difficult to conceive of their selecting different central areas in this way. In some cases in which helleborein and cynotoxin were given, they also tended to cause diarrhœa rather than gastric symptoms.

As regards untoward symptoms from the heart, the commonest was partial failure of conduction through the bundle of His. This was elicited in a number of cases, and in others in which it was not present in rest it appeared on slight exertion. In other instances, excessive sinus slowing was met with, or could be induced when the patient was resting after going upstairs. In some cases in which these effects were induced by digitalis, they followed the use of strophanthus and squill also, when twice the dose had been given.

In none of the cases was there any rise of blood pressure from these drugs, and in some there was a slight fall, as has been observed by previous investigations. Yet the efficiency of the circulation and the heart was undoubtedly increased, as was manifest in the disappearance of cyanosis, œdema and other symptoms of imperfect aeration and nutrition of the tissues. The pathology in these cases is probably as follows: the heart is inefficient and the supply of blood to the brain accordingly deficient, and this arouses the vasomotor centre to activity, leading to constriction of the peripheral arterioles and a fair blood pressure in the brain. The inefficiency of the heart thus leads to peripheral constriction, and as the efficiency is improved under digitalis this constriction abates and the blood pressure may remain unchanged or even fall.

The characteristic slowing of the heart was induced in our cases, the pulse falling from 90 to 100, or more, to about 70 or 80. This rate seemed to be that at which the patients were most easy, and in several cases in which the rate afterwards rose under smaller doses they asked that the former dose might be given again.

A number of other points observed in the action of this series would require more detailed description of individual cases than time permits at present. The effect of digitalis on the mammalian heart is composed of two definite and, to some extent, opposing factors—one the stimulation of the inhibitory apparatus, the other the effect on the muscle. In man the results of the first factor are obvious in the slowing, in the auriculo-ventricular block occasionally, or in sinus arrhythmia. The muscular factor is less obvious; the apex-beat and pulse are stronger and fuller, but these cannot be dissociated from the slowing of the pulse, which gives more time for recuperation between the beats. The diminution of the heart's dullness may also suggest action on the muscle, but it may be the indirect result of changes in nutrition, and, in addition, cannot be measured with any approach to accuracy. We have not been able to devise any method in which the effects of the two factors may be differentiated when both are present; and in this the clinical experience follows the experimental, for the vagus effects of digitalis could be shown and measured in mammals many years before the muscular. The problem is of great practical importance as well as of theoretical interest. For the muscular action is the distinguishing feature of digitalis. A number of other drugs act in practically the same way as far as the inhibitory factor is concerned, and might be substituted for digitalis if this is the essential feature in its action. On the other hand, if the muscular factor is the necessary one, as I have held, we may be able to lessen the prominence of the unnecessary inhibitory one. In other words, Nature has supplied us with a mixture of inhibition and muscular action in digitalis. Are these present in the most desirable proportions for each and every case of heart disease, or might we not improve on nature by increasing one or the other of them according to the needs of each patient? Some authorities have always regarded the inhibitory as the important factor, but this seems to ignore the argument that inhibition may be induced by other drugs which have not been shown to have the specific effects of digitalis in heart disease, and I have always regarded the muscular action as the essential factor in the effects. This faith has been a little shaken by our investigation, in which the subjective symptoms, and some of those obtainable by physical and graphic examination, were always improved coincidentally with the slowing of the pulse. In cases of auricular fibrillation, in which digitalis is of especial benefit, both theory and practice point to the inhibition factor as of importance. Here the heart is disorganized by the ventricle receiving irregular and too rapid impulses from the fibrillating auricle, and it would seem probable that by reducing the number of these by inhibiting their passage through the bundle of His, relief might be given to the labouring ventricle. In order to determine this point, we propose to treat some of these cases with drugs, which, resembling digitalis in their inhibitory action, have no such effects on the cardiac muscle, the one we have chosen to commence with being aconite. Our experience is too limited as yet to lay the results before you, but they do not seem to indicate that this drug can take the place of digitalis in these cases; that is, the muscular action appears to be an essential factor, perhaps the essential factor in the therapeutic effect.

On the other hand, it would seem desirable in certain cases to be able to elicit the muscular action without the inhibitory. For example, the use of digitalis has sometimes to be reduced, because it hinders the transmission of impulses through the bundle of His, and causes partial block. Or in cases of complete block, we may desire to accelerate the ventricle without inhibiting the auricle. Bachmann has suggested the use of digitalis in these cases, to elicit the muscular action in the ventricle. For such purposes a drug resembling digitalis in its muscular action, but devoid of its inhibitory factor, is desirable, and we have, therefore, taken up the study of helleborein, which has less action on the inhibitory centre than any other of the glucosides which I have examined in this respect.

By using these three—aconite, digitalis, and helleborein—in a series of cases, it seems probable that new light may be thrown on the uses and action of the digitalis series, and perhaps by proper combinations of them we may be able to improve on the natural combination of inhibition and muscular action. The investigation promises to be a long one, and I have brought it before you as an example of the problem which lies before us in practical therapeutics rather than as one on which we have already reached results.

In conclusion, let me once more invite the experimental investigator, be he pharmacologist or physiologist—to return to the problems of the sick bed and abandon the attitude of aloofness from practical medicine into which he was driven by the necessities of the case half a century ago. He will find many problems awaiting him, which can be solved, I believe, by the methods of exact analysis which he has learned in the laboratory, the solution of which is a crying need for the progress of medicine. It is true that it is beyond the powers of most men to control a hospital service and at the same time to direct an experimental laboratory efficiently. In most cases, perhaps in all, one or other service will suffer from such overloading. But, on the other hand, I believe that in the future pharmacology will have to associate itself more closely with practical therapeutics than it has done hitherto. Wards will be considered an essential part of the equipment, just as much as the laboratory; and the activities of the two divisions will be co-ordinated as closely as possible in one department. The practical points developed in the laboratory will be applied directly in the clinic, while the problems of the sick bed will be carried to the laboratory for elucidation. In this department of pharmacology the duties must, of course, be apportioned, one member of the staff being responsible for the experimental, another for the clinical division, and the general direction of the department may be entrusted to the one or to the other according to circumstances. The essential point is that each should have some training in the other's methods and be inspired with interest in the promotion of the common subject, and that both departments should co-operate closely. I am convinced that some such arrangement would promote the advance of both the scientific and practical sides of the subject, and that without some such scheme of co-operation with the clinic, experimental pharmacology will fail in its purpose, or will, at any rate, remain as at present, poised between the sciences and the clinical arts, appreciated by neither and constantly threatened with complete neglect by both.

Therapeutical and Pharmacological Section.

November 15, 1910.

Professor A. R. Cushny, F.R.S., President of the Section, in the Chair.

On the Action and Uses of Sulphur and certain of its Compounds as Intestinal Antiseptics.

By Robert B. Wild, M.D.

From the pharmacological standpoint sulphur is a particularly interesting element, and hardly seems to have attracted the attention it deserves in the numerous studies in physiological and pathological chemistry which have been published in recent years. It is one of our oldest and most familiar drugs; it is an invariable constituent of all proteids; it is remarkably versatile in its allotropic modifications and active in its chemical combinations; it can either replace oxygen in compounds or combine with it in different degrees of oxidation; it combines readily with hydrogen and many metals. The compounds formed by sulphur are easily reduced or oxidized, so that the presence of sulphur in a compound confers upon it a chemical mobility which may be as valuable in the unstable equilibrium of living protoplasm as it is in gunpowder. Moreover, the compounds of sulphur with oxygen or hydrogen are singularly active as protoplasmic poisons. It is familiar to all that sulphur in contact with living protoplasm in the presence of oxygen is rapidly oxidized to form powerful antiseptic bodies, and that use is made of this property in the arts, and occasionally in surgery, when the application of sulphur to a foul ulcer is followed by sterilization, and, if the action is allowed to continue, by destruction of tissue as the sulphur becomes oxidized to its highest degree—viz., to sulphuric acid. In the absence of oxygen other combinations are formed, and when decomposing organic substances are present, hydrogen sulphide is produced either directly or through the medium of a metallic sulphide. This is what takes place in the alimentary canal, where the passage of sulphuretted hydrogen gas is the familiar and unpleasant result of the ingestion of sulphur or compounds containing it. Decomposing organic matter can even reduce the more stable sulphates, as is seen when sulphuretted hydrogen is produced in marshes, owing to the reduction of calcium sulphate in the soil by decomposing organic matter, such as dead leaves.

The object of this paper is to bring forward certain facts relating to the action and uses of sulphur and some of its compounds, and, upon these facts, to ask for a reconsideration of the position now assigned to sulphur in almost all the text-books of pharmacology, where it is considered simply as a mild purgative, in respect of its internal use. That it is a useful purgative in certain conditions is perfectly true, but my object is to show that purgation is not its chief action, and that its value in therapeutics is largely due to the local antiseptic action of the compounds formed from sulphur in the alimentary canal; also that other drugs and articles of diet, which give rise to similar compounds in the body, owe their value in medicine and dietetics to a similar intestinal antisepsis.

My attention was first called to this subject by noticing the popular use of sulphur in the children attending the out-patient department of the Skin Hospital. Certain classes of the general public—in the North, at any rate—look upon sulphur in its familiar combination with treacle, or taken in milk, as an almost indispensable "spring" medicine, even if they are well, while it is largely given for any eruption on the skin at that time of the year, and often with considerable benefit. That certain forms of skin eruptions occur most frequently in the spring is borne out by the statistics of the Skin Hospital, and this increase appears to be connected with the rise in the average daily temperature which occurs in March or April, according to the season. Presumably, this rise in the temperature increases the activity and rate of growth of bacteria, just as it does that of other vegetable organisms; at this time we find a considerable increase in suppurative lesions, due to staphylococcic and streptococcic infection on the surface of the skin, and also an increase in those forms of disease which are due to, or accompanied by, disturbance of the alimentary canal, a disturbance frequently the result of bacterial activity within or without the body.

I have no intention of entering into the dermatological aspects of this question here, but merely say that outbursts of pustular acne, furunculosis, urticaria, erythematous rashes of a multiform character, acute papular and vesicular eczema, with seborrhœic dermatitis in various forms, make up what are usually called by the patients "spring rashes," and that in many of these cases there is clinical evidence of disturbance of the alimentary canal. For some fifteen years I have treated a large number of these cases by sulphur, and am quite satisfied with the results when compared with other drugs. Looking upon it as a simple purgative, it was at first given in large doses, but it was soon found that in many cases small doses—5 gr. or 10 gr.—were effective, and some other explanation was required than that of mere purgation. This explanation I believe to be the local intestinal antiseptic action of the sulphuretted hydrogen formed in the alimentary canal. For many years now I have used sulphur or one of its compounds in other cases where an intestinal antiseptic seemed to be indicated, and though not always successful, I believe it to be more efficacious than the many drugs more commonly used for the same purpose.

How inefficacious the commonly used intestinal antiseptics are is well shown in the paper of Drs. Friedenwald and Leitz in the *American Journal of the Medical Sciences* for November, 1909,[1] where beta-naphthol, bismuth salicylate, salol, aspirin, ichthalbin, lactobacillin, thiocol, bichloride of mercury, and thymol, when tested by Steele and Strasburger's method, only reduced the fæcal bacteria to a slight extent, from nothing with salol to 8 per cent. with benzo-naphthol, and 12 per cent. lactobacillin; whereas dietetic modifications alone caused a reduction of 16 per cent. I have not had the opportunity of testing the action of sulphur by this laborious method; that it does not sterilize the intestine, however, is certain, as I have obtained growths on gelatine from the fæces after fifteen days' continuous treatment by small doses of sulphur, though the growth seemed less active than a cultivation obtained before the treatment was commenced.

Sulphur has frequently been used with considerable benefit in dysentery, dysenteric diarrhœa, and the summer diarrhœa of children. Richmond, in South Africa, found sulphur in 20-gr. doses an efficient remedy in dysentery. These doses are not strongly purgative, and in some cases the purgative action was diminished by giving it with Dover's powder. It is worth noting that chronic gout and rheumatism—the other conditions for which sulphur was employed by our forefathers—are now believed by some physicians to be connected with the production of abnormal toxic substances in the alimentary canal either from deranged digestive processes or through the action of micro-organisms. The old "Chelsea pensioner" remedy contained one-third of its weight of sulphur, and the trochiscus sulphuris of the Pharmacopœia was introduced primarily for chronic rheumatism.

---

[1] *Amer. Journ. Med. Sci.*, Philad., 1909, n.s. cxxxviii, p. 653.

I do not propose to deal with the general action of the sulphur compounds on the body after absorption. Most of the conditions for which they are employed, such as acne, boils, and carbuncles, are of bacterial origin, and, as sulphuretted hydrogen is certainly excreted by the skin, a local antiseptic action is probably the cause of the benefit derived, at any rate in part. It is often forgotten that sulphuretted hydrogen is extremely fatal to living organisms, and that even a small quantity will prevent development. The unpleasant smell prevents its use in surgery, but the smell of sulphuretted hydrogen is not necessarily to be taken as a sign that putrefaction is still going on; it may, indeed, be an indication that putrefaction is, for the time, arrested.

Though colloidal sulphur is soluble in water under certain conditions, the pharmacopœial forms are quite insoluble in pure water; a statement made in some books that it will dissolve 1 in 70,000 is possibly due to the use of impure precipitated sulphur containing water or calcium sulphate. I have kept a carefully weighed piece of sulphur in two gallons of pure water for several months exposed to the air, and the water changed several times; when carefully dried and weighed there was no perceptible loss of weight in the sulphur. The case, however, is different if decomposing organic matter is present; after one or two days a smell of sulphuretted hydrogen is perceived, further decomposition, for the time, prevented, and after some time a part of the sulphur is lost. Perhaps the common country practice of placing a lump of sulphur in the drinking water for dogs and other animals may be based on some observation of this kind. After much search I found the custom referred to a statement of that fine old natural philosopher, Stephen Hales, who died in 1761. On looking up his works, however, I find the only reference to be a statement in the "Philosophical Experiments" that oil of sulphur prevents water from putrefaction, and he recommends its use on board ship. It is, however, clear from the context that by "oil of sulphur" Hales meant some preparation of sulphuric acid, as he compares its strength with that of the more familiar oil of vitriol.

I have carried out a large number of simple experiments by adding sulphur to various mixtures in test-tubes and bottles and keeping them either at room temperature or in an incubator at 37° C. for several weeks. Some of the tubes were open, others plugged with cotton wool, others tightly corked. The same weight of sulphur and total volume of fluid were used in all the tubes of any one series of experiments. The mixtures were regularly examined and tested for sulphuretted hydrogen by acetate-of-lead test-papers and also by the smell. In some cases, to avoid personal bias, the testing was done for me by my assistant (Mr. J. Grier, M.Sc.), who did not know what the tubes contained, and merely noted what he found in each tube, which was labelled only by a number. I need not weary you by details of these experiments and may summarize them as follows: Dried powdered milk with water, or lightly-cooked minced beef with water, gave no H_2S. Sulphur with water alone gave no H_2S. Powdered milk with sulphur and water gave H_2S in under sixteen hours, strongly marked in twenty-four hours, less marked in forty-eight hours, and gone in seventy-two hours. Beef with sulphur and water gave H_2S in twenty-four hours, more marked in forty-eight hours, strongly marked in seventy-two hours, and then gradually diminishing until no test could be obtained, but a peculiar, unpleasant, cabbage-like smell was present. The beef, however, remained undecomposed for an indefinite time, but the sulphur gradually disappeared and had to be replaced. In one tube I examined the beef microscopically after two years' keeping at the room temperature, and was still able to see the muscular striation; some of the beef still remains after five years; the sulphur has been replaced four times and the water more frequently. After prolonged keeping some of the tubes showed a growth of mould on the surface of the liquid and an unpleasant smell was present even though the beef was still undecomposed; cultivations on gelatine from these tubes gave a growth which liquefied the gelatine. Apparently the sulphuretted hydrogen slowly formed from the sulphur was sufficient to prevent growth of putrefactive organisms but not sufficient to destroy them, and the sulphur was gradually used up in forming the sulphuretted hydrogen or more complicated organic sulphur compounds eventually decomposed into that body. The action of these bodies is an antiseptic and not a disinfectant one.

I have attempted to determine what is the effect of sulphur in the presence of the various secretions of the alimentary canal. No change was found or H_2S produced by mixtures of sulphur with diastase, with liquor pepticus and hydrochloric acid, with liquor pancreatis and sodium bicarbonate, or with bile. Mixtures were then made to contain the digestive ferments, with acid or alkali in proper proportions and coarsely-minced beef or mutton or dried-milk powder; to some of these, sublimed or precipitated sulphur was added. The control tubes without the sulphur in no case gave any sulphuretted hydrogen. In mixtures of liquor pepticus with hydrochloric acid, sulphur and beef or milk powder, digestion proceeded unaltered and no sulphuretted hydrogen was produced. In mixtures of liquor pancreatis with sodium bicarbonate, sulphur and beef or milk powder, digestion proceeded normally, but sulphuretted hydrogen was produced in considerable quantity; on keeping, however, no fæcal smell was developed, as was usually the case in the tubes which contained the same mixtures but without sulphur. Bile, with beef and sulphur, produced sulphuretted hydrogen, and the mixture remained undecomposed for a considerable time.

It seems clear from these various experiments that in the presence of acid no formation of sulphuretted hydrogen occurs, that alkali alone is not sufficient to produce sulphuretted hydrogen from sulphur, but that an alkaline reaction distinctly favours its production when albuminous materials are also present. In the alimentary canal the action of sulphur will therefore only take place at a point where its contents have ceased to have an acid reaction, and the formation of sulphuretted hydrogen, and possibly sulphides, will be hastened by an increase in the alkalinity. Another important point is that the presence of the sulphur did not in any way hinder the action of the digestive ferments, but it prevented pancreatic digestion passing on to the production of substances which cause a fæcal smell. No difference could be found between the action of sublimed and precipitated sulphur; in some cases, however, the latter gave the reaction for sulphuretted hydrogen a little earlier than the former. In some other experiments, however, with oxidizing agents, to which I do not propose to refer further at present, a difference was found between the two forms of sulphur: for example, a weak solution of potassium permanganate was decolourized by precipitated sulphur much earlier and more completely than by sublimed sulphur.

The metallic sulphides, in my experience, are less useful as intestinal antiseptics than sulphur itself; if the stomach is acid they are decomposed rapidly, eructations of sulphuretted hydrogen occur, and probably none of the sulphur reaches the intestine. If the stomach is empty the metallic sulphides are apt to cause considerable irritation and cannot be given in doses likely to be effective. The potassa sulphurata is not suited for internal use at all, but the calx sulphurata can be given, and in considerably larger doses than those commonly employed, especially if it be exhibited in a rather firm gelatine capsule or coated pill. I frequently find it beneficial in suppurative conditions in doses of 1 gr. three times a day, but two precautions are necessary—viz., it must be freshly prepared, and it must be given so as to avoid decomposition in the stomach. I doubt whether it has much intestinal action, and it rarely purges even when 2 gr. are given three times a day; it is more suitable for use where rapid absorption of the sulphide is desired. I find many practitioners have discarded the calcium sulphide as useless, and, on inquiry, it usually turns out that they take no care to insure the activity of the preparation they employ, and that they limit themselves to the small doses recommended by Ringer, but neglect his recommendation to give it every hour.

Some sulphides, however, including sulphuretted hydrogen, may be used as intestinal antiseptics in the form of natural mineral waters, and it is worth consideration whether this action does not offer a more reasonable explanation than purgation alone of the undoubted benefit frequently derived from the internal use of the sulphurous waters of Harrogate, Llandrindod, Aix, and many other spas. The large amount of water in which the sulphides are dissolved minimizes the local irritant action, and taken, as they usually are, on an empty stomach, they are not decomposed but reach the intestine in an active state. The benefit derived from these waters is often out of proportion to the degree of purgation, and it is interesting to note that the class of patients most successfully treated shows a considerable preponderance of cases in which the disease may be ascribed to auto-intoxication, the result of gastro-intestinal disturbance. The good results now obtained at Harrogate from lavage of the large intestine with sulphur water in cases of colitis may be similarly due to local antisepsis.

Sulphurous acid, the sulphite and thiosulphate of sodium, are moderately powerful antiseptics and are of some importance now that they are used for preserving foods, wines, and other liquors; in medicine they are given internally for gastric fermentation. They appear to be rapidly oxidized to sulphates either before or after absorption, and, as they are very soluble salts given in comparatively small doses, absorption probably takes place in the upper part of the small intestines; it is doubtful whether they reach far enough to be reduced to sulphides or to have any intestinal antiseptic action. My experience of their use is limited, but so far I have not found them to lead to the production of sulphuretted hydrogen.

The sulphates require a somewhat fuller consideration. Magnesium and sodium sulphates are more extensively used than any other saline purgatives, such as sodium phosphate or citro-tartrate, in spite of the fact that their taste is more unpleasant. This wider use is probably due to the greater benefit derived from them, and I believe that this greater benefit is due to the reduction of a small part of the sulphate to sulphuretted hydrogen, and that in this way the sulphates have an intestinal antiseptic action as well as a purgative one. As purgatives they are given in large doses with the object of increasing the volume of the intestinal contents by preventing absorption of the fluid. A considerable amount of the salt is carried along the whole length of the intestines and passes out with the fluid stool, so that if purgation is not too rapid there is every opportunity for reduction to take place.

I have no intention of discussing the mode of action of the saline purgatives. Dr. Hertz brought the subject before this Section in December, 1908,[1] and gave evidence that when magnesium sulphate was administered some absorption took place. That this is certainly correct is shown by a series of urinary analyses conducted for another purpose by Dr. Elsie Royle under my direction at the Christie Hospital. The amount of magnesium in the urine was found to vary in a surprising and irregular manner, and it was only by recollecting that compound senna mixture was used as house medicine, and ascertaining from the sister the dates at which doses had been given, that these variations were explained. Those patients who took the compound senna mixture regularly twice a week had a high percentage of magnesium in the urine, and those who took an occasional dose showed a sudden rise on the following days; in most cases the inorganic sulphates in the urine were also increased, but not the organic sulphates. The total amount of magnesium in the urine, however, was never sufficient to account for more than a small fraction of the amount taken—e.g., in one case after a single dose the magnesium in the total twenty-four hours' urine increased from 0·01 grm. to 0·11 grm.; in another case while on regular doses 0·114 grm. was excreted daily, when the mixture was stopped this fell to 0·07 grm. Each dose of the mixture exhibited contained at least 8 grm., and, allowing the excretion of the absorbed salt to be spread over three days, the total excreted by the kidney would not exceed 0·34 grm. In a similar way the increase in the inorganic sulphates only accounted for a small proportion of the anion, so that by far the greater proportion of both ions of the magnesium sulphate passed out by the bowel.

---

[1] *Proceedings*, 1909, iii, p. 21.

I have found out by inquiry from patients and by repeated personal experiment that sulphuretted hydrogen is passed by the bowel much more frequently after magnesium or sodium sulphate has been taken than after sodium phosphate or the effervescing citro-tartrate. If the stool occurs quickly, say, within an hour or an hour and a half, little or no sulphuretted hydrogen is passed with it, but it is often passed as flatus during the day or with the following stool; if the first stool is delayed, sulphuretted hydrogen is almost invariably passed with it. My attention was particularly called to this subject by a medical friend who suffers from gall-stones, and for whom sodium sulphate had been prescribed to be taken several times a day. He wished me to explain why he could take a dose before breakfast without any discomfort, but that doses taken later in the day caused after a time most unpleasant eructations of sulphuretted hydrogen from the mouth. I could only suggest that in the morning, after a twelve to fifteen hours' fast, the sodium sulphate passed too far down the bowel before meeting with the conditions for reduction to take place, but that after a meal, when the upper part of the bowel contained food, reduction of the sulphate occurred sufficiently high up for the gas to pass upward through the stomach.

The value of magnesium sulphate in dysentery appears to be well attested, and it seems at least probable that an antiseptic action in the intestines may play an important part. Sodium sulphate has been used for dysentery, particularly by French physicians, for over fifty years. In such conditions as acute urticaria the sulphates also appear to be more efficacious than other salines, and this condition is usually dependent upon some digestive disturbance with intestinal auto-intoxication. The various sulphated natural mineral waters have also to be considered from this standpoint, as they contain sodium or magnesium sulphates. The enormous trade in those imported into this country testifies to their wide popularity. It may be that the sulphates in them are in a condition which permits of a more rapid reduction than is the case in a simple solution of the salts in water; most people, at any rate, consider them to be more effective than their equivalent saline contents dissolved up in an equal quantity of water.

The non-purgative mineral waters which contain calcium sulphate are also worthy of note, since it is well known that suphuretted hydrogen is produced very readily from calcium sulphate in the presence of decomposing organic matter. At times new wine is found to contain sulphuretted hydrogen as the result of the reduction of calcium sulphate during the process of fermentation. Possibly the adulterated form of precipitated sulphur made with sulphuric acid in place of hydrochloric acid, and which contains two-thirds of its weight of calcium sulphate, may not be so bad a preparation after all. It was preferred by some of the old physicians to the pure sulphur precipitatum, and is still used by the public. I have been told many times that this form of "milk of sulphur" acts differently to the ordinary forms. Whether sulphuric acid itself can be reduced in the alimentary canal does not appear to be known. It is worth some investigation if only from the fact that no explanation has yet been found for the universal preference for sulphuric acid to other acids in the treatment of diarrhœa.

Passing now to the organic compounds of sulphur, a brief reference only is necessary to ichthyol and its congeners. With its large sulphur content ichthyol is already recognized as an intestinal antiseptic, and is used for that purpose in chronic urticaria and other conditions. The organic compounds to which I wish to refer are the vegetable substances so largely used dietetically, and to some extent medicinally, which contain sulphurous volatile oils. The dietetic value of fresh vegetables has always been recognized, and an examination of the various kinds cultivated for food shows a surprising preponderance of those which contain organic compounds of sulphur. It seems hardly likely that this is merely an accident. Our dietetic habits have been formed by a process of natural selection, in which the experience of ages has shown the greater dietetic value of these sulphur-containing vegetables, and has thus led to their extensive cultivation and improvement. It is a familiar experience that the consumption of these vegetables is followed by formation of sulphuretted hydrogen in the intestine or even in the stomach, a fact popularly expressed by the designation of "windy foods" so often applied to them. Of these vegetable foods, onions, leeks, and garlic are perhaps the best recognized as containing sulphur; indeed, Sir William Whitla recommends the use of onions as a convenient and effective means of giving sulphur internally. To these we may add asafœtida, which we are told is used as a food in Persia and other parts of Asia; the medicinal use of asafœtida is largely due to its volatile oil, and it may be that the compound asafœtida pill given by some surgeons to remove tympanites after abdominal operations has a real value in diminishing bacterial activity in the intestines. Onions, garlic, and asafœtida appear to be most used for cookery in warmer countries, and among the class of people who, owing to climatic or social conditions, have not always the opportunity of obtaining food in a fresh condition, and it is likely that much of their value as foods may be due to the intestinal antiseptic action of the sulphur compounds they contain, or which are formed by their decomposition.

The natural order Crucifera contains most of the plants which yield sulphurous volatile oils, and to this order most of our ordinary table vegetables and salads belong. The sulphur is usually present as a thiocyanate. The sulphurous volatile oil from black mustard, which is in the Pharmacopœia, is itself a powerful antiseptic, and, much diluted, has been successfully used by Roswell Park for sterilizing the hands of the surgeon. When taken internally mustard gives rise to sulphuretted hydrogen, so that all the vegetables which contain the mustard oil may be regarded as antiseptic substances, both in the stomach and intestines. White mustard has a somewhat similar oil to black mustard, but less pungent. Other cruciferous plants which contain sulphur are *Cochlearia armoracia* (the horse-radish), *Raphanus sativus* (the radish), *Brassica rapa* (the turnip), *Nasturtium officinale* (the watercress), which mostly contain mustard oils. *Lepidum sativum* (the garden cress), with several other varieties, contains the benzyl isothiocyanate; *Cochlearia officinalis* (the scurvy-grass), like many of these vegetables, had great reputation as an antiscorbutic, and its sulphur content becomes interesting when compared to the old reputation of Harrogate water as a cure for land scurvy. Then we have *Brassica oleracea* (the cabbage), with its varieties, *acephela* (the Scotch kail), *bullata* (the Savoy cabbage), *capitala* (the red and white cabbage), *botrytis* (the cauliflower), *gemmifera* (the Brussels sprout), all in common use, and which contain bodies closely allied to the mustard oils.

Many other plants could be mentioned which contain sulphur and have had their place in folk medicine, but I will only refer briefly to the *Humulus lupulus* (the common hop). May it not be that there is some truth in the contention that the old-fashioned beer, made from malt and a plentiful supply of hops, was more wholesome than that made from glucose and hop-substitutes, and that this greater wholesomeness was in part, at least, due to the small quantity of sulphur in the hops, and which is not present in the various bitters used as hop substitutes?

I cannot enter, to-day, into a consideration of the action of sulphur upon other organs. It is certainly a diaphoretic when given in regular doses; it also increases the sebaceous secretion, and, I believe, makes it more of an oily character; as an expectorant it is apparently useful in some forms of bronchitis. It has also, when long continued, a distinctly depressing effect on both the circulatory and nervous systems, and a word of warning is necessary as to the danger of using it for too long a time. I have seen marked symptoms of depression produced by sulphur itself, by the calx sulphurata, and by Harrogate waters. In one case where a patient continued the use of sulphur for months on account of acne vulgaris, which was quiescent while she took it, the nervous depression reached actual melancholia, which was completely recovered from on stopping the sulphur. Of course the chief disadvantage is the offensive character of the stools and occasionally of the breath. This is most marked when full purgative doses are taken frequently; there is, as a rule, no serious complaint when only 5 gr. or 10 gr. are given two or three times a day, which is sufficient when an antiseptic effect only is required.

As to the method of administration, the Pharmacopœia contains ample and excellent preparations. For large doses the confection serves all purposes; for smaller doses the lozenge is not unpleasant—if the sweetness is objected to by adults, compressed tablets are easily made to contain 5 gr.; and for children it can be mixed with chocolate or given as the compound liquorice powder.

To briefly summarize the advantages of sulphur as an intestinal antiseptic:—

(1) It is almost tasteless and easily administered.

(2) It is insoluble in the stomach, and the greater part of it passes along the whole length of the alimentary canal.

(3) It does not interfere with the action of any of the digestive secretions.

(4) It forms active antiseptic substances in the intestines when their contents become neutral or alkaline. Some of these substances are gaseous and penetrate to all parts of the intestine.

(5) It is sufficiently non-poisonous to be given in effective doses.

(6) It has valuable laxative properties which promote an early evacuation of the intestinal contents.

(7) It is cheap.

The Therapeutic Use of the Digitalis Group.

By H. Hume Turnbull, M.D.

Although digitalis is, of course, well known to be of the utmost value in some cases of cardiac failure, it has been found disappointing in other cases, and thus the drug has been at once condemned as unreliable in its strength and in its action on the heart. As Mackenzie has pointed out, however, this uncertainty does not depend on the drug itself, but on the differences in the cases treated.

During the past twelve months a series of observations have been carried out at the Mount Vernon Hospital to determine which cases show the best response to treatment by this group of drugs, and also what is the most satisfactory method of administration. It has been found possible to obtain consistently good effects in certain types of cases, while in others little or no action on the heart could be demonstrated; and there can be little doubt that careful work and exact

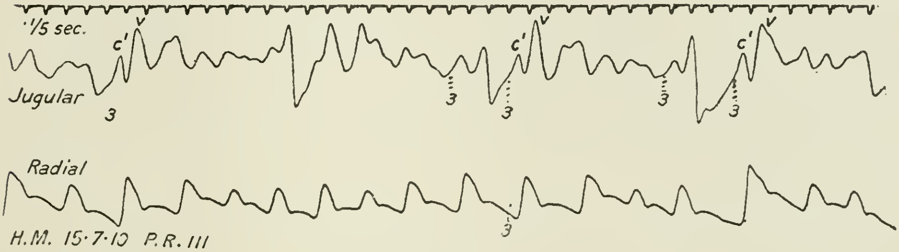


Fig. 1.

Shows the irregular, rapid pulse of auricular fibrillation. The patient was very distressed and orthopnœic, and marked œdema of the lungs and extremities was present.

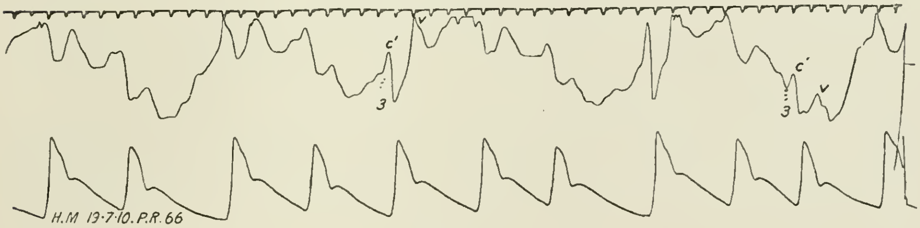


Fig. 2.

Tracings from the same patient as gave fig. 1 after 5½ dr. of tincture of digitalis had been taken in three days. The pulse is seen to be much slower and more regular, the rate being only 66 per minute.

observation will allow of a definite and satisfactory prognosis being given in any given case.

I do not wish to go fully into the results produced in the different types of case, but would merely state that our results confirm the old observation that cases with a rheumatic history react best to these drugs, and that their special use is to be found in cases of rheumatic heart disease, in which the auricle is fibrillating. In these cases the action is prompt and very striking, the rapid, wildly irregular pulse is quickly steadied and becomes slower and more regular, though in only a minority of cases is complete regularity produced, and that condition can only be maintained by keeping the patient under the action of the drug.

If the rheumatic heart fails, while pursuing its normal rhythm, with efficient contraction of the auricle, and the picture of dyspnœa, dropsy, &c., is produced, the reserve power may frequently be restored by rest, without drugs; but if the failure is due to the onset of auricular fibrillation, or occurs while the auricle is in a state of fibrillation, the use of digitalis or its allies is imperatively necessary, and unless these drugs are resolutely pushed until the pulse slows, the patient will gradually drift to his death.

Rest.—All our cases were given a preliminary course of rest, so that the drug should not get the credit for results due to other causes. The only exceptions were cases where the symptoms were so urgent as to call for immediate relief.

DOSAGE OF DIGITALIS.

In many cases in which good effects have been expected from the use of digitalis such have not followed, and this has caused much distrust as to the reliability of the drug; but the true explanation will often be found in the fact that the dose has been insufficient for the purpose. In ordinary cases, where the symptoms have been pronounced, though not extremely urgent, it has been our practice to give ♏xv of the tincture four times a day, and to continue the administration until the "physiological" effects are produced. If the condition of the patient is urgent, ♏xv are given every four hours, or ½ dr. three times a day, which is the largest dose we have yet found necessary, though there is every reason to believe that in great urgency larger doses could be quite safely given, and that a more rapid effect would be produced.

In considering the quantity of digitalis which must be given to produce "physiological" effects, two factors are important:—

(1) The amount given per day.

(2) The time during which the administration is continued.

These are usually in inverse ratio to one another—i.e., large doses produce the full effects rapidly, while smaller ones produce the same effect, but require a longer period to do so. However, it must be remembered that, if the dose is too small, no effect will be produced, though the case is one quite suitable for the use of the drug, and would respond to full doses; therefore it seems much more satisfactory to give full doses at first, till the patient is completely under the influence of the drug, and then diminish the dose till an amount is found which will keep up the best condition.

In all cases careful records have been kept of the amount necessary to produce the first "physiological" symptoms, and then of the total amount of the drug taken. In a series of twenty-seven administrations, an average of 4½ dr. of the tincture of digitalis was necessary to produce the first physiological effect, the minimum being 2 dr. and the maximum 12 dr. In these cases the average amount given before full effects were produced was 5¾ dr., the smallest amount being 3¾ dr. and the largest 12 dr. In this last case the patient felt very well till he had taken 12 dr., then he began to complain of headache and nausea, and the drug was stopped.

The first symptoms complained of were as follows: Twelve cases complained of headache, eleven cases complained of nausea, eight cases suffered from vomiting, four cases complained of giddiness, three cases complained of loss of appetite, two cases complained of feeling the heart thumping unpleasantly. In two cases multiple ventricular extra-systoles were noted in the pulse tracing before any complaint was made by the patient, and these gave rise to no inconvenience, though the drug was continued. This latter condition—viz., extra-systoles—seems to account for some of the cases which have been recorded of so-called digitalis poisoning, the irregularity being regarded of serious import, though in both cases under notice the patient was extremely well during this period.

If the drug be continued after "physiological" symptoms are observed, the final results produced are similar to the first effects, but of increased severity.

Strophanthus.—In the case of strophanthus the question of dosage is more uncertain. The amount necessary to produce symptoms varied between wide limits, the smallest amount being 3 dr. and the largest 29½ dr., and while in one case the full effect was obtained with 3¾ dr., in another it required 40½ dr., administered over a period of twenty-nine days. The commonest symptom produced by this drug was diarrhœa, occurring in more than half the cases, and usually accompanied by headache, while nausea or vomiting was produced in half the cases. In one case no subjective symptoms were complained of, though frequent extrasystoles were produced. This sample of strophanthus, tested pharmacologically by Professor Cushny, was proved to be twenty times as strong as the preparation of digitalis which we used.

Squill.—Tincture of squill in ♏xv doses four times a day produced no symptoms in most cases even in amounts up to 21 dr. in twenty-one days; but in one case of mitral stenosis with normal rhythm heart-block, after exercise, came on after 13 dr., and, after 20 dr. had been taken, headache was complained of and the heart-block was more marked, so the drug was stopped. However, this dose of squill proved sufficient in several cases to maintain the slowing and good effects produced by digitalis. The squill used was one and a half times as strong pharmacologically as the digitalis.

In cases with normal rhythm the slow pulse produced by digitalis is in most cases somewhat irregular, the whole heart taking part in the irregularity (*see* fig. 3). The sinus rhythm is alternately slowed and quickened, apparently by vagus action, and this phasic irregularity is often so marked as to be easily recognized by palpation or auscultation, and, unless care be taken, may be mistaken for heart-block and considered as a contra-indication to digitalis. A jugular tracing, however, immediately clears up any doubt as to the nature of the irregularity (*see* figs. 4 and 5).

Although a failure of reserve force, so marked as to give rise to marked dyspnœa and dropsy, is usually taken as the indication for digitalis, very satisfactory results may often be got by a full course of the drug in cases where the symptoms are less urgent, consisting only of dyspnœa on exertion or inability to lie down comfortably at night.

Cases of failure of reserve force, due to cardiac degeneration from other causes, so-called cardio-sclerosis, showed very little reaction to digitalis as compared with the rheumatic cases. In several no slowing whatever was produced, although the drug was pushed to the production of vomiting, though occasionally the patients seemed somewhat improved as a result, dyspnœa and cardiac pain being lessened and the reserve force somewhat increased. Auricular fibrillation in such cases often gives rise to very few symptoms of any kind, and digitalis appears to have little action in slowing the heart.

Several cases have been treated as out-patients on doses of ♏xv of tincture of digitalis three times a day, with most satisfactory results, and no danger is to be apprehended from its use in this way. The physiological subjective symptoms are marked and unpleasant, and the patient always stops the drug himself—if it is not stopped by the physician—before any danger point is reached. Difficulties in the use of the digitalis arise in practically only two classes of case, and from widely different causes:—

(1) In cases in which symptoms similar to those produced by the drug—i.e., vomiting or persistent headache—are already present.

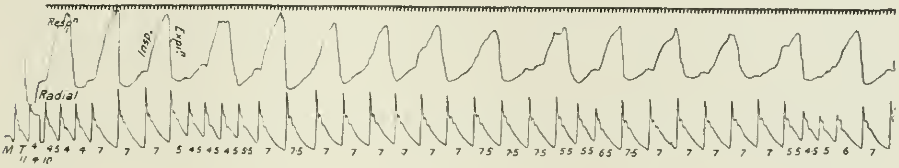


Fig. 3.

Shows the type of slowing usually produced by digitalis. The pulse is slow and very irregular, and the nature of the slowing is shown in the next figure. The tracing shown is from a case of mitral stenosis after 13 dr. of tincture of squill, the effects of which are similar to those of digitalis.

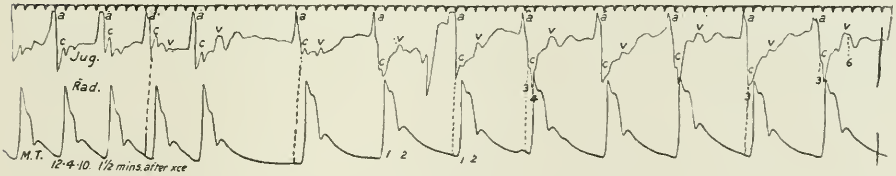


Fig. 4.

Simultaneous tracings of the jugular and radial pulses, showing that this slowing is due to a standstill of the whole heart, the sinus rhythm being slowed, but all auricular impulses pass to the ventricle.

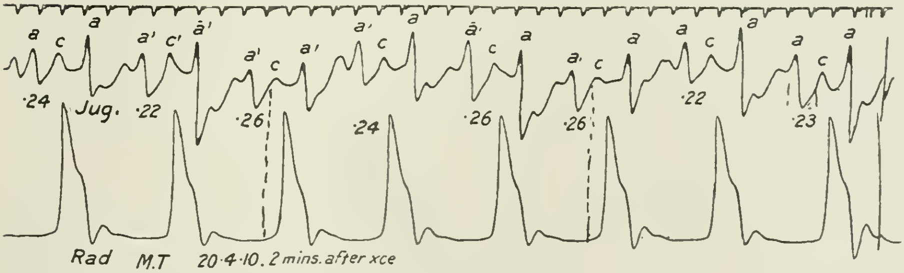


Fig. 5.

Shows the condition in the same patient after 20 dr. of tincture of squill. The radial tracing is very similar in appearance to that shown in Fig. 4, but in the jugular it will be seen that the auricular rate is faster and only alternate impulses reach the ventricle.

When a patient with acute failure is already suffering from frequent vomiting, it seems advisable that a drug such as digitalis, which is nauseous to taste and which usually sets up vomiting as an early symptom, should be used. However, in one case we have been able, by carefully pushing it, to give sufficient for definite heart effects to be manifested, without apparently increasing the vomiting at all. Still, it seems probable that in these cases the intravenous injection of some such preparation as strophanthin would give better results. In another case, that of a man who has for some years suffered from persistent headache, this seems to be increased by the use of all this group of drugs, and we have been unable to find a combination which will keep his heart action satisfactory and yet not cause severe headache. His condition is one of auricular fibrillation with mitral stenosis of rheumatic origin, and if digitalis or its allies be withheld, the heart rapidly reaches a rate of 130-160 beats per minute, and his condition becomes very grave. With about a drachm of tincture of strophanthus a day he can get about in perfect comfort, except for severe intractable headache.

(2) The other condition requiring care in the use of this group is one of damage to the a-v bundle, shown by increases of the a-c (P-R) interval or by partial heart-block. In these cases heart-block may be produced or aggravated by digitalis, so that care is necessary in its administration; but if a careful watch is kept on the patient, the drug may be used even in such cases with much benefit.

No rise of blood pressure has occurred in any of our cases (with one exception to be mentioned below) as a result of the use of digitalis; in fact, in several cases the systolic pressure has fallen definitely under its use, and there seems to be no real justification for the statement that medicinal doses cause a rise of blood-pressure in man.

In two cases in which fatal heart failure set in while the patient was resting in bed (no cardiac drug of any kind having been used), the blood pressure rose 20 mm. of mercury above its normal level, and remained raised till death. In one the normal systolic pressure for four weeks was 120-5, and one hour before death it had risen to 140.

As in several cases the blood pressure has fallen when the symptoms were relieved by digitalis, it seems probable that the rather high readings so frequently obtained in cases of heart failure are the result of some embarrassment, and not an index of a pre-existing arterial contraction; and if this is the case the high blood pressure is rather a call for than a contra-indication to digitalis. It is difficult to obtain clear data on this point, as so many cases of severe heart failure show auricular fibrillation, or the widely irregular pulse prevents accurate records of blood-pressure being taken.

The one case mentioned above in which blood-pressure rose was that of a man who was admitted to the hospital suffering from general dropsy, orthopnœa, and very marked cyanosis. He had an enormous heart with systolic and diastolic aortic murmurs, and a systolic murmur at the mitral orifice, and the veins of the neck were full and pulsating. The pulse was regular, but the jugular tracing showed only the merest trace of an auricular wave, suggesting that the auricle was so distended as to be practically paralysed. He had been vomiting a great deal for some days. Digitalis was carefully pushed, and after five days the vomiting, which had ceased, recurred, and the drug was stopped when he had taken 5½ dr. He then was able to lie down, the dropsy was almost gone, urine had increased from 20 oz. a day to 130 oz., and the pulsation in the veins of the neck had disappeared. He was still very cyanosed, however, and the blood pressure, which had been 174 mm., was now 198 mm. The pulse had not slowed at all. Early the next morning he died quite suddenly. Here it is at least arguable that the rise in blood pressure was due to some pathological stimulus affecting the vasomotor mechanism, as the heart showed no slowing or other digitalis effects (digitalis was commenced as soon as he entered the hospital, so the possible effects as yet cannot be excluded), and the rise is similar to those shown by progressively failing hearts. Also, no data are available as to what the patient's normal pressure was.

Another use for digitalis, which is as yet only a suggestion, based on the empirical fact that the drugs of this group are the only ones known to have any demonstrable effect on the heart clinically, is in cases of persistent tachycardia due to stimuli from some abnormal point on the ventricular wall. In one such case tachycardia had persisted for four and a half months, the pulse-rate being 150 as against the previous normal of about 55 to 75. The patient was a man, aged about 74, previously quite healthy, who presented no definite abnormal cardiac signs. The sounds were clear but rather weak, the vessel walls thickened, and the chest emphysematous. Tincture of digitalis was given in ♏xx doses three times a day, and, after he had taken 7 dr., the condition of the pulse changed markedly, being slowed to 88 beats per minute and continuously irregular. The interpretation of the jugular-pulse tracing is not simple, but the condition appears to have been one of auricular fibrillation. The same night he felt very ill and the following day complained of nausea, so the digitalis was stopped after he had had 8 dr. The pulse had meanwhile reverted to the rapid type, even though he was still taking digitalis, but two days later it was regular, 75 beats per minute, and the jugular tracing showed a definite "a" wave. The rhythm, however, was interrupted by fast paroxysms, and though digitalis, in doses of ½ dr. per day, was recommenced, the normal rhythm was soon entirely replaced by the tachycardia. This rapid rate continued for some weeks without change, the condition being precisely similar to that which obtained before treatment was begun, and then digitalis was recommenced in the form of Nativelle's granules, one three times a day. After he had taken thirty-three granules he complained of nausea and loss of appetite, and stopped the drug. The next day the pulse was found to be 75, of normal type, and regular except for "nodal" extra systoles, single and in short paroxysms. Two days later the auricles were found to be fibrillating and the patient felt very ill; but then the auricles took up their normal beat again, and the pulse became normal and regular in type. This regularity continued, save for an occasional extra systole, and the

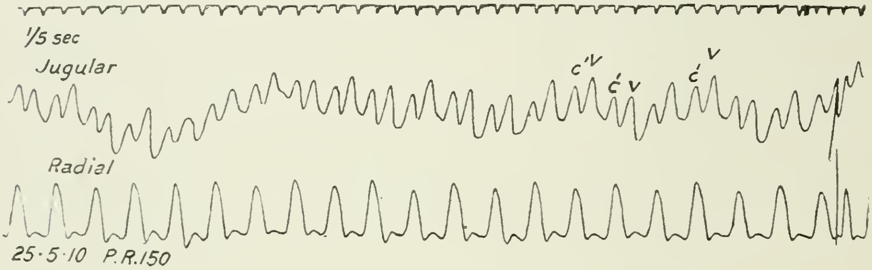


FIG. 6.

Simultaneous tracings of jugular and radial pulses during the tachycardia. The jugular pulse is of the ventricular form, and the rate is 150 per minute.

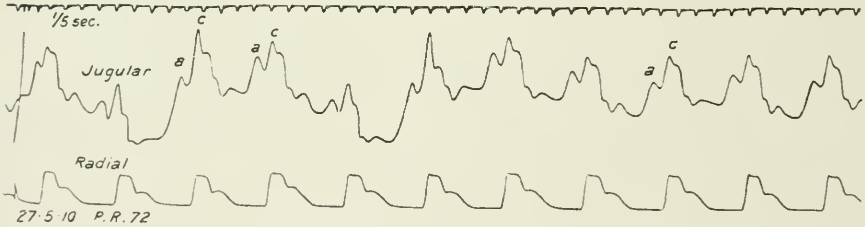


FIG. 7.

From the same patient as gave fig. 6, after he had taken 8 dr. of tincture of digitalis. The pulse-rate is now 72, and the jugular pulse is of the normal type.

patient felt in every way much better and stronger, and was able to go to the country for a change. He has remained well ever since (three and a half months), the pulse-rate is 75 per minute, the beat a normal type, regular except for an occasional extra systole. He is able to go about in perfect comfort, and walks about three miles, half of it up hill, every afternoon, without the least distress. This patient showed a symptom which, though not common, is occasionally met with when digitalis is pushed—viz., a state of pronounced mental confusion with delusions and loss of memory, which lasted about four days and then entirely disappeared. The confused state appeared just after the first signs of nausea.

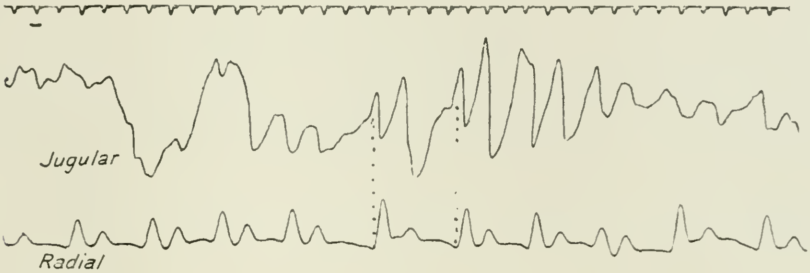


FIG. 8.

Simultaneous tracings of the radial and jugular pulses in a case of mitral stenosis with auricular fibrillation during a period of severe heart failure. Note the irregularity of the radial pulse, typical of auricular fibrillation. Rate 105.

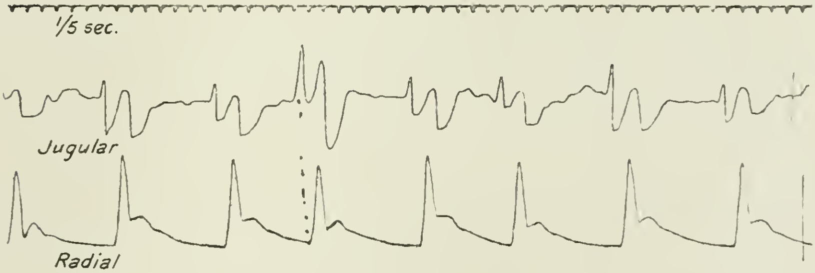


FIG. 9.

From the same patient as gave fig. 8, when 6 dr. of tincture of digitalis had been taken. The patient's condition had greatly improved. Rate of pulse, 50.

To sum up: In our experience, if it is decided that digitalis should be tried in a given case, the most satisfactory way to give it is in full doses, pushed till definite physiological effects are produced. Then the drug is stopped entirely for a day or two, till the physiological effects, except the heart-slowing, have passed off, and then it is resumed in smaller doses until an amount is found which keeps the patient in the best condition. To arrive at this dose it is better that the patient should be up and about, so that he can carefully test his own powers of response to effort, and if he is an intelligent man he may usually be left to find out for himself how much of the drug he must take to keep well. In one case of auricular fibrillation, who had been in hospitals at short intervals for some years and was quite unable to work, an almost regular pulse of 40 was produced by digitalis, and the patient is now doing regular work in perfect comfort on a dose of about 1 dr. of tincture of digitalis per week. On his discharge from hospital he took ½ dr. per day for a month, then reduced the dose to ♏xv per day. He found this amount not always necessary, so he stopped it, and takes now about 1 dr. per week, usually in one dose, the signal for a dose being a feeling of undue rapidity of heart action, or any feeling of breathlessness on exertion. He works about twelve hours a day, pulling a heavy barrel-organ along country roads, and shows no symptom whatever of heart weakness.

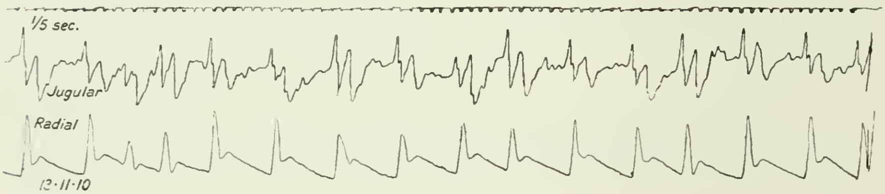


FIG. 10.

From the same patient as gave figs. 6 and 7. Shows the condition four months after discharge from hospital. He had been taking about 1 dr. of tincture of digitalis per week, and had taken ½ dr. the day before this tracing was obtained. Rate of pulse, 54.

DISCUSSION.

Dr. HARRINGTON SAINSBURY desired to mention one point about the use of digitalis—namely, the difficulty clinicians found, in an acute case, in waiting for the effect. The older experience with the then customary doses was that three or four days were required in order to develop the action of the drug. Recently there had been produced a digitalis derivative called digalen. It was an excellent preparation, which acted more rapidly when given orally, and still more so when used hypodermically; the latter use would bring out the effect within twenty-four hours. Another recommendation of digalen was that it did not cause so much disturbance of the alimentary tract. Passing from this, Dr. Sainsbury drew attention to a point raised in the paper—viz., the question of heart failure and rise of blood-pressure. Blood-pressure could only be maintained by active contraction of the heart; accordingly if the heart maintained its driving power, as shown by the raised blood-pressure, and yet the circulation failed, it would become necessary to coin a new word instead of "heart failure." He could not understand the co-existence of a rising blood-pressure with a failing ventricle.

Dr. E. I. SPRIGGS said that the Section would be grateful to Dr. Turnbull for his paper. It was difficult to separate the effect of drugs on the heart from the effect of rest, and in some cases a week of rest before the drugs were given might not be long enough. In some observations at the Victoria Hospital on the effects of digitalis, strophanthus, and squill in heart disease of children, he had often found that a week's rest was not enough, for after a week had passed the heart would begin to improve without any drug. Therefore he kept the patient in bed until the condition of the heart, in regard to regularity and rest, was stationary for several days, and then the drug was given. As a result, failure of compensation at the end of that time was comparatively rare. The recuperative power of the child's heart was so great that he had had but few cases which resisted rest and the use of one of those drugs. In such squill was given for a week, then discontinued until the pulse remained at a constant rate for at least five days. If the compensation was not recovered strophanthus was given for a week, and later on digitalis for a week. In others the order of the drugs was reversed. The conclusion arrived at so far was that digitalis was more effective in slowing the heart and arresting its irregularity than strophanthus or squill. The drugs were those prepared by the Society of Apothecaries and physiologically standardized. With regard to giving digitalis to out-patients, if the patient was intelligent one could leave it to him, and nothing was more striking than the tenacity with which they would sometimes carry out the instructions of their doctor, notwithstanding possible discomforts. It was not always safe to leave it to women, and certainly not in the case of children. Vomiting could occasionally be prevented by giving 20 gr. of bismuth oxychloride, shaken up in water, at the same time as the medicine. Vomiting might also follow the injection of crystallized digitalin under the skin. In one case, a girl aged 8, weighing 43 lb., the injection of $\frac{1}{240}$ gr. (or $\frac{1}{4}$ mg.) twice a day was followed on three occasions by vomiting, which came on in two, seven, and nine days respectively, ceasing each time on the omission of the drug.

Dr. J. Gray Duncanson said he first wished to speak of dosage. He did not refer to that gentleman who took his drachm of tincture of digitalis once a week—he would probably be just as well without it—but to those severe cases in which digitalis was of proved value. He agreed that 1 dr. in twenty-four hours was a fair dose, but he recommended its administration at shorter intervals, say, every two or three hours; so long as the tincture of digitalis was obtained from a firm of repute, which should always be a rule, he did not mind whether it was physiologically standardized or not. Dr. Martin, of Newcastle, had written an admirable paper drawing attention to the danger of placing too much reliance on physiological standardization where frogs were used, as he found they differed in their susceptibility to the drug at various seasons of the year. The second point was as to whether digitalis raised the blood-pressure. He thought it did. Dr. Turnbull said it was difficult in hospital to know what a patient's blood-pressure was; but that was not so in private practice, where the medical man had a long personal acquaintance with his patients. A case came under his observation some years ago. A man, whom he knew personally, though he was not his patient, had been under the treatment of a heart specialist in London unknown to his own doctor. According to the prescription ordered he was taking 1 dr. of tincture of digitalis in twenty-four hours, and this had gone on for months. Dr. Duncanson took his blood-pressure, which was over 200 mm., advised him to give up the digitalis, go to bed, and send for his doctor. He was dead in a fortnight.

Professor Wild wished to refer to the question of dosage. The dose of the tincture of digitalis used to be ♏xv to ♏xxx. When the last Pharmacopœia was issued the dose of poisonous tinctures was reduced to ♏xv, and many people thought that was too low, when no provision was made for increasing the strength. When one worked out experimentally the relative strengths of the preparations of digitalis, it was found that ♏xv to ♏xxx corresponded better to the ordinary dose of the infusion. It was a point to consider whether, in the next edition of the Pharmacopœia, the original dosage should not be restored.

Dr. Cameron said vomiting had been referred to in a somewhat airy way by Dr. Turnbull, who called it a physiological effect. He (Dr. Cameron) had been accustomed to see ill-effects from vomiting after the use of digitalis. Many patients began to vomit when they were put upon much smaller doses of digitalis than those which had been referred to—patients who had not previously vomited. Once it was set up in a severe case of cardiac failure it was difficult to check. The physical effort of vomiting caused a severe strain on the heart. He had watched patients with severe heart failure, in whom the end immediately followed vomiting, and others who died after passing, or being about to pass, a constipation motion. Another point was that

Dr. Turnbull omitted to give the ages of the patients he had spoken about. He agreed with Dr. Spriggs that it was rare to see cardiac failure prolonged in a child. Children came to Guy's Hospital with recurrence of heart trouble after rheumatism; but they were usually admitted with a little pyrexia and with other signs of slight rheumatism, and their cardiac trouble was short-lived. Moreover, it was difficult to compare cases with good muscle—uncomplicated severe cases—with those in which there was such muscular degeneration as was demonstrable upon death.

The PRESIDENT (Professor Cushny) said he also objected to vomiting being spoken of as a physiological effect of digitalis. The father of digitalis always began his treatment by producing vomiting, and, judging by his records, probably few people had been more successful than he was. In the one or two cases that died, that authority did not regard it as failure of the digitalis so much as failure on the part of the patient to vomit adequately. With regard to standardization, one or two of the speakers seemed to consider that it was a set-back for physiological standardization that the drugs acted in a different fashion on man. But that had been recognized for years. Everyone knew that the frog responded twenty times as strongly to a good tincture of strophanthus as to a good tincture of digitalis, and there was no question that this relation expressed the actual strengths of the two tinctures. When given by the mouth in man, however, a large proportion of the strophanthin was not absorbed in an active form, so that at first sight there seemed to be a discrepancy in the results. As a matter of fact, tincture of strophanthus was, of course, standardized against strophanthus, and tincture of digitalis against digitalis, so that was no real difficulty in regard to them.

Dr. TURNBULL, in reply, apologized for speaking of vomiting as physiological. It was no doubt not quite accurate, but the difficulty was to find a better word to express the results obtained from full doses. Tincture of digitalis was used because it was the preparation most commonly used for heart trouble by the general practitioner. With regard to the rise of blood-pressure at the end of heart-failure, he did not think it was very extraordinary that it did rise, because until the man was dead there was enough cardiac power to keep the blood circulating, and there was no reason why it should not be kept circulating at a pressure of 140. In the case he mentioned he took the blood-pressure an hour before the patient died, and it was 140, his normal being 120. He could offer no explanation, and merely recorded the fact. With regard to Dr. Spriggs's criticism as to rest, his paper had been much too short to enable him to bring out all that he desired to say. Using Dr. Mackenzie's division of the work of which the heart was capable—as, first, the force needed to keep the heart going with the patient lying in bed, the "rest force"; and, secondly, the "work force," viz., that needed to enable the patient to walk about and do his ordinary work—very few of the cases had lost that "rest force." They had not the ordinary symptoms of failure of compensation—marked dyspnœa and dropsy. In some of those who had dropsy it cleared up in a week. But one did not simply aim at keeping the patient comfortably in his bed; he was allowed to go upstairs, if he could, and lead the ordinary hospital convalescent's life. Each drug was tried until marked symptoms were obtained; then it was stopped. If his condition was not entirely relieved another drug was tried. Sometimes the three could be tried. He had not seen any cases where the patient remained dropsical after digitalis and strophanthus. Nearly all the patients were adults, but their ages varied, and he thought it would make too long a list of figures if he gave particulars of all the cases. Most were 20 to 30 years of age, and the cases with cardio-sclerosis were between 40 and 50. There were forty or fifty cases in the series, and only one in which the blood-pressure had been raised. In auricular fibrillation, where the pulse was continuously and completely irregular, it was difficult to know what was the right pressure. The results in these cases must be inaccurate, and so they had been neglected. All in which he had spoken of the blood-pressure had been cases of normal inflammation, in which the blood-pressure could be easily taken, and there could be no doubt about the comparative accuracy of the observation. In answer to one criticism—that of Dr. Duncanson—the fact that his patient had been taking digitalis did not prove that digitalis was the cause of his blood-pressure; at all events, in the absence of any previous record of blood-pressure. He did not think there was any evidence that that patient died of digitalis poisoning, or of the high blood-pressure. With regard to the ill-effects of vomiting in those cases, one always heard it was difficult to stop, but in none of his cases was there that difficulty. It always stopped without treatment in twenty-four to thirty-six hours. Vomiting was a safeguard against the patient taking too much, and in most of his cases the patient stopped it of his own accord. Within thirty-six hours of stopping the drug the vomiting ceased. He was aware of the inadequacies of the paper, for which he apologized, and thanked the Section for its reception.

Therapeutical and Pharmacological Section.

January 17, 1911.

Professor W. E. Dixon, Vice-President of the Section, in the Chair.

The Rôle of Auto-inoculation in Medicine: a Plea for its Rational Extension.

By Clive Riviere, M.D.

My thesis readily divides itself into two arguments which may be briefly stated as follows: Firstly, that a form of vaccine therapy has long been applied, unconsciously, but successfully, by physicians in their treatment of infective processes in the body. Secondly, that the success attending this practice might be increased if it were developed into a conscious aim under the guidance of recent knowledge of the subject of treatment by vaccines.

Part I.—Empirical Treatment of Infective Diseases an Example of Vaccine Therapy.

You will remember how gratified, as well as surprised, was M. Jourdain in Molière's comedy, when it was revealed to him that for forty years, unknown to himself, he had been talking "prose." When, however, it was pointed out to the medical profession that they had been practising the art of vaccine therapy for generations, not knowing what they did, the initial surprise was followed, perhaps, by a feeling of annoyance. It was annoying to see how far empiricism had out-stepped rational medicine, and to find so late in the day an explanation of the success which had certainly followed the "poulticings" and "fomentings" of the past. M. Jourdain soon proved that the prose he had used for so long in ignorance was good prose, and the science of the Maître-de-Philosophie failed to improve upon it. So far the same might be said for medical practice; the fomentation is a good remedy, but a knowledge of the principles which govern its action does not appear, at present, to have improved its application. It is to be hoped, however, that a different experience to M. Jourdain's is to follow, and that recent knowledge will now enable rational medicine to steal a march on empiricism.

Up to the present the local application of heat by poultices and fomentations has stood as the example of "empirical vaccine therapy." To these might be added numerous other homely, and sometimes barbarous, remedies, directed to the same end, such as the sucking of infected wounds, and the dressing of those with hypertonics and irritants, such as salt and pepper. All these remedies reinforced Nature's method of meeting infection by an increased blood supply to the offended part. But it is my object to show that the unconscious augmentation of irrigation and auto-inoculation did not stop with these simple remedies, but that the physician has held in his hands the weapons of the vaccine therapist from time immemorial, and has, indeed, used them, too, with no inconsiderable success.

Nature was the earliest "vaccine therapist." The processes by which infections are naturally overcome in the body form the basis on which the science of vaccine therapy is being built up. As soon as the invader "lands" in a tissue, there is a call for more blood, blood plasma acts on the microbe and its toxins, destroying them both by chemical processes (oxidation, hydration, dehydration, &c.) and by the action of specific antibodies, and leucocytes rush out to ingest the foe. If this proves insufficient, further antibodies are formed in response to the toxins carried over the body in the blood-stream, and the resistance of the plasma bathing the lesion is thereby raised. We thus have two natural processes to study and imitate :—

(1) *Irrigation* of the infected area with plasma and leucocytes, and this by itself may be enough to cure a mild invasion (such as a mild mucous membrane catarrh).

(2) *Auto-inoculation*, or the washing of the bacterial products into the blood-stream, to stimulate in the tissues the formation of antibodies specific to the invading micro-organism. These are the processes (other than the injection of vaccines) which the physician can direct with success, and it is this "natural vaccine therapy" that was exploited with success in the time-honoured practice of applying heat (poultices,

&c.) to an inflamed area. By this means not only was "irrigation" assisted, but such irrigation, itself of value, necessarily led to an increase of that auto-inoculation on which the improved resistance of the body depends. Such an effect is obvious so far as the poultice is concerned, but it is not so obvious, and it is my purpose to point out, how also the drug treatment of infective diseases may have depended for its success, so far as it has been successful, on a similar encouragement of these processes of "irrigation" and "auto-inoculation."

My contention will best be brought before you by its application to familiar examples. Let us consider infections of mucous membranes, and, in this connexion, turn to bronchitis, where there is general agreement as to the means to be employed.

Bronchitis.

It will not be disputed that the classical treatment of bronchial infections, though the outcome of empiricism, is, where thoroughly carried through, highly successful. It consists, shortly, in the giving of the so-called "depressant expectorants" till secretion is abundant, and then changing to the "stimulating expectorants," with perhaps, later on, certain of the balsams or volatile oils. This treatment is based on our experience that abundant exudation is necessary for cure. Abundant exudation implies either plus infection or adequate response, and the relative amount of these two factors, in any given case, demonstrates itself by the course which the disease takes.

If, now, we inquire into the pharmacological action of the "depressant expectorants," of emetine, of salts of antimony, of apomorphine, of the iodides, what do we find? We find that all these drugs cause a watery exudation through the bronchial mucous membrane. Emetine, antimony, and apomorphine are emetics, the two first through their irritant action on the gastric mucous membrane, the last by its action on the nervous centres in the medulla. Now, profuse secretion from the mucous surfaces of nose, pharynx, and bronchi is a constant by-product of the act of vomiting, as is also an increase in the sweat, tears, and saliva. But a similar effect is produced, though to a less extent, by sub-emetic doses of these drugs, and in this manner they become reliable expectorants. When we turn to the iodides we find a different pharmacological action; these substances are excreted through the bronchial and other mucous membranes, and cause a watery flow by their local action. In some cases iodine itself is liberated, leading to the more striking irritant effects generally known as "iodism." Thus it appears that these drugs, the classical stand-by in bronchitis with scanty secretion, though their mode of action varies, all lead to one result—namely, the bathing of the infected mucous membrane in a protective muco-serum or, in other words, increased "irrigation." Given a sufficient blood supply, they cause a rush of fluid through the newly formed mucous cells and glands on to the surface, imitating in an inaccessible area the osmosis we can so successfully obtain in a dry sinus by the local application of hypertonic solutions. Have we not here, also, an explanation of the value of emetics pushed to vomiting point in cases of bronchitis? That the removal of secretion by vomiting is not the sole gain by the administration of these large doses is shown by the benefit occasionally obtained from their use, even when vomiting does not occur. Surely the increased flooding of the mucous membrane with healing plasma is no small element in this favourable result.

At a later stage of catarrh, or in chronic bronchitis, the blood supply to the mucous membrane may also be in default. This depends on the general blood-pressure, which is generally raised above the normal in chronic bronchitis, perhaps as a compensatory measure. Now are used the "stimulating expectorants" squill and its congeners, and ammonium carbonate. These also, like the depressant expectorants, are gastro-intestinal irritants and emetic in full doses, and in virtue of this action increase the secretion from the bronchial mucous membrane. At the same time they raise the general blood-pressure by their action as vasomotor and cardiac tonics, and hence increase the blood supply to the bronchial tubes by both systemic and pulmonary blood-channels. Thus again, by our treatment, we are "irrigating" the diseased area, supplying blood-serum and leucocytes to the infected site in imitation of the natural process. When this is considered complete we attempt to coax the mucous membrane back to conditions of healthy secretion by such mild astringents and stimulants as the volatile oils (copaiba, cubebs, eucalyptus, &c.) and terebinthinates (terebene, &c.).

It must be borne in mind, of course, that the peculiar position of the bronchial mucous membrane demands recognition also of a mechanism for the expulsion of secretion, in addition to the repulse of the invader, and to aid this mechanical action the use of drugs acting on the respiratory centre, such as ammonia and strychnine, is sometimes called for. It will be remarked that most of the drugs we have mentioned are in request for other purposes besides that of expectorants, and it is interesting to note that in their other uses also their action might be interpreted as mainly one of "irrigation." In stomach catarrh we recognize the advantage of an emetic, though we do not always dare subject our patient, unless a child, to so unpleasant a remedy. We talk in this connexion of "emptying the stomach," but may not the most important result achieved be the "irrigation" of the stomach wall, which is a necessary accompaniment of emetic action? The efficiency of ipecacuanha in dysentery, and its value in small doses in some catarrhal conditions of the gastro-intestinal tract, clearly point to its known action as an "irrigator" of the diseased mucous membrane. Again, the terebinthinates and such volatile oils as copaiba and cubebs have had a recognized province in the treatment of infections of the urinary tract. Such an effect has been shown to have no clear dependence on antiseptic action, and is it not likely that their value has really depended on their irritant properties, or, in other words, on the "irrigation" they have caused? Examples might be multiplied, but I do not wish to waste your time, or add to my own responsibility by wandering over too wide a field.

PART II.—THE ADVANCES ON EMPIRICAL TREATMENT SUGGESTED BY A CONSIDERATION OF THE PRINCIPLES OF VACCINE THERAPY.

Now, in all the examples we have considered, the influence of the physician has been solely in the direction of irresponsible "irrigation." But Nature's armamentarium contains, in addition, the weapon "auto-inoculation," a weapon to be brought into use where "irrigation" alone has failed. The physician has neglected this weapon; he has been an "irrigator" pure and simple, whether with his poultice or in the drug treatment of bronchitis and other infective diseases. To be sure, auto-inoculation has necessarily occurred as the result of his irrigation, but has he directed it? Has he applied his fomentations at such intervals that time may be allowed between for the formation of antibodies? Has he calculated at what intervals he should auto-inoculate his phthisis cases to maintain most successfully their toxin immunity? Has he sufficiently calculated the dangers of hyper-inoculation and a "negative phase"? As a rule, he has done none of these things, and it is with the hope of directing attention to the neglect of auto-inoculation in medicine that the present paper is presented to you.

It is hardly necessary in these days to indicate the lines on which the vaccine therapist works; one must presume that the phases of a vaccination experiment are familiar to all. Suffice it to remind you that a sufficient interval must be left between every two inoculations for the "negative phase" of immunity to be passed, and a "positive phase" achieved.

It is obvious that the "auto-inoculator" has but one way of achieving his purpose, he can inoculate only by *increasing the blood-supply to the diseased focus.* But by doing so he also "irrigates," so that one act must serve these two purposes, though different considerations govern the indications for "irrigation" and "auto-inoculation." For "auto-inoculation" purposes the act should be *periodic* to allow time for the formation of antibodies and to avoid negative phases, while for purposes of "irrigation" the more continuous the flow the better. A happy union of these two aims may best be achieved by a strong "auto-inoculation" at certain intervals, and a fair amount of "irrigation" (with, of course, some amount of unavoidable auto-inoculation) maintained between. Auto-inoculation is open to one disadvantage of a more or less serious nature—namely, the fact that we must auto-inoculate into the blood-stream instead of into the tissues. It is, of course, well known that toxin immunity (exemplified in the preparation of antitoxins) is better achieved by subcutaneous than by intravenous injections, and the evidence generally seems to show that the most active formation of bacterial antibodies takes place in the tissues. Bacterial products must necessarily be greatly diluted in the blood-stream, and when they reach the tissues by this path are not so fitted to call forth a response as they would be if injected subcutaneously. Nevertheless, this apparent disadvantage accompanies all natural methods of cure, and by auto-inoculation we are but imitating Nature's mechanism. It may be that the tissues conjoining the diseased focus play a large part in this "antibody" formation, but at any rate we need hardly fear to follow Nature's lead.

It may be said that every method of treatment suffers certain disadvantages and limitations. Those of the artificial injection of vaccines may be briefly stated as follows:—

(1) The nature of the causative microbe may be impossible to determine; it may be out of reach, and general opsonic tests may be doubtful or misleading.

(2) The microbe, though its nature is determined, may be impossible to cultivate outside the body, as may happen in the case of certain strains of influenza bacillus.

(3) Many microbes may be at work, and it may be doubtful how far each one is responsible.

(4) There is of necessity a certain loss of time while the micro-organism is being found and a vaccine prepared.

The use of "auto-inoculation," on the other hand, suffers none of these disadvantages but shows, to be sure, certain *weak points* of its own. The following may be cited :—

(1) The dose given by auto-inoculation can never be so accurate as where subcutaneous injection is employed. Nevertheless, with a skilled appreciation of the nature of the infection and the response of the individual, the dose can usually be fairly accurately gauged, and readily confirmed by experiment. It may be remarked in this connexion that even for direct vaccination the dose is to a considerable extent experimental, both the toxicity of the particular strain of organism (which varies greatly) and the resistance of the patient having to be taken into account.

(2) The fact, already mentioned, that auto-inoculation occurs into the blood-stream instead of into the tissues.

(3) The possibility of doing harm : (*a*) Where, in an organ like the kidney with a firm capsule, irrigation is limited by space, and an increased blood supply might lead to stasis with suppression of urine and death ; (*b*) by the washing out of germs into the blood-stream and so generalizing a disease ; this is hardly a serious consideration except in the case of tuberculosis, where manipulation of an abdominal tuberculosis (never to be recommended) might lead to such a result. It is obvious that these dangers are not those of the method, but of its abuse in the hands of the careless or ignorant.

(4) Localized disease may be so localized that insufficient auto-inoculation may be producible for its cure. Such localization, on the other hand, may be a bar to successful treatment by the subcutaneous injection of vaccines also, unless special measures for increased irrigation are carried into effect.

It is obvious from a consideration of the weak points belonging to "direct vaccination" and "auto-inoculation" respectively, that their provinces do not overlap, that each has its special field of operations, and that they are in a large manner complementary the one of the other. Where disease is mild and recent a control of natural methods should prove sufficient ; where infective conditions are unduly prolonged artificial "irrigation" and "auto-inoculation" will cut them short. In chronic conditions, where the disease is within reach of the blood supply, the same measures should be successful in many cases.

In certain conditions it is obvious that the "direct" injection of vaccines is best. In very localized lesions, boils, or tuberculosis of surface structures, it is readily and successfully employed. In chronic septicæmias it offers the only chance of success, since this must depend on getting the bacterial products into the tissues. In conditions between these extremes there remain many and various infections where auto-inoculation and direct vaccination must strive for the mastery, with some triumphs, I would predict, for the former in the hands of an ingenious "auto-inoculator."

I have pointed out in what manner the time-honoured medical treatment of certain infective diseases falls into line with modern conceptions of natural methods of cure, I now propose to consider what modifications and amplifications of such treatment our recent knowledge of vaccine therapy suggests.

Bronchitis.

To turn once again to examples already given, we found that in bronchitis our attention has been directed too exclusively to irrigation, and that even here we have not always known when to stop. In acute and subacute infections of the bronchial mucous membrane "irrigation" is probably all we need consider, and the main point is to see that the flow of serum through the mucous membrane is continued long enough to destroy all the invading germs. It is where bronchitis tends to become chronic, or where repeated catarrhs occur, that a conception of the principles of vaccine therapy may enable us to add "auto-inoculation" to "irrigation" with a more reasonable expectation of success. In either case it appears that the invader has found a secure habitation in the mucous membrane, and the amount of exudation poured out (even where this is considerable) is only sufficient to hold him at bay. Now our means of getting rid of it are twofold: (1) By raising the protective power of the blood by auto-inoculation; and (2) by irrigating the focus in a thorough manner with this plasma of raised power. To auto-inoculate we must irrigate powerfully, and that at suitable intervals. Since we do not know the micro-organism with which we are fighting, it behoves us to strike such intervals as are generally suitable, since the inoculation curve does not vary much in length where different organisms are concerned. Once a week where auto-inoculation is large, or twice a week where small, will probably be most suitable, and we may now consider the means at our disposal.

We appear to have at least three means of strongly "irrigating" the bronchial mucous membrane :—

(1) *By increasing negative pressure in the bronchi* during inspiration a passive congestion of the mucous membrane will be produced, and this may be brought about by the use of a Kühne's mask, or by inspiring rarefied air in the pneumatic cabinet. This may be applied once or twice weekly, but it must be remembered that the mucous membrane may be filled with blood, and yet secretion may be inadequate. For this reason depressant expectorants, and perhaps alkalies, should be pushed for an interval preceding this treatment, and also given in smaller doses between to ensure sufficient irrigation in the intervals between each auto-inoculation. To relieve congestion remaining after use of the mask, the circulation might be relieved by deep costal respirations. At the same time counter-irritation of the chest wall and warm air will tend to relieve any passive congestion remaining.

(2) *By the use of vapours which are irritant to the bronchial mucous membrane* and so tend to cause an increase of secretion through it. For this purpose creosote and the cresols contained in various preparations may be used, evaporated by heat in a small chamber in which the patient sits, the treatment being preceded with advantage by a short course of depressant expectorants. By this means a considerable temporary irrigation of the diseased area is accomplished, and this leads to an auto-inoculation which, applied at the right intervals, should increase the antibodies in the patient's plasma. Here we have, perhaps, an explanation of the action of creosote vapour in bronchiectasis, and of the milder cresols in recurring catarrhs. Do not these act primarily as "irrigators" and "auto-inoculators," their antiseptic action, so greatly lauded, being of quite secondary and insignificant importance?

(3) The third method of strong irrigation and auto-inoculation, applicable to bronchitis in children only, is by the weekly or twice-weekly exhibition of emetics till vomiting is produced.

When should we apply these means of auto-inoculation in bronchitis? In recurrent catarrhs they might be used at intervals between the attacks to raise the resistance and destroy the infecting organism. In chronic bronchitis, or in bronchitis becoming chronic, they should be used just as injection of vaccines are used, at suitable intervals till success is achieved or despaired of. In cases of chronic bronchitis with freedom during the summer months an attempt might be made during this interval to dislodge the invader at the time when its hold on the mucous membrane was least secure.

Among the three methods here suggested for obtaining auto-inoculation in bronchitis my personal experience is confined to the use of the second only. Some practical success achieved by this means has encouraged me to add to the purely theoretical considerations of my paper these suggestions for their practical application to the treatment of this one variety of infective disease.

It will be readily perceived that the scope of the methods advocated is not bounded by the few examples for convenience considered here. To microbic infection of all regions the same rational means apply, though it is obvious that anatomical and other considerations must here and there change or modify their application. Thus in the kidney, and in other organs invested with a fibrous capsule, it is obvious that irrigation and its accompanying auto-inoculation can be of but limited extent, and an attempt to augment them beyond a certain point could only lead to stasis. Yet the theoretical considerations should not be disregarded, and even in kidney disease may lead us to new views. Thus we shall be led to look upon albuminuria in cases of kidney infection (not, of course, albuminuria of other causation), not as a mere loss of nutritives from the blood, but as the sign of a useful vital function—namely, the irrigation of the diseased focus with healing plasma. Thus (except so far as plus albumin implies plus infection) we can regard the quantity of albumin with equanimity, and see that its increase (perhaps under our own cautious manipulations), in cases subacute and tending to chronicity, may be but a step on the road to cure rather than a sign of further tissue damage. If it were not for its capsule, we are tempted to feel how much might be done for the kidney in conditions of infection. Such a thought necessarily brings the operation of "decapsulation" into our minds, and we are led to see that, at any rate in theory, this was a step in the right direction.

A glance round the field of medical treatment seems to reveal "irrigation" and "auto-inoculation" at the bottom of many successful empirical methods. The cold bath treatment of enteric fever so successfully employed in America, the use of ipecacuanha and of Epsom salts in dysentery, tar and other irritants in skin inflammations, emetics and purgatives in gastro-enteritis, abdominal massage in tuberculous peritonitis—may not all these be claimed as instances of "irrigation" and resulting "auto-inoculation"?

Lastly, might we not also improve on these methods, successful only in part, by a fuller recognition of this side of their action, and by directing our attention in future to the periodic spacing and control of that auto-inoculation we have for long unconsciously employed?

DISCUSSION.

The CHAIRMAN (Professor Dixon) said all present had probably lived long enough to know that periodically in the history of medicine there had been panaceas for disease. One followed another, and for the time being nothing sufficed but that use must be made of the new nomenclature and the new method of treatment. As an example one might take Ringer's work on salts. All that work had been done over again, but now instead of salts, the term "ions" was used. The interesting paper of Dr. Riviere had given another explanation for the action of a number of drugs. But he did not think the author intended to give any direct proof that his explanation was the correct one as opposed to the old-fashioned view. Personally he (Professor Dixon) had been charmed by his paper, but possibly people who were devoting their time to the administration of vaccines might regard his views with serious misgiving.

Dr. BAINBRIDGE said he did not quite understand what evidence there was that auto-inoculation occurred in some of those treatments. He supposed the evidence was that derived from massage of tuberculous joints and the like and the resulting changes in the opsonic index. He thought it was conceivable that the effect of altering the circulation through a given organ was as likely to be due to improved nutritional supply and improved resistance of that organ to infection as to any absorption of bacterial products and the production of antibodies. He also asked what was the difference between a depressing emetic and a stimulating emetic. He gathered that the definition had lost its meaning, and that in the doses usually given, emetics had no effect on the circulation.

Dr. CLIVE RIVIERE, in reply, dealt first with the effect of albumin passing through the kidney. He said that he looked upon the albumin merely as a measure of the plasma which might be passing through the kidney, and consequently carrying with it bodies which would act on the micro-organism present. In answer to Dr. Bainbridge, he reminded him that in the paper he divided the subject up into irrigation—or the exploitation of the pre-existant defence—and auto-inoculation, which added to this antibodies specific to the micro-organisms present in the lesion; in theory the one would precede the other, but in practice the two would go hand in hand. With regard to depressant expectorants and stimulating expectorants, he could only say that some of the modern books still kept up the distinction. Some of the expectorants, such as squill and the ammonia salts, acted on the circulation, according to some pharmacologists. In answer to a question by Dr. Cameron as to whether the emetics acting on the centres would also increase secretion from the bronchial tubes, it seemed, according to modern text-books, that emetic action was accompanied by an increase in the secretion of the bronchial tubes, and that it did not matter whether the drug acted directly on the mucous membranes or on the respiratory centres. Some of the older books implied that many of the drugs excreted through mucous membranes were also excreted through the bronchial membrane and acted in that way. But that did not seem to be the view taken by the more recent books. With regard to the cold bath treatment of typhoid fever, he suggested that the effect of cold on the surface was to throw the blood supply into the inner parts, and consequently irrigate the intestinal vessels, in the same way as in the drug treatment of bronchitis the bronchial mucous membrane was irrigated.

Therapeutical and Pharmacological Section.

February 21, 1911.

Professor A. R. Cushny, F.R.S., President of the Section, in the Chair.

On Drugs used as Local Anæsthetics in the Mouth.

By F. R. Smyth, M.R.C.S., L.D.S.

The subject of local anæsthesia is one which has attained considerable prominence during the last few years, and would probably be held in higher repute than it is but for the abuse which it has received at the hands of unqualified and unregistered practitioners, an abuse which is partly responsible for a certain amount of prejudice which exists in the public mind against some drugs which are used as local anæsthetics. Nevertheless local anæsthesia to-day holds a well-recognized place in surgery, and few would venture to deny that it has a sphere of usefulness, and that with its aid painless operations can be and are performed without involving loss of consciousness on the part of the patient.

Up to the time of the introduction of general anæsthetics the elimination of pain during surgical operations had always been one of the greatest problems of medical science. Before the discovery of general anæsthesia, which took place about the year 1844, efforts to overcome pain during operations had been comparatively futile. As late as 1832 Velpeau wrote: "To escape pain in surgical operations is a chimera, which we are not permitted to look for in our time."

Prior to the introduction of cocaine as a local anæsthetic, various methods had been tried for the purpose of locally obtunding pain. Physically reducing the temperature of a part by the application of cold is said to have been introduced in the middle of the sixteenth century by Bartholin and Severino. In 1807 it was utilized by Larrey, the celebrated army surgeon of Napoleon's time, for amputations. A more practical method of employing cold as an agent was introduced in 1866 by Sir B. W. Richardson in the form of the ether spray. The principal means now in vogue of applying cold is by means of ethyl chloride sprayed on the part. The compression of nerve-trunks for the purpose of producing local analgesia seems to have been practised from very early times, the modern means of applying this method being the Esmarch elastic bandage. For obvious reasons, of course, the use of the Esmarch bandage is inapplicable in the mouth. Various narcotic drugs, such as henbane, aconite, and the juice of the poppy-head, applied locally, were also made use of, but with small success. In connexion with solutions of narcotic drugs the galvanic current was called into use as long ago as 1807, by Reuss, and supporters of this method claimed to be able to produce, in many instances, complete local anæsthesia with solutions which would be quite inert if applied without the galvanic current. The modern process of cataphoresis is a revival of this principle, but is now comparatively little used. General medication by hypodermic injection was introduced by Alex. Wood in 1853, and this led to efforts being made to produce local anæsthesia by the injection of such drugs as morphine and tincture of opium. The results, however, were not encouraging.

The discovery of cocaine is credited to Niemann in the year 1859. The drug was but little used, however, until 1884, when Carl Koller, of Vienna, demonstrated its effects as a local anæsthetic at the Ophthalmological Congress at Heidelberg. This opened a new era in local anæsthesia, and within a short time cocaine came into general use. In the same year, 1884, Hall showed by experiments upon himself that he could inject solutions of cocaine hydrochloride into the infra-orbital nerve and anæsthetize the teeth. In the following year, 1885, Halsted, and some other investigators working independently at the same time, demonstrated that they could inject 9 ♏ of a 4 per cent. solution ($\frac{1}{3}$ gr. approximately) into the inferior dental nerve at its entrance to the inferior dental canal, and produce complete anæsthesia of the gums and teeth on that side of the jaw. The alarming symptoms which sometimes manifested themselves after injection of cocaine and the numerous fatalities which followed the early use of the drug proved a serious setback, but further experiments led to reducing the dose employed and also the concentration of the solution. According to Buckley, of Chicago, cocaine stands to-day with scarcely a rival as a local anæsthetic, but probably this view would not by any means meet with general acceptance.

It is my intention in this paper to give first a short description of the action of cocaine and then briefly to discuss the principal drugs which have been recommended as substitutes. Before doing so, however, it is necessary to refer to certain important general considerations as to the physical and physiological action on the tissues of solutions of drugs used for hypodermic injection.

In the first place it is essential that the solutions used should be isotonic—i.e., of equal osmotic pressure with the tissue fluids. It has been shown that aqueous solutions which are isotonic all possess the same freezing point, and the freezing point of human blood being 0·55° C., if we prepare solutions which freeze at this temperature we shall have fulfilled this condition. A 0·9 per cent. solution of sodium chloride, known as physiological saline, is the standard example of an isotonic solution.

If physiological saline solution is injected under the skin at body temperature, neither swelling nor shrinking of the cells occurs. If, however, hypotonic solutions—i.e., solutions containing *less* than 0·9 per cent. of sodium chloride—are injected, swelling of the tissues results, while hypertonic solutions, containing *more* than 0·9 per cent. of sodium chloride produce shrinking of the cells. If the freezing point of the solution is sufficiently far removed from that of the blood, the resulting tissue disturbances may lead to gangrene. In addition to this, both hypotonic and hypertonic solutions require more time for their absorption than isotonic solutions, and therefore the anæsthetic effect will be delayed.

Another most important factor on which the success of the operation largely depends is the production of local ischæmia, and the usual means by which this is brought about is by the addition of suprarenal extract to the solutions to be injected. Local ischæmia retards the absorption into the general circulation of the drug injected, the effect of which is twofold, viz.: (1) it increases the action of the drug on the local area; and (2) it minimizes the danger of toxic effects. In addition to this it enables us to use a solution of lower concentration, which again lessens the danger of intoxication.

This last consideration is an especially important one in regard to cocaine, for it has been shown that the relative toxicity of a given quantity of cocaine solution depends on the concentration of the solution. Reclus and others have demonstrated that a fixed quantity of cocaine in a 5 to 10 per cent. solution is about five times as poisonous as the same quantity in a $\frac{1}{5}$ per cent. solution. It is now generally agreed that the best strength of cocaine solution for dental purposes is 1 per cent., to which adrenalin chloride solution, 1 in 1,000, is added in the proportion of one drop to each cubic centimetre. Of such a solution 25 ℳ may with safety be injected at one time, thus keeping the maximum dose at a ¼ gr.

As regards the general action of cocaine, the first effect is that of a stimulant to the central nervous system. In small doses cocaine increases the higher functions of the cerebrum, while in larger doses the effect spreads to the lower areas and produces a great stimulation of the motor centres which may even lead to convulsions. The stage of stimulation is soon followed by one of depression, the depressant effects being frequently much the more prominent. Respiration is at first accelerated and at the same time deepened. Later it becomes gradually slower and weaker and stops from paralysis of the respiratory centre. Respiration ceases during the convulsions when these occur. As to the circulation, the heart is much accelerated, this being due either to direct action on the muscle or to stimulation of the accelerator mechanism, not to paralysis of the vagus, as is often stated, because stimulation of the vagus slows the heart even late in the poisoning. Cocaine being a local vaso-constrictor, there is a rise of blood-pressure.

The symptoms of cocaine poisoning may present very considerable variations. Small quantities may make the patient excited, restless, and somewhat garrulous, or may produce a calm languorous state, similar to that induced by small quantities of morphine, except that there is less tendency to sleep. The pulse is accelerated, respiration becomes quick and deep, the pupils are generally dilated, and headache and dryness of the throat may be complained of. Tremors, or slight convulsive movements, often occur. Later powerful tonic or clonic convulsions may supervene, the heart is extremely accelerated, breathing becomes rapid and dyspnœic and may finally be arrested during a convulsion. In other cases the convulsive seizures may be almost entirely absent, and fainting and collapse occur. In these cases the heart is slow and weak, the skin cyanotic and cold, respiration is depressed, and death occurs from cessation of respiration. Vomiting is occasionally seen in the early stages but is not common.

The fact must not be overlooked that the milder symptoms, such as tremors, faintness, &c., which are frequently seen after injections of cocaine and other drugs, are often due merely to nervous apprehension on the part of the patient, or even to the pain caused by the hypodermic needle and the actual injection, and not to any poisonous action on the part of the drug.

Having spoken of the general action of cocaine and of the symptoms of poisoning it is necessary to allude briefly to its local action. Cocaine has no action on the unbroken skin, but when injected subcutaneously it quickly produces loss of sensation by paralyzing the terminations of some of the sensory nerves, especially those conveying impressions of pain and touch. It is a general protoplasmic poison having, according to most authorities, a special predilection for nervous tissue. Buckley, however, denies this, affirming that it acts equally on all kinds of tissue, and that its action on the nerves is more prominent simply for the reason that they are the medium of sensation. In addition, cocaine is a vaso-constrictor, producing local ischæmia, this effect, however, is ultimately followed by congestion. It has also a slight irritant action, and when instilled into the eye occasionally produces a considerable degree of conjunctivitis.

Of the numerous drugs which from time to time have been introduced as substitutes for cocaine the following are the most important:—

Beta-eucaine.
Novocaine.
Stovaine.
Tropa-cocaine.
Alypine.
Nirvanine.
Holocaine hydrochloride.
Acoine hydrochloride.
Orthoform.

Of these, the last is insoluble in water and is not used for hypodermic injection, but possesses marked anæsthetic properties when applied locally in the form of powder to painful wound surfaces. Of the others, the one which possesses the greatest advantages for use in the mouth is undoubtedly novocaine. The reasons for making this claim will be stated subsequently, when comparing the individual properties of the various drugs named.

In estimating the value of a drug as a local anæsthetic in comparison with cocaine, Professor Braun has laid down the following conditions as essential:—

(1) In comparison with its anæsthetic effect, it must be less toxic than cocaine.

(2) It should be readily soluble in water, solutions should be stable, and should be capable of being sterilized by boiling.

(3) It should be non-irritating to the tissues.

(4) It should be compatible with adrenalin, and should not diminish the vaso-constrictor power of the latter drug.

As is well known, cocaine when boiled undergoes decomposition, so that in the case of this drug sterilization by this means cannot be carried out.

Beta-eucaine is a synthetic substitute for cocaine, which was first prepared by Vinzi in 1895, and has been largely used. The hydrochloride is the salt usually employed—it is a white crystalline powder soluble in 20 to 30 parts of water. The lactate is more readily soluble, and is therefore to be preferred. According to Prinz, of Washington, beta-eucaine is about three and a half times less toxic than cocaine, but slightly inferior in its anæsthetic effect. Solutions can be boiled without undergoing decomposition, this being a marked advantage over cocaine. It is, however, distinctly irritating to the tissues, and destroys to some extent the vaso-constrictor action of adrenalin.

One of the earliest substitutes for cocaine was tropa-cocaine. It was prepared synthetically by Liebermann in 1892. It is less poisonous than cocaine, but has less anæsthetic power. It is readily soluble in water, and solutions may be boiled without decomposition. Combined with adrenalin, it almost completely destroys the vaso-constrictor power of the latter.

Stovaine was prepared in 1904 by Fourneau, a French chemist. It is a crystalline white powder, readily soluble in water, its solutions having a distinctly acid reaction. It is said to be about half as toxic as cocaine, and to possess greater anæsthetic power. It may be sterilized by boiling, but is strongly irritating to the tissues, and may cause gangrene. It partially destroys the action of adrenalin. Stovaine has obtained a certain degree of notoriety in connexion with spinal anæsthesia.

Alypine is chemically closely related to stovaine, being synthetically prepared from the same source. Its solutions react neutral, but, like stovaine, it is strongly irritating to the tissues. According to some authorities it is more toxic than cocaine, others state that it is less so.

Nirvanine, holocaine hydrochloride, and acoine hydrochloride are synthetical products of the orthoform group. They are of little value as local anæsthetics, on account of their irritating properties, and one of them, namely, acoine, is considerably more poisonous than cocaine.

Lastly, to speak of novocaine, which, as already stated, appears to answer to the requirements of a local anæsthetic better than any other known compound. It was first prepared in 1905, and, like the other substitutes for cocaine which have been mentioned, is a synthetic body derived from the benzoic acid group. Chemically it is the hydrochloride of the methylester of p. amido-benzoic acid. It is a white crystalline powder which dissolves in an equal amount of cold water, and may be boiled without decomposition. Its solutions are neutral.

It possesses anæsthetic power equal to that of cocaine; it is non-irritating to the tissues, and when combined with adrenalin it increases rather than reduces the vaso-constrictor effect of the latter. For dental purposes the most suitable strength to employ is a 2 per cent. solution. As regards its toxicology, Prinz states that it is about six times less poisonous than cocaine, and that as much as 4 gr. have been injected with no ill-results. Liebl, a German investigator, injected into his own body 6 gr., and one hour after the anæsthesia had passed off he again injected 12 gr. in a 10 per cent. solution. Slight symptoms of intoxication followed, but had all disappeared in a hour and a half, leaving no after-effects.

In a "Report on Local Anæsthetics Recommended as Substitutes for Cocaine," in the *British Medical Journal*, 1909, Le Brocq,[1] of Cambridge, published the results of his investigations, which were carried out by experiments on frogs, mice, and rabbits. Attention was directed especially to studying the properties of the drugs in relation to the conditions enunciated by Professor Braun. The drugs reported on were beta-eucaine, stovaine, novocaine, tropa-cocaine, and alypine. His general conclusions as to relative toxicity were that beta-eucaine was the least toxic of all, while novocaine was rather less than half as toxic as cocaine. Alypine he found to be one and a quarter times as toxic as cocaine. Stovaine was found to possess the greatest anæsthetic power of all, but to be strongly irritating, and to produce sloughing. The results given by novocaine were more favourable than those given by any other drug, its anæsthetic power being found equal to cocaine, its toxic effect considerably less, and there being no irritation whatever to the tissues.

My own experience with novocaine, which extends to well over 100 cases, leads me to conclude that with the vast majority of individuals, assuming the dose injected does not exceed $\frac{1}{3}$ to $\frac{1}{2}$ gr., no toxic symptoms appear, but that its effect varies enormously on different subjects. To quote two extremes: I have injected as much as $\frac{4}{5}$ gr. without any sign of toxic symptoms, while in another case, after only $\frac{1}{6}$ gr., there was marked syncope, the patient for a time being almost pulseless.

As regards the relative anæsthetic effect of different drugs, my own observations would lead me to affirm that the selection of the drug is not a matter of supreme importance, but that other factors are of much more weight. The chief of these is probably the condition of the gum tissue into which the injection is made. Unless it requires considerable pressure to drive the anæsthetic solution into the tissues, successful anæsthesia is seldom produced. The best sign that the desired effect is being produced is the blanching of the area of tissue surrounding the point of injection. Where the gum is inflamed, congested, or spongy it is seldom possible to gain complete anæsthesia. Occasionally where the gum is apparently healthy, for reasons which are not obvious, the injected solution seems to escape into loose tissue and cannot be confined to the desired area. This more surgical aspect of the question, however, is outside the sphere of our present discussion and will not be gone into further.

---

[1] *Brit. Med. Journ.*, 1909, i, p. 783.

Besides novocaine I have used beta-eucaine, Parke Davis's eudrenine (1 per cent.), alypine, and various preparations containing 1 per cent. of cocaine. With most of them I have been able to obtain successful anæsthesia in some cases, but with all of them have had a certain proportion of failures.

In conclusion, I fear that these remarks have been somewhat disjointed, but I trust they may serve to promote a free discussion, and that those present will be kind enough to give us the full benefit of their knowledge and experience.

DISCUSSION.

Dr. H. H. DALE remarked that there was one point which Mr. Smyth did not mention, and which might, perhaps, be regarded as somewhat off the track of the subject, but which was of some interest to him, as a week ago he had to have a tooth extracted, and it was done under local anæsthesia. The dentist used novocaine, which was given with adrenalin. A few seconds after each of the small injections he was conscious of a very marked acceleration of the heart's action. The dentist said it had been obvious to him also, as he had been watching him, and his carotids must have been beating, for a short time, at 120 to 140 per minute. He wondered whether Mr. Smyth had had similar experiences, and whether it was due to a small quantity of the adrenalin having got into the general circulation. When the drug was given hypodermically, constitutional symptoms were very rare: the blanching of the tissues at the site of the injection was so complete that the circulation was practically stopped in the neighbourhood of the injection, and the base entered the blood-stream at such a slow rate that it produced none of the characteristic effects on the circulation. But in the case of the vascular tissue of the gum a small quantity might be absorbed. He would be glad to hear whether what he experienced was to be attributed to an unusual sensitiveness to the drug, or whether it was the general experience of dental surgeons to see constitutional symptoms, attributable to the adrenalin injected, and not to the local anæsthetic.

Mr. HEDLEY VISICK said that, as a dental surgeon, he had had a good deal of experience with the drugs named in the paper. In reference to Dr. Dale's remarks, he had noticed in several cases after adrenalin had been administered, there seemed to be acceleration of the heart-beat; in some cases it was very marked, and patients would speak of it. Possibly the patient's dread of the pending operation might have something to do with that symptom. One boy, aged 14, actually vomited, and his heart beat very rapidly. In his case, novocaine and adrenalin were used, and he attributed the effect to the latter; the boy, however, was in a weak state when the drug was administered. He asked whether Mr. Smyth could say by what tests the scientific determination of the toxicity of the drugs was established, as one naturally wished to use the safest drug. Some men stated that novocaine was six times less toxic than cocaine. He had used cocaine and novocaine, each in about an equal number of cases, and had only had one patient really ill, and that was the case of the boy already mentioned, though some had complained of a feeling of nausea. In his experience anæsthesia took much longer to establish under novocaine than with cocaine, and when the dentist was busy, that was a consideration, besides the desirability of relieving the patient of the tooth, and suspense as soon as possible. The solution he now used was Parke Davis's "Codrenine," which was a solution containing cocaine and adrenalin, each cubic centimetre containing one-third of a grain of cocaine hydrochloride and one one-thousandth of a grain of adrenalin chloride, with chloretone added as a preservative. He had obtained such good results from the use of this preparation that he did not feel disposed to depart from it. With regard to pain, some patients said after the operation that they felt slight pain; however, the previous passing of a probe into the gum, even to the bone, had not caused pain, so that possibly their impression arose from the fact that they were fully conscious of the amount of force used in the extraction, which they felt must cause pain. Most patients, however, admitted that they felt nothing whatever. As an experiment, he had tried using normal saline solution in one or two cases without any cocaine or other drug, and he had quite good results from it. Possibly there was some hypnotism or other mental state to account for the result, unless it was due to pressure of the solution on the nerve-endings; he would have thought, however, that the salt solution would have passed too quickly into the blood to act as a local anæsthetic. The patients, however, assured him that the pain was appreciably deadened; and after all, they were the best judges.

Dr. F. W. PRICE asked how many teeth it would be safe to extract at one sitting under local anæsthesia only. He was called in, in consultation, to see a woman who had had eleven teeth extracted under cocaine at one sitting; she almost died, and that led him to ask the question.

The PRESIDENT (Professor A. R. Cushny) remarked that several surgeons had told him that adrenalin injected into the gum caused flushing of the face and acceleration of the heart-beats. Many physicians had told him that when injected elsewhere in the body, adrenalin had no particular effect on the heart. The vascular supply and innervation of the gums was a little different from that of the skin; the gum seemed, at any rate in the dog, to be supplied with very many more vaso-dilator fibres, and there were thus greater facilities for absorption from the gum. If the adrenalin constricted the vessels of the subcutaneous tissue, it would scarcely be absorbed. But if it dilated the gum vessels owing to the predominant vaso-dilator effect, that might aid its absorption. In conclusion, he desired that the discovery of the anæsthetic effect of cocaine should be attributed to the right man—namely, a pharmacologist named von Anrep; not the person mentioned by Mr. Smyth.

Mr. SMYTH, in reply, said he had learned much in the discussion with regard to the action of adrenalin which had not occurred to him before. He had noticed that after a local anæsthetic was injected there was a certain acceleration of the heart, but not to the same extent as Mr. Visick had noticed it. What he had noticed more was the quickening of the respirations, and that he had attributed partly to nervousness, as it was more marked in nervous persons. No doubt the action of the adrenalin was responsible for the quickening of the pulse-rate in many cases, as it did occur in people who certainly were not nervous. With regard to the method of estimating the toxicity of the various drugs, Le Brocq, of Cambridge, conducted his experiments by injecting solutions of the anæsthetic drugs into rabbits, frogs, and mice, and noting the amount of the drug which caused death. He did not know what the other investigators did in their researches. He could not say he had found a great disadvantage from novocaine taking longer to produce anæsthesia than cocaine, although with the former, as a rule, one certainly did obtain the best results after waiting three or four minutes, but in most cases one could perform the operation within about one minute after the injection, because the process of injecting took some little time. He had had no personal experience with codrenine, but had heard it well spoken of. He felt safer with novocaine than with cocaine. He thought the amount of pain felt by the patient depended very much on the patient's temperament. The patient knew all that was going on, and his power of hearing was not dulled by the drug, nor was his sensitiveness to muscular force, and considerable force was expended in extractions. The degree of sensitiveness of a patient should be estimated before deciding to operate under local anæsthesia; where the patient was particularly nervous, it was better to give a general anæsthetic. He was not a very ardent supporter of local anæsthetics, and he did not think they at all excluded general anæsthetics; indeed, he thought he performed more of his operations under general than under local anæsthesia. With regard to Mr. Visick's remark that he had injected normal saline, and obtained an anæsthetic effect, that was what one would expect, although he had not himself tried it. It was known that the injection of any fluid into the gum did produce some degree of anæsthesia, and he believed it was largely a pressure effect, as pressure was one of the means of causing local anæsthesia. With regard to the maximum number of teeth which it would be safe to extract at one time, he thought a more important question was the amount of drug injected. As many teeth might be extracted as could be rendered anæsthetic with a safe quantity of the drug. In a patient in ordinary health, one-third, or even half, a grain of novocaine might safely be given, and ought to be sufficient for eleven or twelve teeth, unless very firmly rooted; though, of course, there would be more shock in such a case than where there were fewer taken out. His experience had been that it took a very small quantity to produce a good effect if considerable resistance were met with in injecting. By the time 2 ♏ or 3 ♏ had been injected there was blanching of a considerable area of gum, and sufficient anæsthesia for the extraction of one tooth. He had mentioned a case where he injected $\frac{4}{5}$ gr. of novocaine, but the patient was a robust type of individual, a farmer, about 50 years of age; in this case three very firmly rooted molar teeth were removed. One should feel one's way carefully, and if there were any signs of toxic effects, one would not inject any more of the drug. The drug was generally recovered from fairly soon; although ten minutes or so after the operation he had known patients complain of a feeling of giddiness, but it generally soon passed off. The fact which had been mentioned, that adrenalin did not produce marked effects on the heart when it was injected into other parts of the body seemed to support the idea that the acceleration of the heart observed was largely nervous or psychic; though, of course, what the President had said about the innervation of the gum might have something to do with it. He was glad to have his mistake corrected as to whom credit was due to for discovering the anæsthetic effect of cocaine.

Therapeutical and Pharmacological Section.

March 21, 1911.

Dr. J. Gray Duncanson, Vice-President of the Section, in the Chair.

An Inquiry into the Value of Rectal Feeding.

By W. Langdon Brown, M.D.

Although rectal feeding has been practised since the time of Galen, its efficacy has not been established beyond cavil. At the outset its limitations must be recognized. It is quite impossible to nourish the body by means of suppositories, which in all probability cannot be absorbed at all. In some cases the rectum and colon have been found loaded with them at the post-mortem examination. As the large intestine is the principal place for the absorption of water, it is essential, if we are to have any success with rectal feeding, to take advantage of this fact by giving the nourishment in a fluid form. But even in this form its efficacy is open to doubt. The power of the large intestine to digest foodstuffs is very slight. Whether undigested albumen can be absorbed at all is merely of theoretical interest; it is certainly absorbed in much too small an amount to make it of any value if thus administered. Erepsin is the only proteolytic ferment secreted by the intestine which can break down proteoses and peptones into simpler bodies prior to absorption, but can only act on caseinogen and fibrin among the native proteins. As erepsin ordinarily acts after pancreatic juice, we should naturally predigest the proteins by liquor pancreaticus before administering it *per rectum*; this has a further advantage over merely peptonizing agents in digesting carbohydrates and fats also. Even when the rectal feed is completely pancreatized, it is doubtful how far it can be absorbed. Though the large intestine absorbs water readily, food is normally absorbed as completely as it can be in the small intestine. The highly specialized epithelium over the villi is the main agent in this. It is a pure assumption to conclude that the widely different epithelium of the large intestine can act in a similar manner. Yet the administration of small nutrient enemata is based on that assumption. It has been claimed that the larger enema will get through the ileocæcal valve, and be absorbed in the small intestine. Church noted in a case of duodenal fistula that some of a soap and water enema reappeared through the opening. Charcoal particles administered in an enema have been found in the stomach. But we cannot rely on this regurgitation as a regular event. Boyd has looked carefully for it by the charcoal method, without success. Moreover, only large enemata can be expected to reach as high, and the larger the enema the more difficult it is to retain.

Turning from these a priori considerations to actual results obtained, I will first summarize some of the previous observations, and then my own experiments.

ABSORPTION OF PROTEINS.

Voit and Bauer found that it was advisable to add sodium chloride to assist absorption of albumen, though this conclusion is disputed by Ewald and by Czerny. Leube, in 1872, stated that he had been able to maintain life for six months on a mixture of meat and pancreas, both chopped very fine and injected by a syringe. Other observers adopting this method found it difficult to carry out, and apt to lead to marked putrefactive changes. Ewald used eggs, flour, dextrose, and red wine, the last addition being recommended because alcoholic solutions are sometimes absorbed when aqueous ones are not, and because of the astringent action of the red wine. More recent observers have used almost exclusively some of the convenient solutions of peptones on the market. Edsall and Miller, using milk and eggs with pancreatic extract, found that from 40 to 47 per cent. were absorbed. Huber found about 75 per cent. of peptonized eggs were absorbed, and Bial, in experiments on himself, found an absorption of 50 to 66 per cent. of a solution of peptone. Sharkey, using milk and powdered casein, found an absorption of from 50 to 75 per cent. Figures as high as these suggest that a comparatively large amount of nourishment can be obtained in this way. But there is a very important source of fallacy in them; they are based on the amount of protein which could be recovered from the bowel on washing it out. But it is notoriously difficult to remove the bowel contents completely in this way. Even when daily irrigations have been given with scrupulous care, there may be days afterwards an evacuation of a large amount of highly putrid material. And in any case, some of the protein which disappears may have been reduced by putrefactive changes into a form in which it has no nutritional value.

Boyd found that, even assuming that all the nitrogen which could not be recovered had been assimilated, nitrogenous equilibrium could not be obtained during rectal feeding even in those who were accustomed to a diet poor in nitrogen. He concluded that (1) protein food, even when predigested and with salt added, is very poorly absorbed in rectal feeding; (2) the albumen of eggs is very expensive and unsatisfactory in rectal feeding, to which it might be added that the great liability of eggs to putrefactive change adds very much to the nursing difficulties, which are already sufficiently great; (3) there is no relation between the amount of protein injected and the amount absorbed. Absorption seems to depend more on the patient's individual capacity than on the amount given.

Laidlaw and Ryffel estimated the nitrogenous output in a case of rectal feeding during coma, and found that it was approximately equal to that obtained in the later stages of fasting—as, for instance, with the professional faster Succi from the fifteenth to the twentieth day. The nutrient enemata in this case contained the white of nine eggs, 6 oz. of raw starch, and 24 oz. of peptonized milk in the day. It may be remarked that egg-white would not be readily absorbed, while it is doubtful if starch can be digested by the large intestine at all.

ABSORPTION OF CARBOHYDRATES.

Normally carbohydrates are absorbed by the bowel as dextrose, and of all the foodstuffs this appears to be the best utilized in rectal alimentation. Using recovery methods, Deucher found 77 per cent. absorbed, while Zehmisch got an absorption of 67·5 per cent. The highest values were obtained by Boyd and Robertson, who in two out of six cases found 100 per cent. to have been absorbed. It has been urged that here again bacterial decomposition accounts for much of the disappearance of the carbohydrate. Boyd found, however, that the *Bacillus coli* could only account for the disappearance of about 1 per cent., but does not state whether the lactic-acid-forming organisms could not be responsible for more than this. That dextrose is definitely absorbed from the bowel is, however, proved by the following facts: Reach found that the respiratory quotient was raised by rectal feeds of dextrose—a sure sign that they were being utilized by the tissues, and the acidosis of delayed chloroform poisoning or of inanition in œsophageal stricture has been reduced or abolished by this procedure. Now, just as nothing induces acidosis so quickly as deprivation of carbohydrates, so nothing abolishes it so rapidly as their assimilation.

ABSORPTION OF FATS.

No emulsion, however fine, is absorbed in the absence of a fat-splitting ferment. This ferment is normally supplied by the pancreatic juice, and in its absence we have to depend upon bacterial decomposition. It is a simple matter to provide the ferment by liquor pancreaticus, but even then absorption may be very imperfect. In one of Edsall's and Miller's cases only 13·61 per cent. of the fat was absorbed. The fat in the yolk of egg is considered to be better absorbed than other forms of fat, but personally I do not employ eggs in rectal feeding for the reasons already given.

Munk and Rosenstein made observations on a patient with a fistula of the thoracic duct. He was first put on a diet very poor in fat, and then enemata of fat and salt were given. In one experiment 3·7 per cent., and in another 5·5 per cent., was absorbed. They thought that the lower the melting-point of the fat the better it was absorbed. Deucher concluded that no more than 10 grm. are absorbed a day with difficulty even in favourable cases. Boyd takes a more favourable view of the utility of fats in rectal alimentation. His best result was the absorption of 45 grm. out of 103 grm. administered, and he thinks that fat is useful both directly and as a nitrogen sparer. He employed the recovery method, however, which, as already stated, is open to serious fallacies. The exceptional opportunities afforded by the case of thoracic fistula probably gives us the most accurate results as to fat absorption by the rectum, and here the amount was very small.

I have recently been able to carry out similar observations on a case of chyluria from filariasis under the care of Dr. Samuel West. On an ordinary diet the urine was quite milky, and he was placed on a fat-free diet to relieve the pain and occasional hæmaturia caused by the passage of large fatty masses. On this treatment the urine became merely opalescent, but even the addition of milk to his tea caused an obvious increase in the fat in his urine. After determining the amount of bodies soluble in ether and the saponification value of the urine on the fat-free diet, he was given an enema consisting of 120 c.c. of olive oil and 80 c.c. of 2 per cent. solution of Na_2CO_3, which had been pancreatized for an hour. On another occasion he was given a pint of pancreatized milk *per rectum.* It happened that the saponification value and the ether-soluble bodies were actually lower in amount on the days when these enemata were given. This indicates that no fat was absorbed from these enemata by the large bowel.

ABSORPTION OF SALT AND WATER.

It is agreed that salts and water are freely absorbed from the large intestine and the advantages claimed for rectal feeding are probably due to these ingredients. It is well known that the body can stand deprivation of food for a considerable time if these are supplied. W. Pasteur, in 1904, advocated the administration of 10-oz. enemata of plain water at a temperature of 100° F. every four or six hours. He claimed that the results were at least as good as with the ordinary nutrient enemata, while it is far simpler and pleasanter for the patient. Sharkey has used $\frac{3}{4}$ pint of saline four times in each twenty-four hours, and has been equally impressed with the advantages of this method.

NITROGENOUS METABOLISM DURING RECTAL FEEDING.

My own observations were chiefly directed to comparing the nitrogenous metabolism in patients on rectal salines and nutrient enemata. Some of them have been carried out on my own patients, and others on patients under the care of Dr. Samuel West and Dr. Herringham, at St. Bartholomew's Hospital, to whom I desire to express my thanks for the opportunities they have placed at my disposal. The nutrient enema which I have usually employed is composed of—

4 oz. of milk.
1—2 dr. of plasmon.
1—2 dr. of dextrose.
20 gr. of bicarbonate of soda.
1 dr. of liquor pancreaticus.
5 ♏ of tinct. opii.

The liquor pancreaticus is allowed to act for twenty minutes at 37° C., the opium being added just before administration, with the object of increasing the tolerance of the bowel. The bicarbonate of soda is added to imitate the normal alkalinity of the pancreatic juice (1 per cent.). This enema is given every four hours, the rectum being washed out every night and morning. The total foodstuffs thus given in the twenty-four hours amount to—

Proteins	75 grm.
Carbohydrates	75 ,,
Fats...	27 ,,

This is clearly much less than the minimum required to keep the body in nitrogenous equilibrium, even supposing it were all absorbed, which is far from being the case, while its caloric value is only 866.

The larger enema, a pint of milk three times a day, has not yielded me very good results, though recommended by some authorities, the patient usually failing to retain them after the first day or two. This would be equivalent to 68 grm. of each of the three foodstuffs, and its caloric value would be 1,188. I have no personal experience of the enema administered continously drop by drop, but I have heard from those who have that it caused the patients so much discomfort that they were forced to abandon it. The enemata were administered in my cases by a soft rubber catheter attached to the barrel of a 4-oz. glass syringe, the contents being allowed to flow in slowly by gravitation.

And now, as to results. When no nitrogenous food is taken there is a steady fall of nitrogen in the urine till the output reaches 5 grm. a day, and may be lower. One of the first effects of giving nitrogenous food will be a rise in the nitrogenous output in the urine, as the greater part of the urea is exogenous—i.e., comes from the food. On the other hand, the purin bodies and the kreatinin remain constant, while the ammonia may rise a little to neutralize the abnormal acids produced. The nitrogenous output in the urine will therefore be a more accurate criterion of the absorption of nitrogenous material from the bowel than the loss of nitrogen from the rectal washings, because it is evidence of actual assimilation. When rectal salines are given there is a steady fall of urinary nitrogen. Starting with 10 grm. to 11 grm. the first day, it falls to about 5 grm. by the fifth day, and usually remains at that point, though in some of my cases it fell as low as 3 grm., or even 2 grm., a day. If the volume of the urine varies considerably from day to day it is necessary to take the average of three-day periods. Acetone usually appears by the end of the first twenty-four hours, and diacetic acid within the next twenty-four hours. On comparing this with the cases where the standard enema was employed hardly any difference was detected in the nitrogen output. There was a steady fall in the total nitrogen down to 5 grm., and in one case as low as 4 grm. The onset of acetonuria was, however, delayed as a rule, and it was usually less severe and of shorter duration. The great factor in the production of acetonuria is deprivation of carbohydrate, and as the enema contained both dextrose and lactose the disappearance of acetonuria indicated that some of these were absorbed. In some cases acetonuria persisted throughout.

In one experiment a somewhat crucial test was employed. Salines were given till the nitrogen in the urine fell to 5 grm. The standard enemata were then given for two days, but the nitrogen output was hardly affected at all. The same amount of food was then given by the

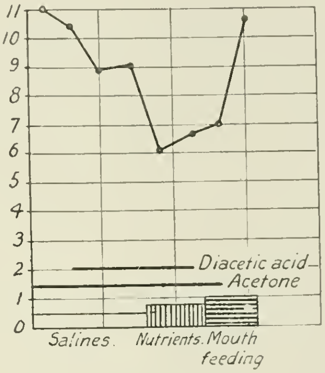


FIG. 1.

Nitrogenous excretion on rectal salines followed by nutrients.

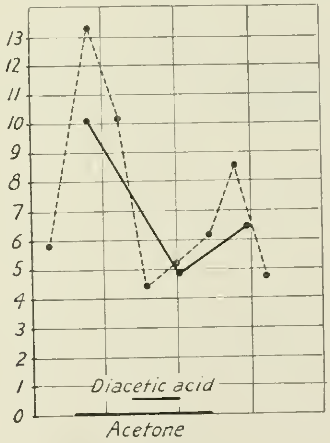


FIG. 2.

Nutrient enemata throughout.

- - - - - - daily excretion of nitrogen ; ——— average of three-day periods.

mouth and the nitrogen output promptly rose. In other cases the effect of adding 5 per cent. of dextrose to the salines was investigated. We may presume that complete evidence of its assimilation would be afforded by a cessation of the acetonuria and a sudden drop in the nitrogen excretion, as the absorbed carbohydrate should spare the nitrogen waste of the tissues, and also do away with the necessity of excreting some of the nitrogen as ammonia to neutralize the acidosis. In one such case the average nitrogen output on three successive days with rectal salines without dextrose was 11·68 grm. of nitrogen, and on the next three days with the addition of 5 per cent. of dextrose to the salines it was 10·18 grm., so that the result was not very striking. Both acetone and diacetic acid persisted in the urine, so that the amount of dextrose absorbed could not have been very large. In another case the addition of 5 per cent. dextrose produced hardly any effect on the curve, and acetonuria persisted. Then food was given by the mouth, the nitrogen output began to rise again, diacetic acid promptly disappeared, and acetone vanished a day later.

A statement usually made in regard to nitrogenous metabolism has always offered difficulties which I think my experiments help to clear up. If to a starving animal or person an amount of nitrogen is given in the food equal to that excreted in the urine it is stated that the whole of the administered nitrogen is excreted. Thus, if with no ingested nitrogen the urinary output is 5 grm. it is asserted that with an intake of 5 grm. the output would be 10 grm.; and it is generally admitted that at least two and a half times as much nitrogen as was being excreted in starvation must be given in the food before nitrogenous equilibrium is attained. For a long time this apparently wasteful method of metabolism was a mystery. Then when it was realized that the change from food protein to tissue protein was so profound, a partial explanation was reached; for the tissues had to have a large variety of the ultimate products of protein disintegration—the amino acids—presented to them from which to select the groups they needed for their anabolism. It must necessarily be that a considerable number of the ingested groups could not be utilized, and these would be converted into urea and excreted. But the appearance of the whole of the ingested nitrogen would mean that none of the amino acids could be utilized. This seems improbable, and I have noted in several cases the rise in urinary nitrogen was not proportional to the ingested nitrogen. Thus in fig. 3 the nitrogen of the food given by mouth was 3·15 grm., while the rise in the nitrogen excreted was only 1·4 grm. Yet acetonuria promptly vanished, indicating that the patient was being nourished. It is probable that not the whole but only a part of the food nitrogen is promptly excreted, which would accord with what we know of the methods of nitrogenous metabolism. The difficulty, therefore, is really non-existent, being based on a mis-statement.

It should be added that all the cases investigated, except one, were those of recent hæmatemesis from gastric or duodenal ulcer. The remaining one was occlusion of the œsophagus from corrosive poisoning.

PRACTICAL DEDUCTIONS.

I think these results go to show that if any protein food is absorbed from nutrient enemata the amount is so little as to make it hardly worth while to subject patients to so much discomfort for so small an advantage. A gain in weight has been claimed as evidence of their value, but this has been observed also on rectal salines. The addition of 5 per cent. of dextrose to the salines is probably an advantage, as enough of it may be absorbed at any rate to check acidosis. Even in 5 per cent. solution it is apt to cause pain by the osmotic currents it sets up, and in higher concentrations this is almost certain to occur. Boyd concludes that the average caloric value of the absorbed food in his experiments was 389—i.e., about one-ninth of that of a normal diet—and his criterion of absorption was a rather lenient one. Moreover, all his cases were those where rectal feeding was well borne, for if it was not other methods were adopted.

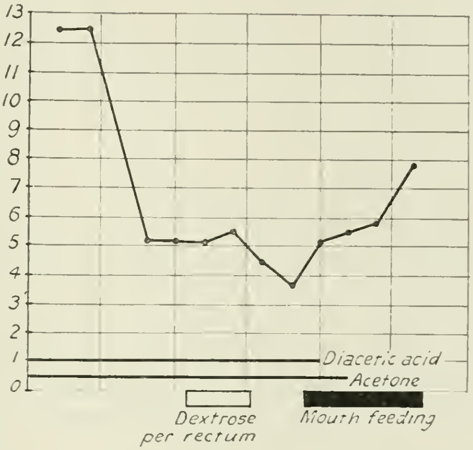


FIG. 3.

Rectal salines followed by salines + 5 per cent. dextrose.

When one begins to doubt the efficacy of rectal feeding, the inconveniences associated with it begin to assume a greater degree of importance. As disadvantages must be mentioned: (1) The thirst, which should be relieved by giving a rectal saline as well. (2) The difficulty in keeping the patient in a cleanly condition. (3) The secretion of gastric juice which it causes. The most conclusive evidence that this occurs is afforded by the observation of Umber, who found in a patient with a gastrostomy wound that the injection of food *per rectum* was followed by the secretion of an acid gastric juice. It is hardly securing a condition of physiological rest to allow this juice to be poured over an ulcerated surface without having any food on which to act. To neutralize this juice I was accustomed to give the patient bismuth lozenges to suck. This serves both to neutralize the acid and to form a protective covering to the ulcer. At the same time, by keeping the salivary glands active, it diminishes the chance of (4) parotitis, which is due to an ascending infection of the salivary ducts. I am quite sure that in this way it is much easier to keep the mouth clean. Incidentally, I should like to protest against the use of glycerine in a mouthwash for this or, indeed, in any other condition. The desiccation which follows only aggravates the state of the mouth. Plain hot water, to which a little potassium permanganate has been added, is, in my opinion, much to be preferred. Ice is also objectionable; though pleasant at the time, it aggravates thirst. (5) Persistent vomiting is an occasional complication in rectal feeding. If this starts it generally persists until mouth-feeding is resumed. Unfortunately, it is often regarded as a sign that the stomach cannot tolerate anything, which is not the case. I believe it is due to the acidosis consequent on starvation. (6) The pronounced subnutrition induced by rectal feeding is very unfavourable to the healing of an ulcer in patients who are already in a poor physical condition consequent on one or more hæmorrhages, and may lead to serious inanition. Self-digestion goes on more rapidly in fasting than in well-fed tissues, and this may lead to extension of the ulcer and recurrence of the hæmorrhage. It is useless to think of building up a patient by this method to a condition of improved nutrition prior to operation, for, say, an impermeable œsophageal or pyloric obstruction. I have only once attempted this at the request of a surgical colleague, and I shall not repeat the attempt.

The aim of rectal feeding is to ensure physiological rest. This is an ideal which in many cases is impossible of realization. The heart must continue to beat, though the valves are studded with vegetations, and the pericardium is a bag of pus. The lungs must continue their labours, though many of the alveoli are consolidated, and the pleural membrane is coated with lymph. The kidneys must continue to excrete, though their swollen tubules are engorged with blood; and though active ulceration may be proceeding in the stomach, the patient must be fed. Rectal feeding is incapable of effecting this, and by favouring self-digestion may frustrate the very object we have in view. In the Lenhartz method of immediate feeding with small and repeated quantities of iced milk and beaten-up egg we have a safer, simpler, and more effective treatment for gastric ulcer, which is free from any disagreeable features. If for any reason this is considered inadvisable, rectal salines with 5 per cent. of dextrose may be used for a time. Between these two procedures there is, in my opinion, no place left to-day in therapeutics for the nutrient enema containing protein or fat.

DISCUSSION.

The CHAIRMAN (Dr. J. Gray Duncanson) remarked that the paper was a most interesting and valuable one from the point of view of the general practitioner. It was a great advantage to know that, notwithstanding the benefits said to be derived from complicated rectal feeding, such method possessed little value beyond that obtained from the administration of isotonic saline solutions; the latter was much simpler, and especially in cases where the patient was being looked after by the relatives or by one nurse. He had not prescribed nutrient enemata for some years, but relied on saline solutions.

Dr. BAINBRIDGE said that at first sight it was somewhat surprising that protein products should not be absorbed, because he supposed that a considerable number of drugs which had molecules as large were absorbed from the rectum—he referred to such a drug as opium; one would have expected, a priori, that amino acids would be absorbed. He wondered whether Dr. Langdon Brown had tried a fairly advanced proteid digest. Then there was the question of the presence of creatin in the urine, which Cathcart had shown to be a fairly delicate indication of starvation, and he would like to know whether that had been observed as an index of the degree of absorption of protein or other food from the rectum. He did not quite understand why a 5 per cent. solution of dextrose should cause pain, because a 6 per cent. solution only was isotonic; therefore he did not see how it could be due to any osmotic effect, unless one started with an isotonic solution, and added dextrose. He would imagine that dextrose would be valuable in post-anæsthetic poisoning where the vomiting was often uncontrollable, and it might tend to reduce the acidosis and actually supply food to the starving tissues. But he supposed that in those cases it would possess no advantages, and would be rather inferior to the subcutaneous method of administering the dextrose. He did not know whether Dr. Langdon Brown had had experience of the two methods in such cases.

Dr. H. H. DALE said it was difficult to see why simple crystalloid substances like amino acids should not be absorbed from the bowel, and he wondered if it was possible that the very rapid putrefaction which took place in the large intestine was really the cause of the non-absorption—*i.e.*, whether they were broken down by putrefaction and converted into simple bodies so quickly that they were not absorbed as amino acids. The author mentioned that nutrient enemata caused a secretion of gastric juice. Presumably that was either a nervous reflex or due to the absorption of something from the enema. If it were simply reflex, one would think that any distension of the large bowel with anything, whether by salines or by nutrient enemata, would have the same effect. If it were the result of absorption, it seemed inconsistent with the evidence that no absorption took place.

Dr. NESTOR TIRARD said the points which interested him were the connexion, or want of connexion, between the acidosis and the question of feeding by salines or by nutrient enemata. He understood one of the charts to represent that there was more diacetic acid and acetone formed during the administration of saline. The other chart appeared to represent that there was only a small amount of diacetic acid when the feeding was by nutrient enemata. He did not know whether it was contended that one was to be preferred to the other on that account. He would have liked to have heard whether during the administration of either saline or nutrient enemata, Dr. Langdon Brown had seen any disadvantage from the presence of the diacetic acid and acetone in the urine. During the past winter he had had two cases, particulars of which he had reported elsewhere, in one of which there were very marked symptoms of acidosis, such as one would expect to find in a case of diabetic coma; the onset of heaviness, drowsiness, and headache—those symptoms having occurred during the administration of nutrient enemata in the treatment of gastric ulcer. During the comatose state diacetic acid and acetone were found present, and as they persisted, the patient was fed by the mouth with bread and milk, as though convalescing from typhoid fever: saline solution had already been given subcutaneously. The feeding by the mouth was done in fear and trembling, but the patient recovered, the symptoms of coma passing off quickly with the disappearance of the acetone and diacetic acid from the urine. The other case to which he referred he would speak of more guardedly. It was a post-operative case, in which a large part of the stomach and the first part of the duodenum had been removed on account of malignant disease, and the patient was being fed by nutrient enemata. That man died suddenly with symptoms of what was commonly styled air-hunger. There was no urine available until the post-mortem examination. Post-mortem search was made for any embolism in the lung which would account for the sudden difficulty in breathing, but such was not found. Urine was withdrawn from the bladder, and acetone and diacetic acid were found in it. He connected the symptoms in both cases with the presence of those substances in the urine. Therefore, although he was in agreement with Dr. Langdon Brown about the disadvantages of attempting to feed patients for too long a time on proteids by the rectum, as to the uncleanliness, the insufficient nourishment and liquid which the patient derived, also as to the advantage of giving salines, he was not sure that he quite agreed with him in all that he understood him to have said about the relative unimportance of the diacetic acid and acetone in the urine as compared with the value of the saline over the value of nutrient enemata.

Dr. LAIDLAW said he had the opportunity of investigating a case which was fed for a considerable time upon nutrient enemata, and in which, on the twentieth day, a very characteristic picture was seen in the urine. It was similar to the cases described by Dr. Langdon Brown, but more advanced. There were about 4 grm. of nitrogen, a large quantity of acetone and β-oxybutyric and aceto-acetic acids. The urea and other nitrogenous constituents corresponded almost exactly with the figures which had been published of the metabolism of the professional fasting men, as, for example, those for Succi on the fifteenth day of his fast. The patient was obviously not doing well, and was gradually getting weaker, so that on the twenty-seventh day the œsophageal tube was passed, and a similar amount of nutrient material was introduced in that way. There, again, the amount of nutrient did not mount up to the maximum value which one would expect to find, and in this manner also corresponded closely with the metabolism of fasting people. When they first came out of their fast, and reverted to mouth-feeding, their nitrogen remained low, sometimes for days afterwards. With regard to the changes which took place in the nutrient enemata, one could not detect more than small quantities of proteoses and peptones, but the characteristic feature of the returned nutrient enemata was the large quantity of lactic acid present. Probably this was the result of bacterial action. He did not think there was any doubt that the people who were supposed to be fed on nutrient enemata were being starved, their urine and metabolism corresponded so closely with what was found in professional fasting men during one of their fasting periods.

Dr. LANGDON BROWN, in reply, said he had not tried nutrients containing products of advanced protein digestion, but he was proposing to do so. He had been anxious to prove that rectal foods, as ordinarily given, were of no use. He agreed that it seemed improbable that under no conditions could one get amino acids absorbed by the large bowel: but the ordinary rectal feed given did not get absorbed. He thought it was possible, as Dr. Dale suggested, that the rapid putrefaction was a factor in preventing absorption. He had not tested the point as to creatin. He thought there being acidosis justified him in saying that the patient was starving. He did not know how to account for the pain which certainly occurred in some of the cases when 5 per cent. dextrose was given, except by osmotic currents due to the addition of sugar to a solution already isotonic. With regard to the suggestion that the vomiting in some cases of rectal feeding was due to acidosis, he admitted that it was open to the retort that certain cases had severe acidosis without vomiting; but there were some cases of acidosis which did not seem to develop any toxic symptoms, whereas, as Dr. Tirard pointed out, others did. He admitted there was a curious dilemma about gastric secretion being excited by rectal feeding. Whether it was a nervous reflex, or whether it was a chemical one, he did not know. If it was chemical it suggested there was some absorption. It was possible that some extractives from meat might be absorbed, and might produce that effect. He hesitated to bring forward the suggestion that some of the secretion was due to excitation of the psychic centres. Many patients thought they were getting nourishment when fed in that way, which would in itself tend to stimulate the secretion of gastric juice. In answer to Dr. Tirard, he reminded him that he was trying to point out that the acidosis was a symptom of starvation, and that there was a little more starvation when the patient was on rectal salines than when he was on nutrients. He thought the difference was entirely due to the carbohydrates in the latter, and not due to the nitrogen. If one simply added dextrose to the saline the acidosis would be checked as readily as by nutrients. But he was not supporting either of those methods. What he had tried to establish—and Dr. Tirard's cases were favourable to that view—was that the proper thing to do was to feed by the mouth. Lenhartz's contention, which was supported by other people's experience, was that it was safe to feed by the mouth, that hæmorrhages did not take place any more frequently, and, in fact, they were less frequent than when rectal feeding was carried out, because self-digestion was not going on in the stomach in the former case. He had compared the nitrogenous output of people fed on Lenhartz's diet with those fed by the rectum, and found the former much better; there was no acidosis, and their nitrogen excretion was maintained at the proper level. Physicians had been much too strict in stopping mouth-feeding in any form in cases of gastric ulceration. The dangers of self-digestion had been largely lost sight of. Dr. Laidlaw's work in comparing the nutrition of the fasting man with a person having nutrient enemata was very useful. The conclusion practically was that the patient fed upon nutrient enemata had the same kind of metabolism as that seen in a professional faster.

Reactions of Arteries to certain Drugs.

By DOUGLAS COW, M.D.

METHOD.

THE arteries used in the experiments which form the basis of this paper were obtained from freshly killed sheep and oxen, with one or two exceptions—a few being taken from recently killed goats and rabbits, and in one or two instances the arteries were obtained from a limb just amputated from a living human subject. The arteries, as soon as they were removed from the body, were placed in oxygenated Ringer's solution at a temperature of 35° C., in which they were kept until required for the experiments. The experiments were performed as soon as possible after the removal of the arteries, though, where suitable precautions were taken, it was found that the arteries retained their power of reacting to stimuli without appreciable impairment for many hours after removal. Fresh Ringer's solution was used in each experiment and was maintained throughout at 35° C. The arteries were carefully freed from the surrounding connective tissue, and the greatest care was taken to avoid contamination of the arteries, all the necessary manipulation being done with clean forceps and scissors.

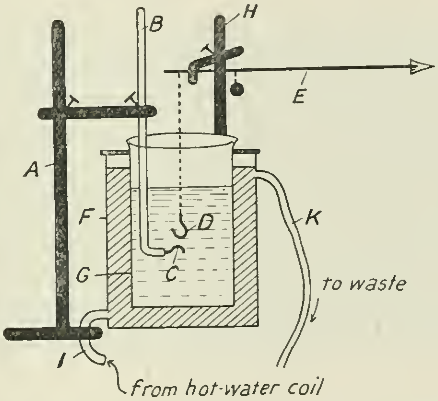


FIG. 1.

Apparatus: *A*, rigid stand; *B*, glass rod; *C*, platinum hook; *D*, platinum hook; *E*, writing lever; *F*, water-bath; *G*, beaker; *H*, upright; *I*, *K*, tubing.

The apparatus in which the experiments were carried out is shown in fig. 1. It consisted essentially of a glass beaker containing a known volume of Ringer's solution; this was supported by a wooden ring, so that the beaker was immersed in the circulating water in a metal water-jacket, the temperature of which was kept constant by means of a copper coil over a Bunsen burner. Into the beaker a vertically placed and rigidly held glass rod dipped, the lower end of which was bent at a right angle and carried a fine platinum hook, with its concavity facing downwards. Immediately above this a second platinum hook, with its concavity facing upwards, was suspended by a silk thread from the short arm of a writing lever. Suitable balancing weights were hung from the near end of the long arm of the writing lever, whose fulcrum consisted of an easily moving pivot-bearing.

Following the method of O. B. Meyer, rings were cut from the arteries about 2 mm. in width and suspended in the Ringer's solution between the two platinum hooks, sufficient tension to keep the silk thread taut being supplied by the balancing weights. The revolving drum on which the writing lever recorded the tracings moved slowly, the same rate being maintained in all the experiments—viz., 1 cm. in 42 sec. passing the writing point. With this arrangement any constriction would be shown by an upward movement of the writing point, likewise dilatation by a downward movement. Liquid stimulants were applied by instilling the drugs into the Ringer's solution from a glass pipette; and by a simple arrangement gases could be bubbled through the Ringer's solution, so that their effect on the arterial ring could be noted. One hundred and seventy-six experiments were performed, the results of eight of which were discarded, as in these instances it appeared doubtful whether the arteries were in a condition to give a normal reaction. In the remaining experiments there appeared be no reason to doubt that the vessels were reacting naturally.

The stimulants employed were: (1) Carbon dioxide, (2) alcohol, (3) adrenalin, (4) barium chloride, (5) digitalis, (6) pituitrin, (7) ergot, &c., (8) nitrites, (9) calcium chloride, (10) sparteine, (11) certain diuretic drugs. Excepting in those instances in which the dose applied is definitely stated, 7 ♏ was the dose used. This gave the following as the approximate strength of the drugs in dilution in the Ringer's solution: Alcohol, 1 per cent.; adrenalin, 0·001 per cent; barium chloride, 0·1 per cent.; digitalis, 0·1 per cent.; pituitrin, 0·1 per cent.; ergot, 0·01 per cent.; nitrites, 0·1 per cent.; sparteine, 0·01 per cent.

CARBON DIOXIDE.

That the products of metabolism due to the activity of an organ cause dilatation of the vessels of that organ is recognized. Barcroft and Dixon [1] conclude that carbon dioxide produces dilatation of the coronary arteries.

I performed five experiments with carbon dioxide as the stimulant, the arteries used were the carotid, coronary and gastric. The results showed in every case a uniform dilatation of the artery when the gas was allowed to bubble through the Ringer's solution, though the solution was liberally oxygenated. The dilatation was more pronounced in the case of the carotid and gastric than in the case of the coronary artery. So long as the gas was passing through the Ringer's solution the dilatation continued. When the passage of the gas was stopped the dilatation perceptibly diminished, though it did not actually cease. Following doses of carbon dioxide produced similar results.

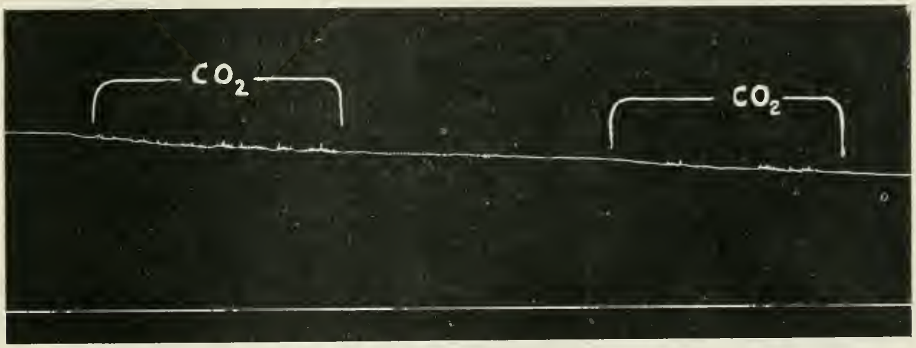


FIG. 2.

Ring preparation of carotid artery of sheep. At the indicated marks CO_2 was allowed to bubble through the saline. Time: 1 cm. in 42 sec.

Seeing that carbon dioxide produces the same effect upon the coronary as upon the systemic vessels, whilst their innervation is different, the presumption is that the dilatation is brought about by the action of the gas upon the musculature and not upon the nerve-endings of the vessels.

ALCOHOL.

Dixon [6], in a short résumé, concludes that alcohol in moderate doses produces dilatation of the superficial vessels; whilst in larger doses slight constriction, followed by dilatation of the internal vessels, takes place. He concludes, too, that this action is partly central and partly peripheral. Kobert [9] states that dilute solutions of alcohol have little or no effect upon the renal vessels.

I carried out five experiments with this stimulant, the arteries employed being the carotid, gastric and coronary. The results showed a slight evanescent constriction, preceding a more pronounced dilatation in every case. The fact that the effect produced was of the same nature in each case argues an action on the muscle and not upon the nerve-endings. The total effect was, however, but slight, which would seem to show that the main action of alcohol on the arteries is not peripheral.

ADRENALIN.

Schäfer and Oliver [16] first showed the constricting effect of extract of suprarenal on the blood-vessels. Brodie and Dixon [2] showed that adrenalin had no constricting effect when perfused through the lungs. Plumier [13] and Wiggers [18], however, deny this. Schäfer [14] concludes that the coronary arteries are not furnished with vaso-constrictor nerves, since these vessels do not constrict under adrenalin. Wiggers [19] found that adrenalin in dilute solution when perfused through the cerebral vessels in situ caused constriction; he also found that adrenalin constricted the coronary and pulmonary vessels. Dixon and Halliburton [7] found that perfusion of adrenalin through the cerebral vessels causes slight dilatation of these vessels when the brain is removed from the body. Langley [10] observed that adrenalin has a specific power of stimulating plain muscle,

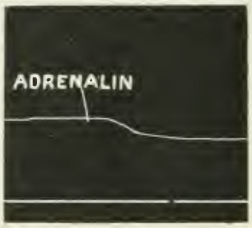


Fig. 3.

Ring preparation of coronary artery of sheep, showing effect of adrenalin. Time: 1 cm. in 42 sec.

that only those muscles are stimulated which are furnished with sympathetic nerves, and that their reaction to adrenalin is similar to that produced by stimulation of these nerves.

Thirty-four experiments with this stimulant gave the following results: A well-marked constriction occurred in the case of the carotid, facial, auriculo-temporal, anterior tibial, popliteal, intercostal, gastric, hepatic, splenic, and renal arteries. In the case of the pulmonary artery, whilst a certain part of the vessel outside the lung was constricted, the intravisceral portion gave no reaction at all. Dilatation and not constriction was produced in the case of the coronary and cerebral vessels (fig. 3). A very short latent period elapsed before the constriction commenced, the constriction was abrupt in onset, the writing point rising at an angle often approaching 90° with the horizontal. The height of the reaction was soon attained, and the vessels remained for a long time in a condition of increased tonus, in fact, it was but rarely that any sign of return to the normal tonus was

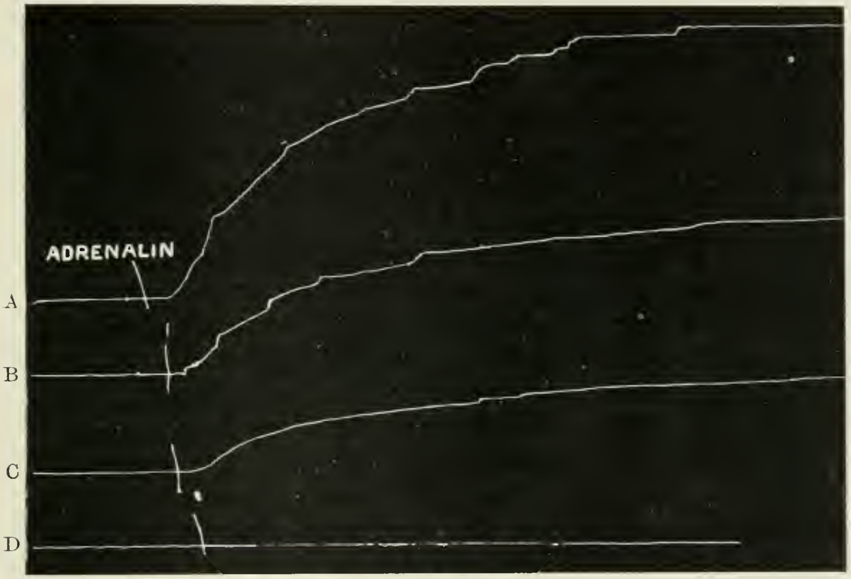


FIG. 4.

Ring preparations of pulmonary artery of rabbit. At the indicated marks adrenalin was applied. A, from main stem; B, from primary branch; C, from branch in root of lung; D, from branch within lung. Time: 1 cm. in 42 sec.

observed during a complete revolution of the recording drum, a period of roughly half an hour. In the case of the splanchnic vessels the onset of the reaction was not so abrupt, and the latent period was generally appreciably longer. In the case of the intercostal artery the constriction was but small in amount, otherwise the reaction was similar to that in the other systemic vessels. In the case of the coronary and cerebral vessels the dilatation was in all respects similar in character to the constriction produced in other arteries, that is to say, the reaction was abrupt in onset after a short latent period, the maximum was soon reached, and the vessels preserved their state of diminished tonus for a considerable time.

A secondary series of experiments was carried out on the pulmonary artery, giving the following results: A ring cut from the main stem produced a decided constriction, a ring from the primary branch showed a less marked effect, a ring from the vessel at the root of the lung showed a very much less marked constriction, whilst a ring from any part of the artery within the lung produced no reaction at all. Again, when a series of rings was taken from 1 in. of the vessel, half of which was in the root of the lung and half within the lung, it was found that the first ring produced distinct constriction; the second, from the very edge of the lung, a less distinct reaction; the third, from a point just within the lung substance, a reaction which was hardly appreciable; and the fourth from a point $\frac{1}{4}$ in. inside the lung, no reaction at all. These results are expressed diagrammatically in fig. 5.

The results of these experiments agree in the main with the results of previous observers, except in the case of the coronary artery. Whereas Schäfer concluded that the increase of flow, which he noted as taking place in the coronary vessels, when the heart was perfused with adrenalin, was due to increased activity of the heart-muscle, my experiments show that adrenalin produces an active dilatation of the coronary arteries.

The following table shows the degree of reaction of different arteries to adrenalin:—

Splanchnic arteries	Very marked constriction
Carotid and its branches...	Marked constriction
Arteries of lower limb	Moderate constriction
Pulmonary (extravisceral)	Moderate constriction
Intercostal	Slight constriction
Pulmonary (intravisceral)	No reaction
Cerebral	Slight dilatation
Coronary	Moderate dilatation

It may be argued, then, that, although there are no vaso-constrictor nerve-endings in the coronary arteries, they do contain vaso-dilator nerve-endings; and the same remark applies to the cerebral vessels. And again, that whereas the pulmonary artery is innervated in that part which stands outside the lung, the intravisceral portion is devoid of vasomotor innervation.

BARIUM CHLORIDE.

This reagent was used as a typical muscle stimulant. Twenty-five experiments were performed. The arteries in all cases reacted by a constriction, which was most marked in the splanchnic vessels, and of smallest degree in the cerebral and intercostal,—vessels which are known to contain very little muscle; the carotid and its branches, the pulmonary, popliteal, anterior tibial, and coronary vessels took a place intermediate between the splanchnics on the one hand and the cerebral and intercostal on the other.

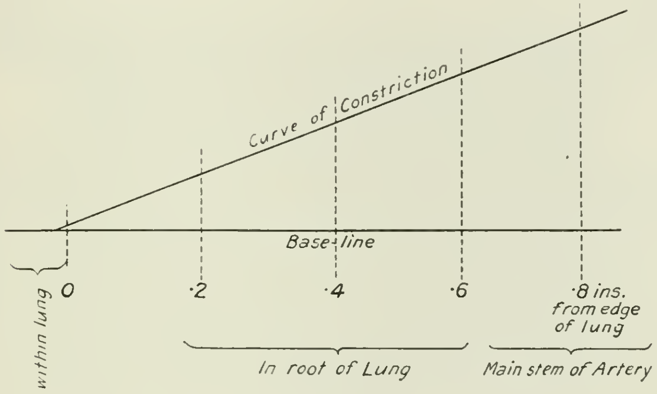


FIG. 5.

Diagram showing effect of adrenalin on pulmonary artery. Vertical height from base-line to curve of constriction represents degree of constriction.

A distinct difference was noticeable in the character of the constriction produced by barium chloride and that produced by adrenalin (fig. 6). In the latter case the latent period was short, the onset abrupt, the maximum soon reached, and the condition of increased tonus long maintained; whilst in the case of barium the latent period was generally longer, the onset of constriction far less abrupt, the maximum not reached so soon, and when reached the condition of increased tonus was not so well maintained.

One may conclude that all vessels that contain an appreciable amount of muscle tissue are constricted by barium, and that its effect is roughly proportional to the amount of muscle tissue present.

DIGITALIS.

I performed twelve experiments with this drug as the stimulant. In order to eliminate any possible errors due to the presence of alcohol, a watery preparation[1] was chosen in preference to the tincture. It was found that different arteries reacted differently to this drug; thus the carotid, facial, auriculo-temporal, popliteal, and anterior tibial vessels were constricted; the gastric and hepatic arteries were first constricted and then dilated, and the coronary artery was dilated. The reaction in all cases was but small in amount. Seeing that the surface vessels and arteries of the limbs are constricted, and that the coronary artery is dilated by this drug, the presumption is that its action is upon the nerve-endings. Since the action on the vessels at the best is but slight and out of proportion to the increase in blood-pressure produced in the intact animal, it would appear that the most important factor in its action must be either its central action or its action on the heart.

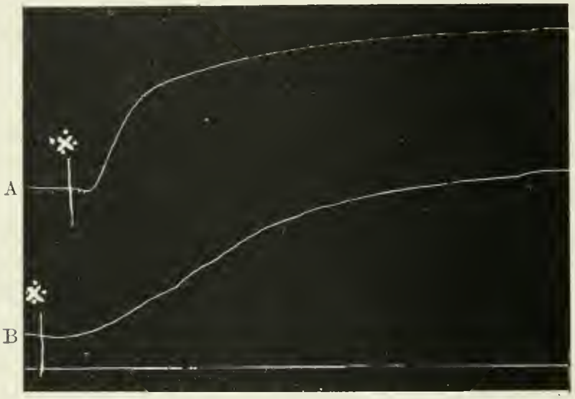


FIG. 6.

Ring preparations of carotid arteries of sheep, showing the relative effects of A, adrenalin, and B, barium. Time: 1 cm. in 42 sec.

---

[1] Digitalone (Parke, Davis and Co.).

PITUITRIN.

Schäfer and Vincent [17] showed that watery extracts of pituitary body constrict the systemic arterioles. Schäfer and Magnus [15] showed that a watery extract of the infundibular portion of the pituitary body increases the kidney volume and produces diuresis. Herring [8] concluded that pituitary extract acts by stimulating vasomotor nerves. Pal [12], also working by Meyer's method, showed that pituitary extract

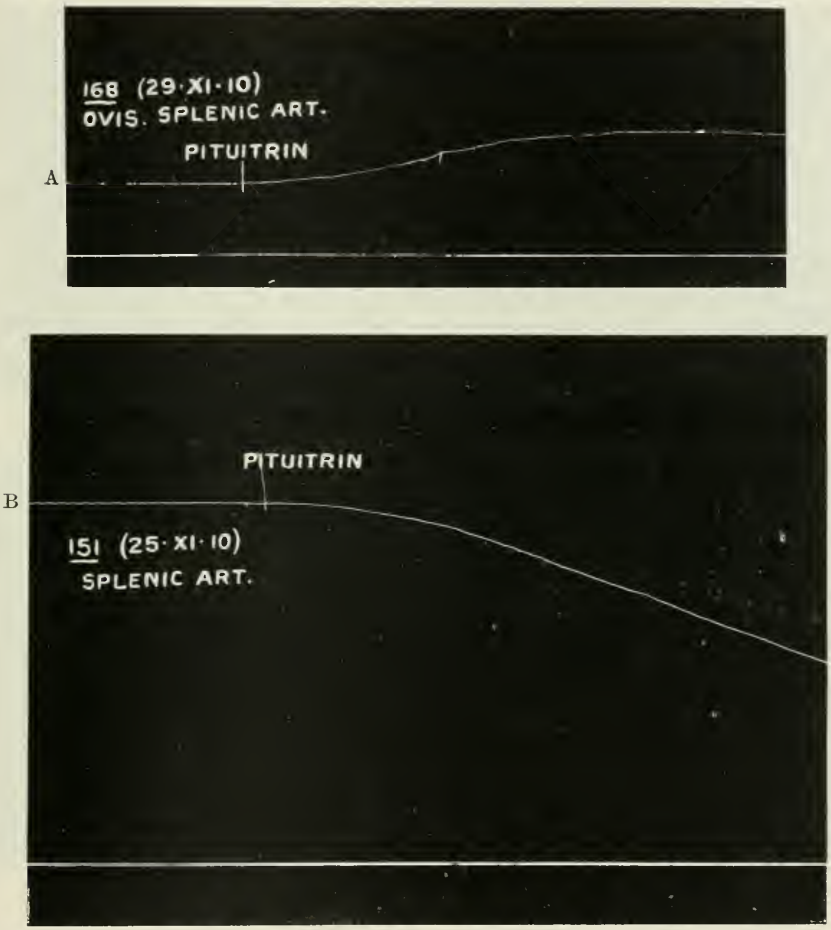


Fig. 7.

Ring preparations of splenic artery, showing effect of pituitrin. In A the reaction is by constriction, in B by dilatation. Time: 1 cm. in 42 sec.

acts on the carotid, mesenteric, and femoral arteries in the same way as adrenalin, but that on the coronary and the peripheral end of the renal artery its action is opposed to that of adrenalin.

Thirty-six experiments were performed—different arteries giving different results. The carotid and its branches were constricted; with the intercostal, cerebral, and pulmonary arteries no appreciable effect was produced; the coronary artery gave varying results, sometimes constriction and sometimes dilatation; the gastric and renal arteries were dilated, whilst it at first appeared that the splenic and hepatic vessels might be either constricted or dilated (figs. 7 and 8). As such

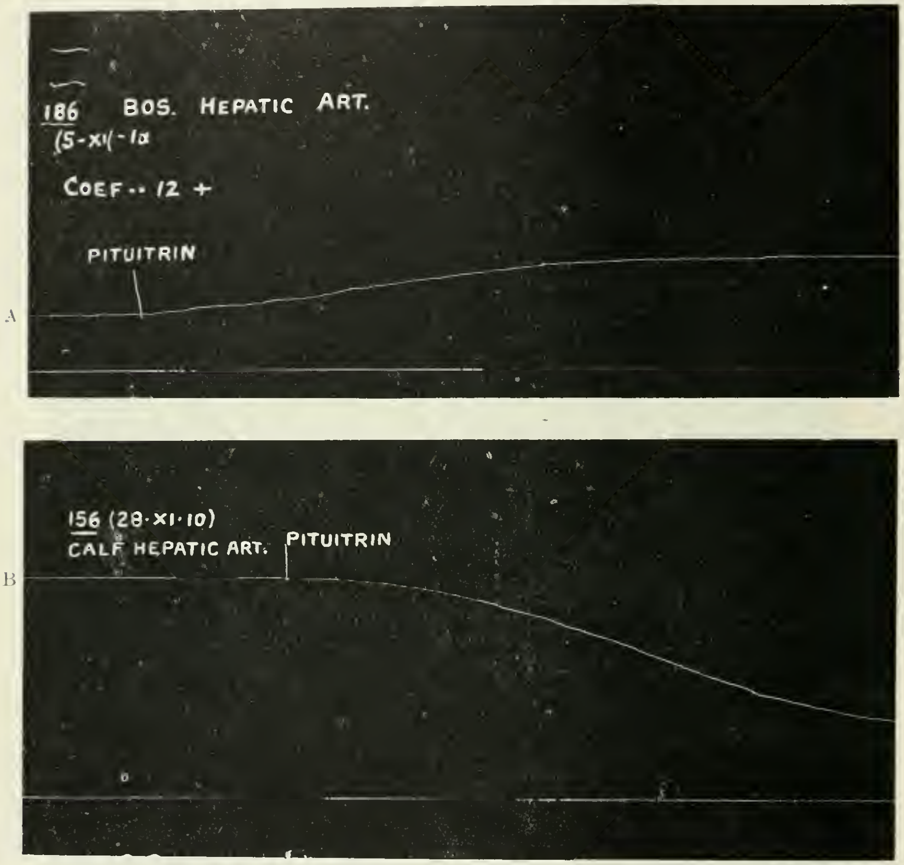


FIG. 8.

Ring preparations of hepatic artery, showing effect of pituitrin. In A the reaction is by constriction, in B by dilatation. Time: 1 cm. in 42 sec.

unequal results were obtained, I performed an extended series of experiments on the splanchnic and renal vessels. I first tried the effect of using different balancing weights on the recording lever in different experiments, weights of 1 grm., 2 grm., and 4 grm. being tried. The result was in all cases constriction. I then tried different doses of the stimulant, instilling doses varying from 2 ♏ to 15 ♏. Here again, however, constriction occurred in every case. I next tried the experiment of taking rings from different portions of these arteries and comparing the results. Here lay the explanation of the previous contradictory results (fig. 9). I found that rings taken from the hepatic, splenic, and gastric arteries in the neighbourhood of their origin from the cœliac axis reacted either by constriction or by a scarcely perceptible dilatation; that rings taken from the visceral ends of these vessels reacted by a pronounced dilatation, and that rings taken from these

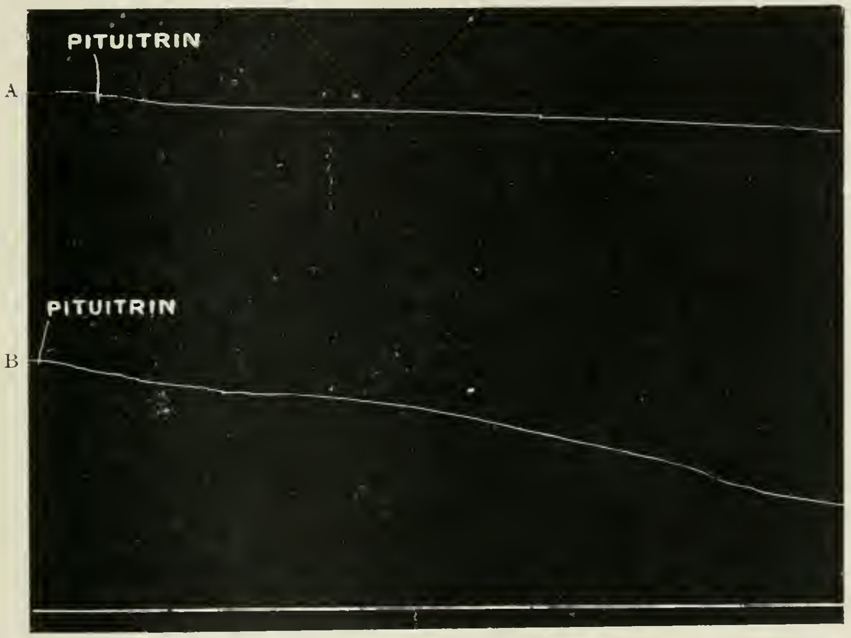


Fig. 9.

Ring preparations of splenic artery of sheep, showing the effect of pituitrin at the indicated marks. A, a ring from near the cœliac axis; B, a ring from near the spleen. The coefficients of these reactions are 10 and 33 respectively. Time: 1 cm. in 42 sec.

vessels from points between their proximal and distal ends reacted by dilatation, which dilatation became progressively more marked the nearer the point from which the ring was cut approached the viscus supplied by the artery in question. Similarly in the case of the renal artery, whilst I was never able to demonstrate a constricting reaction, the dilatation produced became progressively more marked the nearer the point from which the ring was taken approached the kidney. Fig. 10 shows this reaction in graphic form.

The reaction of the vessels to pituitrin, whether by constriction or dilatation, was never so abrupt in onset as that due to adrenalin. Whilst with adrenalin the writing point frequently described a tracing whose direction approached an angle of 90° with the horizontal, in the case of pituitrin an angle of 45° was rarely exceeded. The degree of dilatation produced by pituitrin was considerably greater than the degree of constriction produced, which latter never approached that produced by adrenalin. The condition of increased tonus produced by pituitrin was not well maintained; the height of the reaction was not reached for a considerable time after the reaction had commenced, and when reached the tonus soon started to relax. In no case was the duration of increased tonus comparable to that produced by adrenalin.

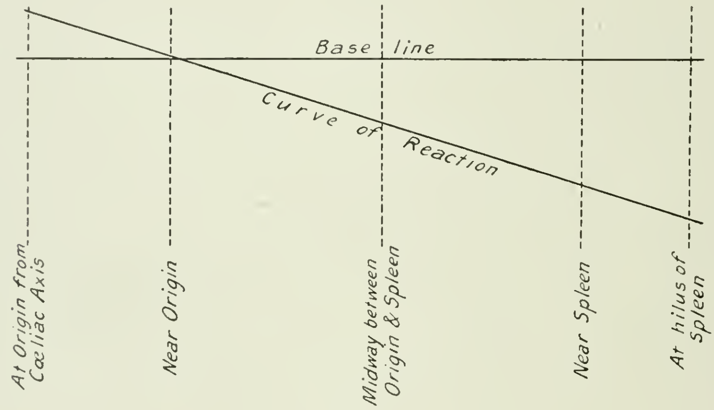


FIG. 10.

Diagram showing effect of pituitrin on splanchnic arteries. Degree of constriction and dilatation shown by vertical distances above and below base-line respectively.

The results of these experiments in the main confirm the conclusions of previous observers. The splanchnic vessels appear to have a peculiar vasomotor mechanism by means of which they can at one part constrict and at another part dilate at the same time to the same stimulant. Since pituitary extract produces a general increase in blood-pressure in the intact animal, it follows that, *cæteris paribus*, the constricting effect on the systemic vessels generally must outweigh the dilating effect on the splanchnic vessels—a theory which my experiments do not suggest; or else we must believe that pituitary extract either has a central action, which may tend to hold in check the local dilating effect on the splanchnic vessels, or possesses both a dilating element and a constricting element, of which either the one or the other may predominate on different occasions.

ERGOT, ERGOTOXIN, TYRAMINE AND ISOAMYLAMINE.

Dale and Dixon [5] and others have shown that certain amine bodies, produced by putrefactive action, have an action similar to that of adrenalin; it has also been shown that certain of these bodies are present in watery extracts of ergot, and that they are responsible for almost the whole of the pressor action of the liquid extract.

I performed seventeen experiments with these drugs. All the arteries used, with but one exception, the pulmonary, were definitely constricted. In the case of the pulmonary artery no effect was produced. The reaction of ergotoxin was of considerably longer duration than that of either the liquid extract of ergot, tyramine or isoamylamine. The amount of constriction produced was at the best small. It may be concluded from the results of these experiments that since the intra-visceral pulmonary artery, which is devoid of vaso-constrictor nerve-endings, is the only artery unaffected by these drugs, their effect is produced by the stimulation of nerve-endings.

SODIUM NITRITE AND AMYL NITRITE.

Brunton [3], in 1870, concluded that the lowering of blood-pressure due to amyl nitrite was caused, not by weakening of the heart's action, but by dilatation of the blood-vessels, and that this dilatation was due to the direct action of the drug upon the vessel walls.

Twelve experiments were performed. The vessels reacted in all cases by dilatation. The dilatation produced by sodium nitrite was, generally speaking, more marked than that produced by amyl nitrite; the latter drug, however, in the apparatus used could not be brought to act on the arteries in the same way as the former, owing to the difficulty of solution. The dilatation produced was in most cases abrupt in character, the maximum was soon reached, and the vessels remained dilated for a considerable time. The fact that dilatation was produced in all cases on the coronary and pulmonary, as well as on the systemic vessels, suggests that the action is on the muscle tissue and not on nerve-endings.

CALCIUM CHLORIDE.

Hans Meyer [11] showed that the administration of calcium salts inhibits inflammatory exudation, and that withdrawal of the normal calcium salts of the body fluids produces a hyper-irritability of the sympathetic system towards adrenalin and other drugs.

Eight experiments were performed with the drug. It was found that, when the arterial ring was placed in saline solution, free from calcium salts, adrenalin and pituitrin produced a greater reaction than when they acted on similar arterial rings in saline solution containing an excess of calcium chloride (fig. 11). The reactions due to barium chloride showed no appreciable difference, no matter in what proportion calcium salts were present.

SPARTEINE.

This alkaloid has been recommended as a cardiac and muscular tonic, on the supposition that it exerted a digitalis-like action on the heart and blood-vessels [4] It has been lately pointed out, however, that this is not the case, but that sparteine is a depressant; that it lowers the blood-pressure by dilatation of the peripheral vessels.

I performed five experiments, using the carotid, gastric, and anterior tibial vessels. A decided dilatation occurred in every case.

CAFFEINE, UREA, AND SODIUM SULPHATE.

A series of nine experiments was performed with these drugs—each of which is known to have a marked diuretic action—comparing their action on the renal artery with that on other vessels. The results were as follows: Caffeine, whilst moderately dilating the renal artery, produced an equal effect on the splenic artery, and had no appreciable effect on the carotid. Urea produced more dilatation of the splenic than of the renal artery, the carotid artery showing no reaction to the drug. Sodium sulphate produced a scarcely appreciable dilatation of the carotid, a more marked dilatation of the splenic, and a still more marked dilatation of the renal artery. Whilst these drugs appeared to have a more powerful dilating effect on the splanchnic than on the carotid artery, no significant difference could be determined between their action on the renal and other splanchnic vessels.

In order to bring out more clearly the comparative effects of the more powerful vasomotor stimulants on different arteries, I have worked out a series of coefficients which roughly represent the degree of constriction or dilatation produced by the different reagents on different arteries. The formula employed is as follows:—

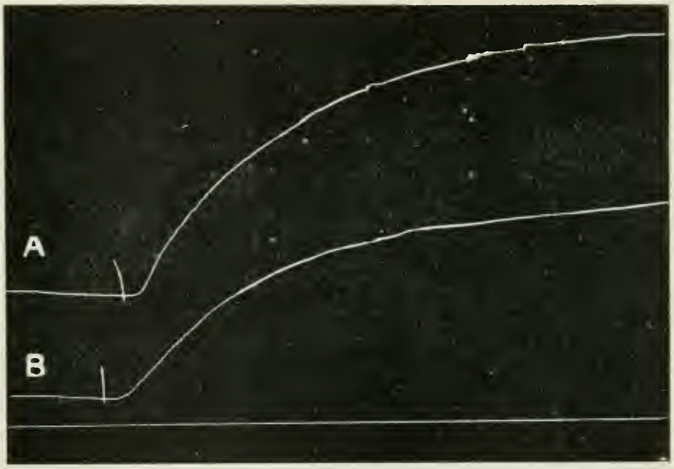


FIG. 11.

Ring preparations of splenic artery of sheep, showing the effect of adrenalin in A, calcium-free Ringer's solution; in B, Ringer's solution containing an excess of calcium chloride. Time: 1 cm. in 42 sec.

$$\text{Coefficient} = \frac{x \times 100 \times 6}{y \times 145}$$

When x = the difference in height of tracing above base-line before and after the application of the stimulant; y = the long diameter of the flattened artery (expressed in millimetres), the lengths of the short and long arms of the writing lever being 6 and 145 mm. respectively.

The following tables show, firstly, the degree of reaction of various arteries to adrenalin, barium, and pituitrin; and, secondly, the graduated response of the pulmonary and splanchnic vessels to adrenalin and pituitrin:—

TABLE I.

Artery	Adrenalin	Barium	Pituitrin
Carotid	+ 27	+ 17	+ 1
Facial	+ 43	+ 33	+ 5
Auriculo-temporal	+ 10	+ 4	—
Intercostal	+ 8	—	—
Anterior tibial	+ 16	+ 5	—
Pulmonary	+ 40	+ 33	—
Coronary	− 9	+ 1	± 4
Cerebral	− 3	+ 2	—
Gastric	+ 68	+ 25	− 40
Hepatic	+ 38	+ 29	− 46
Splenic	+ 47	+ 33	− 54
Renal	+ 43	+ 11	− 32

TABLE II.

Pulmonary Artery with Adrenalin.

Main stem	Primary branch outside lung	Primary branch in root of lung	Branch within lung
+ 42	+ 35	+ 18	0

Gastric Artery with Pituitrin.

Near origin	Midway along course	Near pylorus
+ 12	− 33	− 47

Splenic Artery with Pituitrin.

Near origin	Midway along course	Near spleen
+ 6	− 18	− 33

Hepatic Artery with Pituitrin.

Near origin	Midway along course	Near liver
+ 12	− 17	− 18

Renal Artery with Pituitrin.

Near origin	Near kidney	Within kidney
− 13	− 30	− 30

In a certain number of my experiments a curious phenomenon was evident, though of physiological rather than pharmacological interest. The arterial rings, instead of recording their reactions to stimulants by a tracing line of more or less uniform direction, described a tracing which was broken up into a series of rhythmical units, though the main direction of such a tracing was upward in the case of a constricting reaction.

On analysing one of these tracings it is found that the average duration of one of these rhythmical units is 25 sec. The average duration of a Traube-Hering curve corresponds to eight respiratory curves, or roughly 28 sec. These rhythmical units are rather suggestive of a peripheral origin of the Traube-Hering curve, instead of the previously accepted central theory. At all events, it is evident that under certain conditions the arteries have the power of producing intrinsic rhythmical contractions.

SUMMARY.

From these results it would appear that certain arteries possess certain peculiarities in their reactions to different stimulants. Briefly summarized: The coronary, and to a smaller extent the cerebral, arteries are dilated by adrenalin, whereas this drug produces constriction in all the other arteries. The pulmonary artery may be divided into two portions: the extravisceral portion, which is constricted by adrenalin, and the intravisceral portion, which is either not affected by adrenalin, as my experiments show, or is actually dilated, as shown by Brodie and Dixon. Furthermore, the pulmonary reacts to adrenalin in a progressively diminishing degree, the nearer the part of the artery in

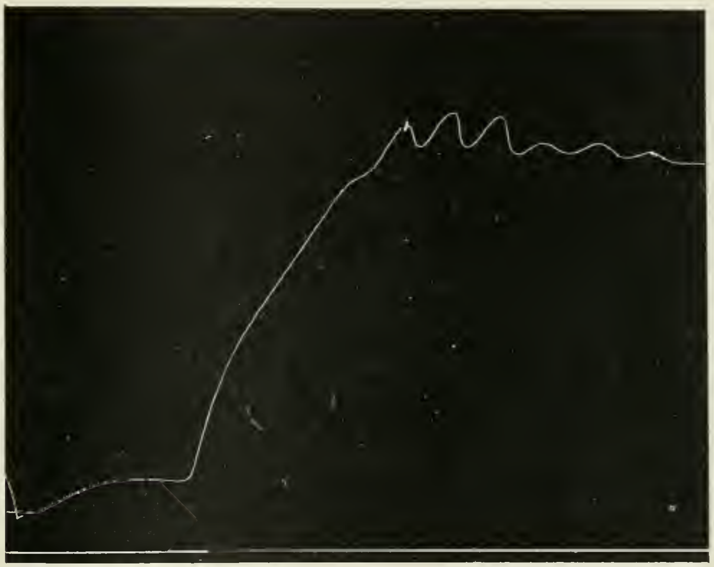


FIG. 12.

Ring preparation of gastric artery of sheep, showing the effect of adrenalin in producing rhythmical contractions. Time: 1 cm. in 42 sec.

question approaches the lung. The gastric, splenic and hepatic arteries are, generally speaking, dilated by pituitrin; this dilatation decreases progressively from the viscus to the origin of these vessels from the cœliac axis, and may even give place to a constriction in the neighbourhood of the origin of these vessels. The renal artery is dilated by pituitrin, and this dilatation increases progressively in the same vessel from its origin from the aorta to its branches within the kidney.

It gives me much pleasure to record my thanks to Professor W. E. Dixon for his kind help and advice.

REFERENCES.

[1] BARCROFT and DIXON. *Journ. of Phys.*, 1906-7, xxxv, p. 182.
[2] BRODIE and DIXON. *Ibid.*, 1904, xxx, p. 476.
[3] BRUNTON. *Journ. of Anat. and Physiol.*, 1871, v, p. 92.
[4] CUSHNY and MATTHEWS. *Arch. f. exp. Path. u. Pharm.*, Leipz., 1895, xxxv, p. 129.
[5] DALE and DIXON. *Journ. of Phys.*, 1909-10, xxxix, p. 25.
[6] DIXON. *Ibid.*, 1907, xxxv, p. 346.
[7] DIXON and HALLIBURTON. *Quart. Journ. of Exp. Phys.*, 1910, iii, p. 4.
[8] HERRING. *Journ. of Phys.*, 1904, xxxi, p. 429.
[9] KOBERT. *Arch. f. exp. Path. u. Pharm.*, Leipz., 1886, xxii, p. 90.
[10] LANGLEY. *Journ. of Phys.*, 1901-2, xxvii, p. 237.
[11] MEYER, HANS. *Brit. Med. Journ.*, 1910, ii, p. 1594.
[12] PAL. *Wien. klin. Wochenschr.*, 1908, xxi, p. 1793.
[13] PLUMIER. *Journ. de Phys. et de Path.*, Par., 1904, vi, p. 665.
[14] SCHÄFER. *Arch. biol. nauk.*, St. Petersb., 1904, xi (Suppl.), p. 251.
[15] SCHÄFER and MAGNUS. *Journ. of Phys.*, 1901-2, xxvii (*Proc. Physiol. Soc.*, July 20, 1901, p. ix).
[16] SCHÄFER and OLIVER. *Journ. of Phys.*, 1895, xviii, p. 230.
[17] SCHÄFER and VINCENT. *Ibid.*, 1899-1900, xxv, p. 87.
[18] WIGGERS. *Journ. Pharm. and Exp. Therap.*, 1909, i, p. 341.
[19] *Idem.* *Amer. Journ. of Phys.*, Bost., 1907-8, xx, p. 206; 1905, xiv, p. 452; 1909, xxiv, p. 391.

DISCUSSION.

The CHAIRMAN (Dr. J. Gray Duncanson) remarked that the paper just read had many points of interest. For discussion he would only refer to one of these. Seven years ago he (Dr. Duncanson) showed that it was, from a clinical point of view, not only erroneous, but even dangerous, to administer suprarenal preparations in cases of hæmoptysis. Dixon had already shown experimentally that transfusion of the lung with adrenalin solution caused dilatation of the pulmonary vessels. Now came Dr. Cow's research, proving that the pulmonary artery did not contract when adrenalin solution was brought into contact with a portion taken from near the lung.

Dr. H. H. DALE said he had been much interested in the paper, and particularly in the results from adrenalin, which fell into line very much with the earlier observations on its action. It was some years ago since it was shown by Langendorff that isolated rings of pulmonary artery responded to adrenalin by constriction, whereas a ring of the coronary artery responded by dilatation. That observation Dr. Cow had now confirmed. In the case of the pulmonary artery there was a superficial discrepancy between Langendorff's observation and that of Brodie and Dixon, who showed that when the lung was perfused and adrenalin added to the perfused fluid the venous outflow, instead of being reduced largely, as in the case of most other organs, was slightly increased. In a paper on another subject a year or two ago he was rash enough to suggest that possibly the discrepancy was only an apparent one, and that if the matter could be examined more fully it would be found that both observations were true, and that adrenalin did constrict the main pulmonary artery and its large branches, as he himself had confirmed on repeating Langendorff's observation, but that the action stopped short at the large branches, the small peripheral branches in the lung being either unaffected or dilated. It was gratifying to hear that Dr. Cow had successfully cleared away any remnant of the apparent discrepancy, and had shown that the apparent difference between Brodie and Dixon's and Langendorff's observation depended on the difference of the reaction of the different parts of the pulmonary artery. Whether or not that was connected with a difference in the innervation—whether the sympathetic innervation extended only a certain distance along the pulmonary artery from its root—there was no direct evidence to show. He took it that that was Dr. Cow's suggestion, and it was consistent with the known facts. With regard to digitalis, he was interested in the statement that it caused dilatation of an isolated slip of the coronary artery, because when perfusing the isolated heart, and measuring the rate of outflow from the coronary vein by collecting the fluid as it dropped from the heart, one noticed, on adding a drug of the digitalis series, that the effect, apart from that on the heart muscle, was to decrease the rate of the coronary outflow. It might be that that was not really due to the direct effect on the coronary artery, but to the increased tonus of the intrinsic muscle of the heart caused by the action of the digitalis drug. He was a little more puzzled to bring Dr. Cow's observation on the effect of pituitary extract into line with what was known about its action before. Pituitary extract caused a very large and rapid rise of blood-pressure, and experiments showed that this was accompanied by a diminution in volume of the abdominal viscera, with the exception, mentioned by Schäfer and Herring, of the kidney. Yet, if he understood Dr. Cow correctly, the large arteries in the abdomen, those supplying the spleen, stomach, and intestine, actually dilated when treated as isolated organs and submitted to the action of pituitary extract. He could only suggest that the action on the main arteries could not be a true index to the reaction of the arteries in the interior of those organs. In the case of the kidney itself, although it was true that the kidney in situ dilated when an intravenous injection of pituitary extract was given, yet the rate of outflow from the renal vein was always markedly diminished by the pituitary extract when the isolated kidney was perfused. In regard to the coronary artery the evidence was clearer than in the case of digitalis, because pituitary extract had very little direct effect on the cardiac muscle itself. There was a scarcely perceptible increase in the tonus of the heart muscle, the rate became slightly slower, the beat became slightly larger; but the most marked effect of adding pituitary extract to the perfusion fluid going through the isolated heart was that the outflow from the coronary vein almost stopped for the time being, the flow being gradually resumed, and slowly recovering its original rate. So when Dr. Cow found that large branches of the coronary artery were dilated, when treated as isolated organs, by pituitary extract, it must be concluded that the reaction of the large artery was not a true index of the action of the drug on the small branches, which was the most important part of its effect; because, as Dr. Cow showed in the case of the lungs, the main artery might be constricted by adrenalin, and yet the constriction might have no effect on the rate of flow through the organ itself, the latter being almost entirely dependent on the reaction of the smaller arteries inside the body of the organ. With regard to ergot, he suggested to Dr. Cow that he should investigate another of the amines known to occur in ergot, and which on certain kinds of plain muscle, such as the muscle of the uterus and the spleen, had an extraordinarily potent stimulant action. He would be glad to indicate what he suggested, and to supply some of the substance. With regard to modification of the effect of adrenalin by calcium, he did not know whether Dr. Cow had seen a paper by Chiari and Fröhlich in a recent number of *Schmiedeberg's Archiv*[1] on the modification of its effect produced by injecting oxalates. He believed their observations had a somewhat similar interpretation to the effects Dr. Cow had described. He felt great interest in the facts which Dr. Cow had brought forward, and he felt sure that some of the reactions which at present seemed anomalous would eventually find their explanation, and add greatly to the understanding of the action of various vasomotor drugs.

Dr. Cow, in reply, said he did not presume to say that his experiments afforded proof that adrenalin acted on the nerve-ending. It was merely a suggestion which fitted the case better than anything else he could think of. With regard to the remark about pituitary extract dilating the large vessels to the spleen, liver, &c., and that the effect on the branches inside those organs might be different, he did not perform any experiments on rings taken from inside those viscera, except in the case of the kidney. It was the same effect in regard to the coefficient; it was still dilated. But in the case of the spleen and liver he neglected to do these experiments. On the coronary pituitrin might produce either dilation or constriction. He thanked Dr. Dale very much for his suggestion, and for his offer to supply him with the new amine, an offer he would avail himself of if he had the opportunity of continuing the work.

---

[1] *Arch. f. exp. Path. u. Pharm.*, Leipz., 1911, lxiv, p. 214.

Therapeutical and Pharmacological Section.

April 11, 1911.

Professor A. R. CUSHNY, F.R.S., President of the Section, in the Chair.

Excitation and Section of the Auriculo-ventricular Bundle.

By W. E. DIXON, M.D., F.R.S.

THE necessity for the presence of some muscular connexion between the auricles and ventricles in the mammalian heart was first maintained by Gaskell [9]. The presence of a bundle of muscle connecting the auricles and ventricles was shown by Kent [15], His [11], Retzer [22], and others, but a more complete description of this bundle has been given by Tawara [25], and confirmed in the main by Keith and Flack [14]. According to these workers a network of special muscle-fibres is present in the auricular walls culminating in a nodal point in the interauricular septum. From this point the muscle-fibres pass down through the auriculo-ventricular ring, and divide in the interventricular septum into two, one group for each ventricle, the fibres subsequently become Purkinje's fibres and end in the subendothelial tissue [21]. The bundle contains no nerve-trunks, but it contains ganglion cells and abundant nerve-fibres [27]. Ludwig and his pupils first showed that the isolated ventricle of the mammalian heart can beat with an independent rhythm. Wooldridge [28] found that by looping a cord round the auricles and applying pressure he could separate functionally the auricles from the ventricles, and that on removing the ligature the auricles and ventricles had assumed an independent rhythm. Tigerstedt, and Roy and Adami [23] produced the same effect. Clamping the bundle in living animals as performed by Erlanger and Hirschfelder [5] and von Tabora, results first in a condition described as diminished conduction by the fibres, in which only some impulses are transferred from the auricles to the ventricles; with the application of greater pressure it is said to be possible to induce complete heart-block. Kronecker [16], and his pupils Imchanitzky [12] and Paukul [20], have obtained contrary results: the latter observer believed that he had ligatured the bundle in the rabbit without disturbing the co-ordination between the auricles and ventricles, and conversely that he had interrupted conduction without touching the bundle. Further experiments brought forward by Kronecker and his pupils with the object of proving that this muscle cannot be essentially conductive in character are considered later. Experiments made upon the isolated heart in which the bundle was incised have been recorded by Biggs [1] on the rabbit's heart, and by Cohn and Trendelenberg [2] on the rabbit, cat and dog.

METHOD.

The experiments[1] were performed on adult rabbits. These were killed by pithing and bleeding, and the excised hearts were perfused with a Ringer-Locke solution. Experimental procedures were not adopted until the heart had become regular, and had continued to beat well for half an hour or upwards. Hooks were then fixed to the apex of the left ventricle and to the right auricular appendix, and connected to levers arranged to write exactly over one another on a smoked surface; an incision was made in the upper part of the right ventricle, and this carried up to the base of the heart. The incisions must be extended if necessary, until the bundle can be defined clearly with the naked eye, the distribution of the bundle varying considerably in different hearts. When the bundle was cut across the incision was made with fine scissors at the upper edge of the muscular part of the septum in a direction running downwards and backwards. It is essential that the bundle be divided as high up as possible, that is, before it divides, and without injuring the cusps of the aortic valve. Sometimes the bundle divides early, and in these it may be extremely difficult to cut efficiently above the bifurcation, because the section must be made high up in the interauricular septum, and this is rendered dangerous by the close proximity of the aorta, which may be accidentally injured. The amount of tissue which must be cut through if the exact spot is chosen is very small, certainly not more than 2 mm. or 3 mm. The bundle of the rabbit is much more difficult to cut completely than that of many higher mammals, dogs, goats, monkeys; the strands of these unite in a common mass instead of dividing prematurely.

---

[1] All the experimental work was undertaken with Miss W. C. Cullis, D.Sc., Lecturer on Physiology to the School of Medicine for Women.

INCOMPLETE SECTION OF THE BUNDLE.

To obtain a typical heart-block it is of the utmost importance that the auriculo-ventricular bundle should be severed before it divides to supply the two ventricles. If the bundle is cut a little low the commonest type of effect is one in which the ventricle stops beating from 10 seconds to 60 seconds. It begins to beat again, at first slowly, but quickly regains speed until a partial recovery takes place, often to a 2 : 1 rate. A third and second cut in about the same position produce a similar type of effect, though generally more pronounced. In these cases of incomplete section auriculo-ventricular arhythmia is not a marked feature, and as the ventricle recovers its speed it generally assumes a sequence as noted—namely, every second, third, or fourth auricular contraction finds a ventricular response. Two facts in these incomplete blocks should be noted: (*a*) the ventricle ultimately assumes its normal rate, and (*b*) except just at first there is no auriculo-ventricular arhythmia. It seems as if the smallest piece of the tissue of the bundle left undivided can carry all the conductions required. As the results of many experiments involving sections of the right and left branches separately, and their ramifications, it has become clear that no part of the bundle can be regarded as supplying any definite part of either ventricle. Even the two divisions of the bundle cannot be distributed entirely to their respective halves, since the most careful and complete section of one branch produces no block of its half. Cohn and Trendelenberg state that in some cases (cats and dogs), they were able to sever the right branch of the bundle, leaving the left intact, when they saw the auricular contraction pass to the right ventricle through the intermediary of the left ventricle.

The incomplete block might be explained by supposing that each incision causes some vagal stimulation, and that as this excitation disappears the ventricles gradually assume the normal rhythm. Against this view it may be stated that the ventricles only very slowly assume their normal rhythm, it may not be until after a period of an hour. The effect, however, cannot be vagal in origin, since it may be produced at a time when the vagal endings are paralysed by atropine. If the bundle be sectioned so as to produce complete standstill of the ventricle, and a large dose of atropine be then sent through the heart, the effect is in no way influenced, and yet such a dose must have paralysed the vagus, even in the rabbit, since muscarine is now found to have lost its inhibitory action on the auricles. Furthermore, if the heart be perfused continuously with a dilute solution of atropine in Ringer 0·001 per cent., an amount quite sufficient to annul any muscarine action, section of the bundle still produces all the effects which have been described. If the cardiac muscle be depressed by such a drug as potassium chloride or chloral hydrate, partial section of the bundle during the period of depression is almost without effect, a fact which suggests that these drugs act upon this specialized muscle before the ordinary musculature of the heart. If a dose of atropine be given, so large as not only to paralyse the vagi, but to depress cardiac muscle, the same result obtains, that is, during the depression partial section is without effect or rhythm; but as the cardiac systole becomes stronger the effect of partial sections returns, and at a time when the vagi are still paralysed. It must be accepted, then, that the action cannot be of vagal origin, and this will be made quite evident after consideration of the effects of complete section of the bundle. It is desirable, however, to draw particular attention to these partial sections, as until one is skilled in cutting at the exact spot they are so easy to produce, and with the smallest shred of bundle remaining intact some conduction occurs and block is incomplete.

If a needle be inserted into an actively beating heart in the position of the auriculo-ventricular bundle before it divides, one of the commonest effects is to obtain temporary cessation of the ventricular beat in precisely the same way and identical with a partial section of the bundle. Recovery is gradual, and generally complete in from two to ten minutes, and the effect may be repeated an indefinite number of times, and can be obtained after large amounts of atropine have been given. Kronecker and Schmey [17] found that puncture of a certain point in the interventricular septum stopped the beat of the ventricle. This fact is urged by Kronecker in support of the neurogenic hypothesis of the heart-beat. The only point in the interventricular septum which causes this effect so far as could be ascertained was the auriculo-ventricular bundle before division. A puncture of this nature must injure some of the muscle-fibres, and it is evident that this fact alone affords no crucial evidence one way or the other as to the origin of the heart-beat.

COMPLETE SECTION OF THE BUNDLE.

When the bundle is properly incised high up before division, so soon as the section is complete the ventricle, after two or three irregular contractions, ceases to beat and remains relaxed and quiescent for a time, varying from three minutes to the end of the experiment two or three hours later. In the latter cases the ventricles respond to mechanical or electrical stimulation, but remain quiescent in the absence of these. In the vast majority of cases, however, after an interval of thirty minutes from the time of section of the auriculo-ventricular bundle, the ventricles have assumed a rhythm of their own, much slower than that of the auricles, but showing no form of sequence with them. The independent ventricular rate is commonly from three to five times slower than the normal heart-beat as shown by contractions of the auricle. In a few cases after such section the ventricular beat quickened to such an extent that its rate became more than half that of the auricles, but in no case was there any form of sequence between the two chambers.

Not infrequently after complete section of the bundle, and when the ventricles had commenced to beat again and assumed an independent rhythm of their own, reversed conduction occurred. Sometimes nearly every contraction of the ventricle was followed by an auricular extra systole, and when the ventricle failed to cause such a systole, the contraction occurred in the auricular refractory period. Biggs stated he had also observed this effect, but Erlanger and Hirschfelder [6] assert that after complete heart-block such conduction never occurs, and Fredericq and Hering [8] state that in heart-block spontaneous extra systoles do not pass to the auricle.

Attempts were made to block conduction by the application of a 5 per cent. solution of cocaine directly to the bundle. This was done by soaking the cocaine on cotton-wool and applying with forceps. No appreciable effect was obtained which could be computed to an action on the bundle, although any nerve-fibrils with which the cocaine came in contact would certainly have been paralysed. Another point upon which some observations were taken was the interval between the beat of the auricle and ventricle (As—Vs interval). Biggs, working on the rabbit, stated that section of the bundle lengthened this period, and Hering [10] states that destruction of the sino-auricular node leads to a diminution of the interval. In these experiments it was found that after an incomplete section of the auriculo-ventricular bundle this interval was always increased, but that recovery was the rule. Thus in one experiment when the ventricular beats were reduced to one-fourth those of the auricular as the result of such a section, it was estimated that the auriculo-ventricular interval occupied about one-fourth of the total cardiac cycle, whilst in the normal rabbit it does not occupy more than one-seventh. After ten minutes the interval had assumed its normal measurement.

From the region of the bundle a fine white line may be seen running outwards on the auricular septum between the superior and inferior vena cava. Section of this line, which is generally easy to see, causes in most cases the auricles to cease beating for a varying time, from about thirty seconds to two minutes, whilst the ventricles continue to beat to their normal rate. Recovery, when once the auricle makes a beat, is very rapid, and arhythmia is never present at any period, that is to say, each recorded auricular beat occupies its proper position in the cardiac cycle. The effect looks very like an inhibition, and microscopical section of the white line shows that it is composed largely of large cells, probably nerve.

The sino-auricular node has been regarded as a centre dominating the rhythm of the heart [7]; if such be the case, it is difficult to see why its destruction does not decidedly influence the rhythm [13]. Lewis and his colleagues [15] explain this by the suggestion that "if the original pacemaker is destroyed it is probable that another portion of the auricle assumes this function without an appreciable change in rate or rhythm." These observers show that the point of primary negativity on the superficies of the right auricle lies superficial to the sino-auricular node. Erlanger [4] from experiments on the cat's isolated auricular strip has shown that rhythmicity is not dependent upon the presence of nodal tissue. In view of these facts it is unnecessary to assume that the sino-auricular node exerts any effect on rhythm other than can be induced by the nerves, vagus and sympathetic, which enter the heart in the vicinity.

The ventricular beat *may be* quite independent of the main auricular contraction, since after section of the white line the ventricular beat is unchanged, whilst the auricular ceases so far as can be ascertained by naked-eye observation; and the auricular beat may be quite independent of the ventricular, since section of the auriculo-ventricular bundle leaves the auricular beat unchanged, whilst the ventricle may cease to beat. The one and only point in our experiments in which the chambers of the heart seem to be correlated is in the auriculo-ventricular node.

It is clear, then, that section of the auriculo-ventricular bundle results in heart-block, and my view is that this is not so much because the auricular peristaltic contractions are unable to reach the ventricle, as because the ventricle is separated from that portion of the interauricular septum in which lies its centre of rhythm.

THE EFFECT OF STIMULATION OF THE BUNDLE.

No observations apart from those of Miss Cullis and myself have been recorded as to the effect of stimulating the bundle of His. The present experiments were made with electrical, chemical, and thermal stimulants. For the electrical experiments electrodes were made of two fine (No. 20) entomology pins; these were passed through a small cube of cork and made firm with wax and elastic glue. They were so small and light that they could easily be placed in any portion of the rabbit's heart and remain fixed, so that it was possible at any moment to pass a tetanizing current through the tissue they enclosed.

If these electrodes be placed in the auriculo-ventricular bundle before division and a faradic bundle be passed through with the secondary coil at 8 cm. or 10 cm., the first and most noticeable feature is marked acceleration of the ventricular heart-beat. If auricle and ventricle are beating synchronously and well, when the current is passed the ventricular rate becomes much increased, relaxation in diastole is hardly apparent, so that the heart is in a condition of tonus and its movements are much too rapid for precise record on smoked paper. The condition closely resembled that of the "delirium cordis" seen in the last stage of digitalis poisoning. The rate of the auricular beat is increased also, but so far as one could judge with the naked eye, the acceleration of the auricle is far behind that of the ventricle. This effect on the auricle is not constant; quite often whilst the ventricle gives the typical result the auricle may be slowed, or may show a series of changes of alternating acceleration and inhibition.

After complete section of the bundle and after a sufficient time has been allowed to elapse (one or two hours) to allow the ventricle to attain its independent rhythm, the same stimulation with the electrodes produces the same effect on the ventricles as before. The effect on the auricular movements when stimulation is applied below the section is usually to cause some slowing. The explanation of these varying effects on the auricular beat is probably furnished by the following series of records from one experiment. In the first place a stimulation of the bundle was effected with the secondary coil at 7 cm. The ventricle entered into a tonic contraction as before and the auricular beats were slowed. The electrodes were left in situ and were not moved throughout this experiment. Atropine was now slowly allowed to pass through the coronary vessels, in all 20 mg. This caused a temporary diminution of force of the ventricular contraction; so soon as this had passed the bundle was again stimulated and the ventricular effect was precisely as before, but acceleration of the auricles also was now a marked feature, suggesting that the slowing noted in the first instance was probably vagal in origin. In every case in which slowing of the auricular movements followed stimulation of the bundle, the administration of atropine converted this into acceleration. The bundle was now cut through above the electrodes but without interfering with them. About half an hour was allowed to elapse for the ventricle to attain its own rhythm and stimulation was again resorted to. The ventricular effect remains typical whilst the auricular beat is not very decidedly affected. It seems, then, that stimulation of the bundle with a weak current causes acceleration of both the auricles and ventricles. As the current is strengthened the diastolic pause of the ventricle becomes less and less and the heart remains in a state of tonic contraction. The effect on the auricle is less marked, and is sometimes masked by vagal action.

The objection to all such forms of stimuli is the leakage of current which must occur into the surrounding tissues; but although by this means of stimulation it is impossible to avoid such leakage, it can yet be shown that stimulation of other parts of the heart, with the exception of the interventricular septum, is without action of this kind. Thus if the electrodes be placed on the outer wall of the heart or on a portion of the cut ventricles the same strength of current has very little effect on the heart-beat, but if the electrode be placed anywhere along the known distribution of the bundle, as in the ventricular septum, an effect is obtained of the same nature as that occurring when the bundle is excited before its division, except that it is less pronounced. Roughly speaking, the further the electrodes are moved from the origin of the bundle the smaller the result obtained. I believe that these effects are true bundle excitations, because they only occur with intensity when the bundle is stimulated before its division, and because as the electrodes are moved downwards on the septum, that is, as the bundle ramifies on the septum, the effect becomes less and less pronounced. Where the bundle does not exist electrical stimulation is without this action. It is obviously necessary, however, to obtain a more suitable form of stimulus.

Heat yielded very unsatisfactory results, but chemical stimuli accurately applied to one spot afforded complete verification of the results obtained by the electrical method. Of these chemical substances a hard crystal of rock salt of a suitable size gave the best effects. In one experiment, commenced an hour after section of the bundle and with the ventricle beating only about one-eighth the rate of the auricle, a crystal of common salt applied to the cut edge of the ventricle produced very little effect. The crystal was then applied to about the centre of the ventricular septum; it caused some distinct acceleration whilst the salt was in apposition, followed as soon as it was removed by inhibition of the ventricular beat. When, however, the salt was applied to the bundle at the point of section, very marked quickening at once followed and the tonus of the heart was increased; the effect in every way corresponded with that already described as caused by the faradic current. Moreover, when the salt was removed the heart continued to beat regularly for a time varying generally from five to ten minutes, before it resumed the slow beat of its pre-stimulation condition. By further applications of salt the effect can be repeated an indefinite number of times. Each period of stimulation of the bundle resulting in acceleration of the heart is followed by a period of inhibition.

Other chemical stimulants besides common salt were used, though for most purposes salt was found the most satisfactory. Most of the soluble ionizable salts of potassium, sodium, lithium, and ammonium were effective stimulants. The acids also acted quite efficiently; calcium salts, on the other hand, had no stimulant action. It was noticed that atropine, after doses so large as to depress and weaken the cardiac muscle, and, of course, to eliminate completely the effect of muscarine, modified the effect of stimulation. Whilst the heart is depressed stimulation of the bundle produces less effect than normally, but as soon as the cardiac systole approaches its normal contraction stimulation by chemicals exerts the usual effects.

Whilst the heart is depressed by drugs, such as chloroform, chloral, or potash salts, as shown by the weakened ventricular systole, the bundle of His loses its characteristic features. As already pointed out, partial section no longer causes partial heart-block, and excitation, by either chemicals or electricity, is without influence on the beat. These facts suggest that such cardiac depressants produce their effects largely upon the tissues of the bundle; in any case this tissue is profoundly affected.

It might be suggested that the effects of the excitation described are not muscular but nervous in origin. As regards nerve-cells this supposition was put to the test by the direct application of a solution of nicotine to the bundle. If the stimulant effects produced by electrical and chemical agencies had their origin in nerve-cells the application of the nicotine should result in the same effect.

If a 1 per cent. solution of nicotine be applied on a piece of cotton-wool to the bundle no effect is to be observed for some time, often for a minute or two. Occasionally, however, there may be some immediate inhibition of both the auricles and ventricles, an effect which suggests excitation of vagal nerve-cells, a supposition which we regard as valid, since such initial inhibition is not obtainable after a suitable administration of atropine. Whether, however, there be initial inhibition or not, after a time (two or three minutes) the heart begins to quicken, and very soon an effect is obtained, both as regards acceleration and increase in force of beat, comparable with that of adrenalin.

This effect can hardly be brought about in the same way as by direct stimulation of the bundle by rock salt, for several reasons. If the bundle contained nerve-cells upon which the nicotine acted, the effect should be immediate, and should slowly cease when the nicotine was withdrawn, as in the case of salt. Nicotine perfused through the isolated heart produces an effect precisely of the same nature as that described when it is applied locally, and this makes it almost certain that the apparent local effect is in reality the specific action after absorption. The fact, however, appears to be clear that the effect of stimulation of the bundle with chemicals is a muscle and not a nerve effect, because the stimulation is only brought about by muscle stimulants, and because the nerve-cell excitants are without effect until after sufficient delay for absorption to occur.

It may be noted here that the effect of perfusing nicotine through the isolated heart has not been recorded. If the heart contains no sympathetic nerve-cells, as is generally supposed, how is the marked acceleration and increased force of the beat to be accounted for? Langley clearly showed that no sympathetic cells were present in the heart directly associated with the accelerator nerves. Yet nicotine produces an action exactly corresponding with such a sympathetic stimulation that is an adrenalin type of action, and this effect is still produced, though to a much smaller degree, after the administration of atropine. Langley and Dickinson [18] note that the second and later applications of nicotine to the frog's heart 'not infrequently cause quickening of the beat. The nature of these cells therefore obviously demands attention. Roy and Adami [24] showed that in certain conditions stimulation of the vago-sympathetic in the neck of dogs caused a marked increase in force of contraction of the auricle and ventricle, sometimes associated with acceleration. Dale and Laidlaw [3] have recently obtained similar results. We believe, then, by a process of elimination that it must be the nerve-cells connected with these fibres upon which the nicotine exerts its specific action. Another possible explanation, which is indeed supported by some facts, is that the action of nicotine is not confined to the nerve-cell, but that it extends some distance down the neurone.

THE EFFECT OF SOME DRUGS.

Adrenalin.—Adrenalin administered to an actively beating heart after complete section of the bundle still causes its typical action. Both the auricles and ventricles beat more rapidly and more vigorously, but both these effects are much more pronounced in the auricles than in the ventricles. The auriculo-ventricular arhythmia is, of course, not influenced.

Some experiments with aconitine were made by painting a solution on the bundle, and they afford some probability that this alkaloid, which it is well known causes death in mammals by sending the heart into delirium, acts directly on the special muscle contained in the bundle. It must be noted that a relatively small amount of any substance in solution when applied on a piece of cotton-wool and held against the auriculo-ventricular bundle is in time absorbed into the coronary circulation and causes the specific effects. We have obtained evidence that several poisons to the heart, such as cocaine, can be applied even in strong solution to the muscle in the manner named without appreciably affecting the beat, whilst if even a trace is injected into the coronary circulation the heart ceases to beat. It seems valid, then, to assume that if the direct application of aconitine to the bundle causes a typical action of the drug relatively rapidly, whilst the similar application to the cut wall of the ventricle is without effect, within the same limits of time, the action is likely to be upon this specialized muscle of the bundle.

Such an application of aconitine to the auriculo-ventricular bundle we have found causes a very marked effect, commencing with acceleration of both auricles and ventricles, followed by complete arhythmia, with often the appearance of reversed conduction. The evidence, however, that this is not the result of absorption is not conclusive.

DISCUSSION OF RESULTS.

The power of rhythmical contraction is an inherent property of all plain muscle, including isolated strips of cardiac muscle and isolated rings of arteries. The intact perfused isolated heart exhibits a rhythm different from the rhythm inherent to isolated strips of cardiac muscle, mainly in that it is much more rapid.

One point from which this augmented rate of rhythm appears to be initiated is somewhere in the region of the auriculo-ventricular node, and the impulses to the ventricle are communicated by the muscle-fibres contained in the auriculo-ventricular bundle. The experiments of Paukul (1909), who believed that he had ligatured the auriculo-ventricular bundle completely without obtaining heart-block, are the only serious objections to the hypothesis that this bundle is the means of conduction between auricles and ventricles. These experiments and those of Cohn and Trendelenberg are at variance with those of Paukul; when the bundle has been completely divided before its bifurcation, complete heart-block has also been observed.

The muscle forming this bundle is very sensitive to stimulation and chemical reagents. This easy stimulation cannot be due to the presence of nerve elements for reasons we have already stated, but, on the contrary, it shows all the characteristics of muscle conduction. Nerve-cell stimulants are without effect upon this tissue. Muscle poisons, on the contrary, very easily influence it. Thus the effects of stimulating the bundle either by electricity or salts are absent when the heart is depressed with chloroform, chloral, or potash salts, drugs which, in the doses concerned, are recognized as affecting muscle only and not nervous structures. It seems clear, then, that conduction from the auricles to the ventricles occurs through the muscular tissue of this bundle.

It is generally accepted that it is the auricular contraction which starts the wave of contraction in mammals, and that this wave meets with a resistance at the auriculo-ventricular groove. These experiments suggest the possibility that both auricles and ventricles may possess a common centre for rhythm, and that the ventricles may exhibit a perfectly normal beat when the auricles are apparently quiescent; this cessation of the auricular beat may be induced either by section of the white line, or in some cases by clamping the right and left auricular appendices (Kronecker). The evidence is not clear whether in these cases auricular standstill is or is not nervous, but whatever the cause the fact remains. Gaskell argues, in the case of cold-blooded animals, that because the auricular beat is not easily seen that does not prove its absence, and that if the auricles be properly examined small beats can be observed. But whether this be so or not in mammals, and we have some reason to believe that during auricular standstill the interauricular septum is contracting, it is clear that the ventricular contraction can be independent of the main auricular contraction. One explanation of the facts is the assumption of a common centre of rhythm. This centre is situated in the auricular septum just above the attachment of the mesial segment of the tricuspid valve, at the region known as the node of Tawara, which is connected with the auricle within the auricular walls by a network of special muscle-fibres spreading out from the node, and with the ventricle by the bundle of His.

The cause of the reversed conduction which may occur after complete section of the bundle suggests that other means of conduction between the auricles and ventricles are available besides those of the auriculo-ventricular bundle. Another explanation may, however, be offered to account for the fact. We have noted that after complete section of the auriculo-ventricular bundle the auricles may exhibit an increased irritability, so that the mere contraction of the ventricle may cause a sufficient mechanical jar to the auricle to induce a contraction. In more than one experiment every ventricular contraction has been followed by a regular auricular contraction, except when the contraction of the ventricle occurred in the auricular refractory period. We have not at present the data to decide upon the cause of this phenomenon.

CONCLUSIONS.

(1) Incomplete section of the bundle of His causes partial heart-block generally without arhythmia and with complete recovery. The effect is quite independent of the vagus.

(2) The smallest portion of bundle tissue left undivided can carry all the conduction necessary for a normal ventricular rhythm. In the rabbit it is not possible to produce block in one ventricle by complete section of one division of the bundle.

(3) Complete section of the bundle causes complete heart-block and after an hour or two in most cases the ventricle assumes its own independent rhythm three or four times slower than that of the auricle.

(4) Kronecker and Schmey's effect is explained by injury to the auriculo-ventricular bundle.

(5) In complete heart-block reversed conduction sometimes occurs.

(6) The As—Vs interval is found to be increased after partial section of the auriculo-ventricular bundle.

(7) The auricles as a whole may be made to cease beating without effecting the ventricular rhythm, suggesting that the ventricular beat is not a simple conduction from the auricular contraction as a whole but that the effective beat is started from a specialized portion of the auricle.

(8) Stimulation of the bundle with electricity or salts causes a tetanus: this effect is marked with the stimulus applied to the bundle, is much less pronounced on the septum and on the course of distribution of the bundle, and is absent on the body of the heart or the cut edge of the ventricle.

(9) Reasons are given for believing (*a*) that conduction by the bundle is myogenic, (*b*) that the auriculo-ventricular node is the one point essential for the production of the ventricular rhythm.

REFERENCES.

[1] Biggs. *Brit. Med. Journ.*, 1908, i, p. 1419.

[2] Cohn and Trendelenberg. *Arch. f. gesammt. Physiol.*, Bonn, 1910, cxxxi, p. 1.

[3] Dale, Laidlaw, and Symonds. *Journ. of Physiol.*, Lond., 1910, xli, p. 1.

[4] Erlanger. *Amer. Journ. Physiol.*, Bost., 1910, xxvii, p. 87.

[5] Erlanger and Hirschfelder. *Amer. Journ. Physiol.*, (*a*) 1905-06, xv, p. 153; (*b*) 1906. xvi, p. 160.

[6] *Idem.*

[7] Flack. *Journ. of Physiol.*, 1910, xli, p. 64.

[8] Fredericq and Hering. "Congrès allemand de Médecine Interne," *Semaine méd.*, Par., 1906, xxvi, p. 223.

[9] Gaskell. *Journ. Physiol.*, 1883, iv, p. 43; *Brit. Med. Journ.* 1882, ii, p. 572.

[10] Hering. *Münch. med. Wochenschr.*, 1909, lvi, p. 845.

[11] His, jun., W. *Arbeiten aus der med. Klin. z. Leipz.*, 1893, p. 1.

[12] Imchanitzky. *Arch. internat. de Phys.*, 1906, iv, p. 1.

[13] Jaeger. *Zentralbl. inn. Med.*, Leipz., 1910, xxxi, p. 545; *Deutsch. Arch. f. klin. Med.*, Leipz., 1910, cl, p. 1.

[14] Keith and Flack. *Journ. Anat. and Phys.*, Lond., 1907, xli, p. 172.

[15] Kent. *Journ. Physiol.*, 1893, xiv, p. 233, and *Proc. Physiol. Soc.*, November, 1892, p. xxiii.

[16] Kronecker. *Brit. Assoc. Report*, 1899, p. 895; *Brit. Med. Journ.*, 1910, i, p. 185.

[17] Kronecker and Schmey. *Sitzungsber. d. Akad. d. Wiss. zu Berlin (Physik-Math. Klasse)*, 1884, xiii, p. 87.

[18] Langley and Dickinson. *Journ. of Physiol.*, 1890, xi, p. 279.

[19] Lewis, B. S., and Adele Oppenheimer. *Heart*, Lond., 1910, ii, p. 147.

[20] Paukul. *Zeitschr. f. Biol.*, Münch. u. Berl., 1909, li, p. 177.

[21] Ranvier. "Leçons d'anat. gen. syst. musculaire," Par., 1850, p. 300.

[22] Retzer. *Arch. f. Anat. u. Phys.* (Anat. Abth.), 1904, p. 1.

[23] Roy and Adami. *Phil. Trans.*, 1892, clxxxiii, B, p. 199.

[24] *Idem. Phil. Trans.*, 1892, clxxxiii, p. 248.

[25] Tawara. "Das Reizleitungssystem das Saugetierherzens," G. Fischer, Jena, 1906.

[26] Tigerstedt. *Arch. f. Anat. u. Physiol.* (Physiol. Abth.), 1884, p. 497.

[27] Wilson. *Proc. Roy. Soc.*, 1909, lxxxi, p. 151.

[28] Wooldridge. *Arch. f. Anat. u. Physiol.* (Physiol. Abth.), 1883, p. 522.

DISCUSSION.

Dr. T. LEWIS said the Section was much indebted to Professor Dixon for his interesting paper. In his paper Dr. Dixon spoke of opening the right ventricle of the rabbit, but showed a diagram of the inside of the left ventricle. So that he (Dr. Lewis) was not quite certain what route had been employed in the investigation of the bundle. [Professor DIXON: The right ventricle was opened.] Dr. Lewis, continuing, referred to section of the bundle and stated that much work had been done on the question; and that the observations on the heart, with the exception of the rabbit's heart, had been very uniform. The conclusion had been that it was the sole path of conduction from auricle to ventricle, and that when it was cut completely, transmission of impulses from the auricle to the ventricle ceased absolutely. Another series of investigations by the Berne school, headed by Kronecker, had been undertaken, and they had been almost confined to the rabbit. They had been contradictory to the general run of others. Kronecker and his pupils (Imchanitzky, Busch, and Paukul) had stated that complete section of the bundle in the rabbit did not produce auriculo-ventricular dissociation—i.e., impulses were still transmitted. Consequently it had become of great importance that the matter should be re-investigated. It was re-investigated by Cohn and Trendelenberg, whose results differed from those of the Berne school, for they found that complete section of the bundle in the rabbit gave rise to the expected results, results with which Professor Dixon's observatious were in accord. The important point was that the results of the Berne school had been criticized, in that, with the exception of Paukul's work, serial sections had not been cut, and that in the instance of Paukul the sections had not been cut in the usual direction; they had not shown that they had cut the bundle completely, but they had been largely guided by the results. The first criticism he would offer of Dr. Dixon's results was therefore that they would have been more valuable if serial sections had been made of the bundles and nodes subsequently to the production of the lesions. The bundle in the rabbit was a very fine structure, which he believed could not have been seen properly with the naked eye: it was sufficiently difficult to follow in the larger mammalia. Dr. Dixon said that if the smallest shred of bundle was left when a section was made in its neighbourhood, conduction was ultimately restored. He (the speaker) felt that Dr. Dixon was not justified in this conclusion, for serial sections of the bundles had not been obtained subsequently to show that only a shred remained. The same criticism applied to his results of stimulation. If one pushed electrodes into the heart at the point where it was believed the bundle or the node was, or placed a crystal of rock salt in the same region, one should subsequently confirm the locality stimulated by cutting sections to see whether the bundle or node was there. With regard to retrograde contraction subsequent to bundle section and the production of dissociation, it was difficult to explain, as Dr. Dixon had said. Biggs stated that he obtained precisely similar results. It was at variance with the observations which had been made on the dog's heart by several observers, and at variance with the observations which Oppenheimer and he had made on the cat's heart. It was found in asphyxial heart-block in this animal that if there was even a small impairment or conduction from above downwards, the impairment of conduction from below upwards was absolute. With regard to the experiment on the white line, he did not feel inclined to make remarks about it without seeing the full protocols of such experiments. With regard to the pacemaker of the heart, Dr. Dixon appeared to regard the auriculo-ventricular node as having some special control of the origin of the heart-beat. He (the speaker) did not think that was a position which could be held to-day, in view of the evidence which had been accumulated. Destruction of the auriculo-ventricular node had no more influence, in fact it had less influence, upon the ultimate rhythm of the auricle than had destruction of the sino-auricular node. That was known from numerous pathological observations, in which it had been shown that the node was destroyed while the auricular rhythm continued in normal fashion. At the sino-auricular node the contraction certainly started, as he believed Dr. Dixon was prepared to admit, and the experiments in which damage had been done to the node had been numerous. There was considerable evidence that the heart-beat did not originate in the ring tissues, or in the neighbourhood of the auriculo-ventricular node. The experiments of Jaeger, which Dr. Dixon referred to, could only be described as clumsy. They consisted of the application of a cautery to the superficies of the auricle, an experiment which he did not think was justified upon a heart; reliable conclusions could not be based upon a heart burnt in that way. The burns were very extensive. Jaeger performed sixty experiments, and there were positive results in only thirteen. Opposed to these results were those of Hering and Lommel; both these workers found that formalin applied to the node abolished the normal rhythm, which then originated in the neighbourhood of the ring tissues. Recently there had been observations from Cohn and Kessel. They used the perfused heart, and had made cuts on three sides on the sino-auricular node, but found no alteration in the rhythm. On cutting on the fourth side, in ten out of the fifteen cases they found there was a standstill of the whole heart. Of the remaining five experiments, in two there was considerable slowing of the heart, and referring to the remaining three, they stated their opinion that the whole of the node was not excised. The full particulars containing their histological examinations were not yet to hand.

Professor DIXON, in reply, said he agreed with much of what Dr. Lewis said. In the dissections of the bundle, it was of course desirable that microscopical examination should be carried out afterwards. That was not done in these cases; all that he was content to do was to examine the bundle with a magnifying glass afterwards, and he believed he was able to satisfy himself as to the portion which had been cut. The same remark applied to the criticism concerning the position of the salt, and he did not règard it as a valid criticism, because the salt could be put to a nicety in that one position, and an effect was produced there, but nowhere else. [Dr. LEWIS: Surely that is arguing in a circle, because you say you get an effect with the salt if you put it on specialized tissue.] Professor Dixon rejoined that it was not a hypothetical situation, but a structure which could be seen. With regard to the auriculo-ventricular node, Dr. Lewis said that if one destroyed that node completely it produced less effect on the rhythm of the auricle than did destruction of the sino-auricular node. Such a destruction had never been performed experimentally; Dr. Lewis argued from pathological lesions entirely, and he (the speaker) considered that to assume complete destruction from a post-mortem observation was open to serious criticism. He hoped that the node would be destroyed experimentally and the effects observed. With regard to the "pacemaker," he was careful to avoid being dogmatic; he only threw out the suggestion as to how the experiments which he had mentioned could be explained on other suppositions. That the auricular beat started in the position roughly of the sino-auricular node, i.e., in the proximity of the great nerves, he admitted, but it was not possible to stimulate the sino-auricular node, i.e., the nodal tissue, without influencing those nerves, and the results obtained were mixed vago-sympathetic effects. If one excised that node, the very process excited the vagus and sympathetic, and there might be a standstill of the heart, or acceleration of it. He had not yet seen the results of Cohn's experiments, but he would be willing to adopt his view if it were based upon sufficient facts. Another point that had been clearly shown by Erlanger was that the auricular rhythm was not dependent on nodal tissue. He did not insist that in a normally beating heart the nerves proceeding from the node did not have a profound effect on the rhythm, but that if there were no sino-auricular node, as after excision, there might still be a normal rhythm of the heart. In the rabbit's heart the only point he found which was essential for rhythm was the auriculo-ventricular node.

He was familiar with the President's classical work on aconitine. He put the last slide on the screen not to show the action of the aconitine, but to show that when there was a slight difference between the rhythm of the auricle and that of the ventricle, a certain type of interference was produced between the auricular and ventricular pull; and these afforded an easy way of determining rhythm of the auricles and ventricles.

Ergot: A Clinical Experimental Study.

By J. Gordon Sharp, M.D.

The present essay deals with ergot and some of its active principles from the clinical and therapeutic sides, and an attempt has been made to apply as far as possible the exactness of laboratory methods to work carried on at the beside.

Introductory Note on the Chemistry and Physiology of Ergot.

The chemistry and physiology of ergot has hitherto been surrounded by so much confusion that one may be pardoned for shortly passing in review the important work that has been recently carried out in the two domains just mentioned. Up to the year 1875 no exact analysis of ergot had been made, and all that each successive worker had done had been to isolate an impure substance and to give it a different name from that of his predecessor; but in this year Tanret isolated a pure crystalline alkaloid to which he gave the name ergotinine. In addition he found an *amorphous* alkaloid which unfortunately he was unable either to obtain pure or to form crystalline salts therefrom. This amorphous alkaloid was named amorphous ergotinine: it was found to be toxic. Little progress was made till Barger and Carr took up the work, and in 1906 they were able to confirm Tanret's research as to the presence of a crystalline ergotinine and they retained the name given to the alkaloid by Tanret; but further, they were able to isolate Tanret's amorphous ergotinine in a state of purity and to form crystalline salts. Both alkaloids were examined by Dale from the physiological side, and he showed that the crystalline ergotinine was, when pure, almost inert, whilst the amorphous ergotinine was found to be highly toxic, and from this circumstance it was named *ergotoxine.* Soon after this Kraft in working on ergot also obtained Tanret's amorphous ergotinine in the pure state and gave it the name *hydroergotinine.* From the chemical standpoint this is a better name than ergotoxine, for as both Barger and Carr and Kraft have shown, this amorphous ergotinine is the hydrate of the crystalline ergotinine, and to put it conversely, the crystalline ergotinine is the anhydride of the amorphous ergotinine. Further, the one can be readily changed into the other by abstracting or adding a molecule of water, as the case may be. The formula suggested for ergotinine is $C_{35}H_{39}O_5N_5$, so that the formula for ergotoxine (using Barger and Carr's name), or employing Kraft's name of hydro-ergotinine, would be one H_2O more, that is, $C_{35}H_{41}O_6N_5$.

Dale was able to show that ergotoxine produced in toxic doses most of the physiological effects of crude ergot, namely, ataxia, dyspnœa, salivation, gangrene, gastro-intestinal irritation, besides the special action on the sympathetic nervous system. Also contraction of the arteries (and raising of the arterial pressure) and contraction of the uterus in small doses ; and in large doses a paralysis of the sympathetic, and, of course, relaxation instead of contraction of both arteries and uterus. It did not, however, produce convulsions.

On analysis of galenical preparations of ergot, Barger and Carr found ergotoxine present in amount far too small to account for their action, and Dale came to the conclusion that probably the galenicals owed their activity to other agents. Buchheim had, in 1874, suggested that ergot acted in virtue of the putrid bodies present in it. An analysis of the galenicals showed that they contained a series of amines, and one in particular, *tyramine*, or *ergotamine*, was found to raise the blood-pressure and to contract the uterus in certain animals. It can be prepared synthetically and is known as *p-hydroxyphenylethylamine*. It is tyrosine less one CO_2 and it is related to adrenalin. *Isoamylamine* and other amines are present in sparing amounts. More recently there has been isolated from ergot *ergotidine*, or *β-iminazolethylamine*, which contracts the uterus of the guinea-pig with solutions of 1 in 250-millionth part, but the most notable property is that it *lowers* the blood-pressure in small doses, the very opposite of ergotamine (tyramine) and ergotoxine. β-iminazolethylamine has received the name ergotidine. It can be prepared synthetically and it is histidine less one CO_2. It appears to be chemically like, but physiologically different from, an ergot principle isolated by Kutscher.

In working with reliable galenicals and with ergotoxine Dale made two valuable additions to our pharmacological knowledge: (1) That good crude ergot and ergotoxine first stimulate and later, if the dose be large enough, inhibit the motor sympathetic. Prior to this work it was thought that ergot contained two principles—one which, for example, contracted, the other which relaxed the uterus. (2) Dale located the probable point of action of ergot and ergotoxine—namely, the myoneural junctions, that is, the point where the nerve joins the muscle.

PREPARATIONS AND DOSAGE EMPLOYED.

In most of the cases the liquid extract has been employed, prepared from good sound Spanish ergot. I have found it active at the end of thirteen months. In labour I have used the fresh infusion, but have found it not more active than the liquid extract. Ergotoxine I have also used and tyramine (ergotamine).

As ergot is more commonly employed in connexion with labour and abortions than in other affections, I have naturally had more experience of its use in this direction than in any others. I have full notes of cases in which the liquid extract has been exhibited thirty-one times, ergotoxine eight times, the infusion ten times, tyramine eight times.

Dosage.

Incidentally here and there the dosage of the various preparations will be dealt with, but it is convenient to summarize them under one heading.

(1) In connexion with parturition 30 ♏ by the mouth (1·7 c.c.) of the liquid extract is the maximum dose that should be employed. Now and then I have given by the mouth 45 ♏ (2·66 c.c.), but I regard this dose as too large, for I have observed inhibition follow this quantity. I have known as much as half a fluid ounce (14·21 c.c.) of the liquid extract to have been given, followed in an hour by another half-ounce, and because it did not cause uterine contraction the preparation was said to be bad. It would not produce stimulation, for it had gone beyond this and had induced inhibition and relaxation instead of contraction of the uterus.

Ergotoxine.—If my contention be correct, the dose of this alkaloid should not exceed $\frac{1}{100}$ gr. (0·00065 grm.) when injected hypodermically. Ergotinine being inert, or nearly so, need not be more than mentioned.

Tyramine I have not found satisfactory, as will be shown later.

(2) *In Abortions and Bleedings generally.*—Sixty minims (3·5 c.c.) of the liquid extract, spread over twenty-four hours and divided into four or six doses, is generally a sufficient dose. Occasionally it is better to prescribe the semi-solid extract in pill form, when the most useful dose is 2 gr. to 3 gr. (0·13 grm. to 0·194 grm.), not more than six times in twenty-four hours. The tincture possesses no advantage over the liquid extract, and I seldom employ it. I have had no experience of the alkaloid in cases other than in connexion with parturition.

PART I.—ACTION ON THE UTERUS.

Selection of Patient for the Exhibition of Ergot.

Only a small number of women in labour need ergot; the majority are best left alone to Nature to terminate the act. Even the absence of the medical man is often preferable to his presence till within an hour or two of the end, according to distance, provided that he has made his diagnosis and is sure of it. Only judgment and experience can teach him how long or how short a time he is to spend with his patient. It is not necessary to detail the general rules for the giving of ergot, for these are known to all. Now and then a man may have to decide whether it is better to give ergot or to apply forceps, and if there is any reasonable likelihood of ergot acting as well as forceps, then by all means let him employ the drug, for its administration causes no terror to the woman. The special conditions calling for the employment of ergot will be spoken of later.

Method of Investigation.

Having decided that a patient should receive ergot, I note the character of the pains for at least twenty minutes as follows: I sit down by the patient's side; the abdomen is covered by a thin sheet unless the patient complains of cold, when an additional covering is laid on and so arranged that I can place the left hand with as little between it and the abdomen as possible; thus the first beginnings of a pain are felt and noted all through its phases till it has passed off. On the wrist of the left hand is a wristlet with a watch in it, and in the back of the wristlet is a card on which anything can at once be noted down. The right hand is free to make vaginal examinations or to make notes on the card. The hands are gloved, and a basin with antiseptic solution stands ready at hand. Having learnt the workings of the uterus without drugs, I now give the dose of ergot and again place my hand on the abdomen and never take it off till I have noted the first signs of ergot action and three or four pains typical of ergot action. Once or twice during the experiment I may place one or two of the fingers through the vagina right up to the os, taking as much care as I can not to disturb the parts more than necessary. I keep the fingers in position till three or four pains have come and gone. Thus I am enabled to note every phase of a pain as to its effect on the abdominal walls and muscles on the uterus and how it contracts and relaxes—the contraction of isolated fibres, &c.; while the right hand reveals the state of the parts internally, such as the vagina, os, and other parts, and how the child reacts to the drug. The lesson has proved valuable, for it enabled me to formulate to my own satisfaction certain rules for the giving and withholding of the drug. It was pleasant to observe the triumphs of one's art—the parts yielding, the child coming down, and the woman spared suffering. (*See* fig. 1.)

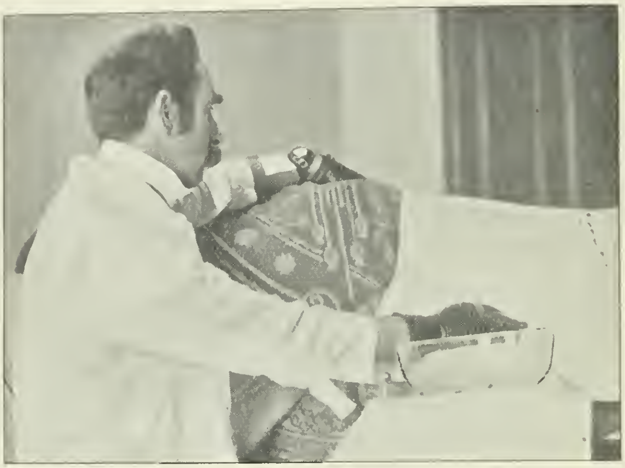


Fig. 1.

Shows method employed by the writer in investigating the action of ergot, &c. The left hand is kept on the abdomen, and on the wrist of the same hand is a watch to record time. The right hand is free to write or to make examinations.

Measurements of the Uterus in Passive and Active Contraction.

Having ascertained the changes that take place in the uterus during a pain as far as one could by the hand, the next consideration was to try if one could obtain any actual measurements, because in a labour pain more is concerned than muscular contraction, for coincident with and as a result of the labour pain the uterus erects itself in the abdomen. Now this erection means that the organ is higher in the abdomen at the height of a pain than when the uterus and its appendages are at rest, or, more correctly, when it is only undergoing passive contraction, for the uterus is never at rest. This erection is more than corrected by the contraction of the great mass of muscle-fibres of the pregnant uterus, so that when at the height of a labour pain the uterus measures more from navel to backbone and less from pubis to xiphisternum than it does when no pain is present. Thus by applying a tape measure from the highest point reached by the uterus to the lowest point at the pubis in rest I found it, say, 16 in. (41 cm.),

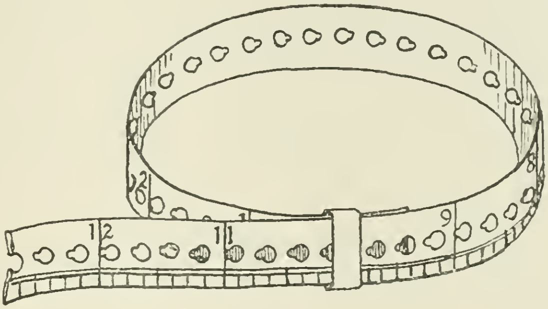


Fig. 2.

Steel tape with slot for registering the changes in size of the abdomen during a labour pain.

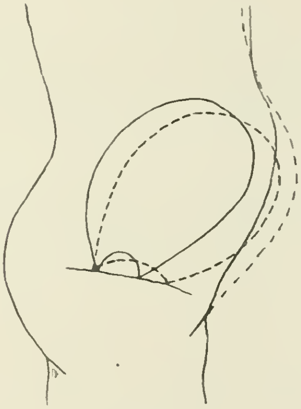


Fig. 3.

The black closed line represents the uterus before the onset of a labour pain; the dotted line represents the organ at the height of a labour pain.

and then on making the same measurements at the height of a pain the length was found to be about 1 in. (2·5 cm.) less, or about 15 in. (38·25 cm.). I had a steel tape made with a slot so that when this measure was applied round the woman's abdomen, at about the level of the navel at rest, it registered, say, 40 in. (100 cm.), while at the height of a pain the tape had run out another inch (2·5 cm.), or 41 in. (10·25 cm.) (*see* figs. 2 and 3).

I know nothing that so well illustrates the relaxed and the contracted states of the uterus as the relaxed and the contracted states of the biceps muscle of the arm. A relaxed biceps at its insertion measures in length, that is, in the length of the bone, say 5 in. (13 cm.), and its circumference at a marked point 10 in. (26 cm.). The same muscle contracted is in length shortened to 4 in. (10·5 cm.), and in circumference increased to 11 in. (28 cm.).

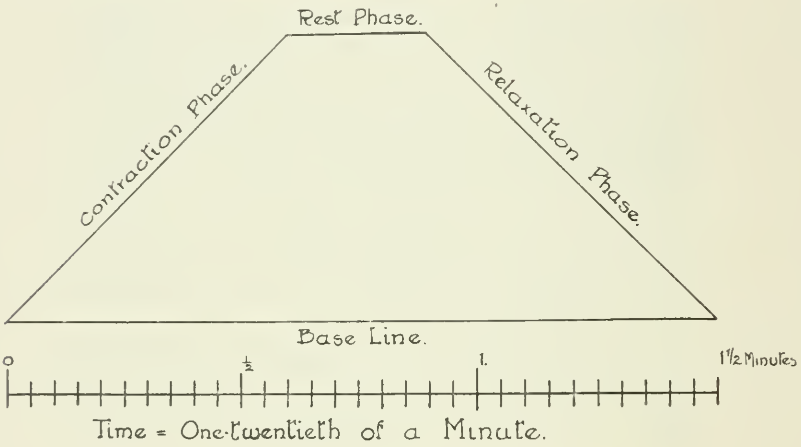


FIG. 4.

Diagram of a normal pain showing the contraction phase, in which the uterus gradually contracts till the highest point is reached, when there follows the rest phase and the uterus is at rest in tonic contraction. There next follows the relaxation phase and the contraction gradually becomes undone.

What constitutes a Labour Pain?

Having followed the foregoing method one is able to appreciate what is meant by the term "a labour pain." It is something more than a muscular contraction, for it is accompanied by pain, and the pain does not pass away till relaxation is complete. First, there is a steady contraction which reaches its height, and then there is a short period when the uterus appears to be at rest; and next, there is a period when the uterus is felt to be relaxing and the woman says the pain has gone. In fig. 4 I have tried to represent a pain in diagrammatic fashion. A time marker is inserted and the contraction phase of the pain rises from a base line up to a certain point, when there follows the rest phase; and lastly, the relaxation phase. In fig. 5 the contraction phase instead of being a steady tonic contraction like fig. 4, is broken up by five clonic spasms, so that the contraction may be described as coarsely clonic or tonico-clonic in character. Both are drawn from actual cases.

It is well to bear in mind that movements and contractions are continually taking place in the pregnant uterus, but these do not cause pain, and one might term them physiological as opposed to the labour pain, which might be described as pathological. If the fingers be inserted up to the os at the beginning of a pain, while the other hand is placed on the abdomen, it will be found that the os attains its maximum dilatation for that particular pain some time before the body of the uterus has reached the height of its contraction. The value of this must be at once apparent, for there is then a circumference of firmly contracted fibres to serve as fixed points for the yet contracting fibres of the great body of the uterine muscle.

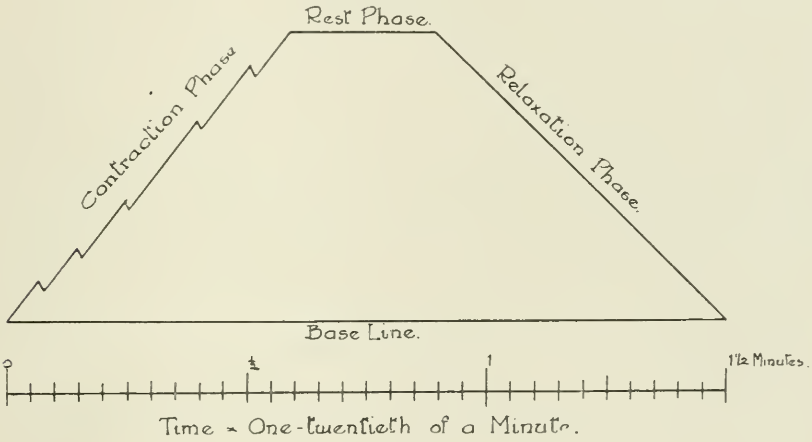


FIG. 5.

The contraction phase of the pain is really tonico-clonic, but the rest phase and the relaxation phase are normal. Compare with figs. 9 and 10.

Frequency and Time of Lasting of Pains.

It need hardly be said that the character of the pains differs both as to frequency in recurrence and as to time of lasting in different women, but the course is something like the following:—

Case I.—In a woman, aged 22, in her case, and within two hours of delivery, the pains appeared with almost unerring regularity for one hour at the rate of one every fifteen minutes.

Case II.—In another first pregnancy in a woman aged 25, and within one hour of the end, the pain came every two and a half minutes till the last pain or two.

The next three cases are more fully treated.

Case III.—Woman, aged 32; first labour within one and a half hours of end. The contraction phase is an almost unbroken rise to the top. The period of rest is almost unbroken by subsidiary waves, and the phase of relaxation is an unbroken downward line.

As to time, the following is an illustration of what may be expected in certain cases. The pains often come in cycles, and the following is a cycle made up of five pains:—

No. 1	All three phases lasted	1½	minutes				
,, 2	,, ,,	3½	,,	and came in	5	minutes after No.	1
,, 3	,, ,,	3	,,	,,	10	,,	2
,, 4	,, ,,	3½	,,	,,	8	,,	3
,, 5	,, ,,	4	,,	,,	9	,,	4

Case IV.—Woman, aged 30; first case. Within one and a half hours of end. Here is an analysis of six pains, studied as to the time occupied by contraction and then by the phases of rest and relaxation:—

No. 1 at 8.25	Contraction	lasts 45 secs.	and rest and	relaxation another	45 secs.
,, 2 ,, 8.31	,,	,, 30	,,	,,	30 ,,
,, 3 ,, 8.35	,,	and relaxation irregular			
,, 4 ,, 8.36	,,	,,	,,	also	
,, 5 ,, 8.39	,,	lasts 30 secs.	and rest and	relaxation nearly	30 secs.
,, 6 ,, 8.41	,,	,, 30	,,	,,	exactly 30 ,,

Such is the class of case usually met with in a first labour, and in a second labour in a woman aged 22 to 27 with the muscular system in tone, the pains as regards rhythm, force and amplitude do not differ much from those of a first case. The uterus is in a state of tone, or even of hypertone, but after the second labour there is a tendency for the organ to pass into a state of hypotone or undertone. Here the contraction phase of the labour pain is neither a steady straight-up contraction nor tonico-clonic in character, but is sinuous; the rest phase is not marked by a state of tone, but the uterus is soft and easily depressed and the relaxation phase is "wavy," as if the organ was contracting and relaxing imperfectly and alternately. Fig. 6 is intended to represent such a condition, and the following details illustrate a somewhat exaggerated case of the kind I speak of. It is a toneless uterus.

The Toneless Uterus.

A woman, aged 30, in her fifth labour and within two hours of the end, had pains coming on every two to two and a half minutes, marked by a sinuous rise and then a rapid relaxation, but during the pain small bundles of fibres were found to be contracting independently, but further the uterus and its appendages were so irritable that when the fingers were inserted into the vagina a general pain was often initiated quite out of the usual series. These extra pains appear to spend energy without performing useful work; but more, they never extend quite to the whole of the uterus, and the organ may experience at one and the same time contraction and relaxation. This is the class of case in which ergot is especially useful.

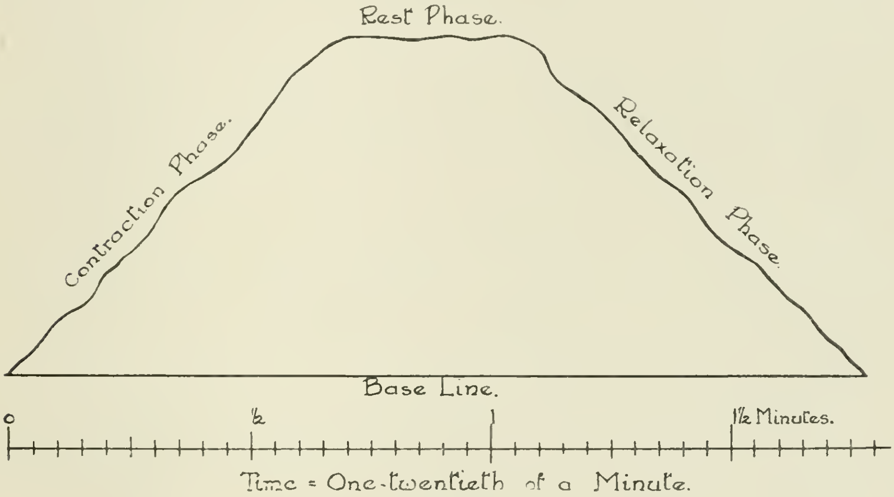


Fig. 6.

This shows the pains of a uterus in undertone, hypotone, tonelessness or beginning of inertia. The phases are "wavy" instead of being straight and firm. There is never a really firm contraction.

Before quoting cases showing the beneficial action of ergot one may be allowed to anticipate events and to discuss the three following points:—

(I) *Conditions of the Parturient Uterus in which Ergot is indicated.*

First and foremost, ergot should not be given unless (1) the os is fully dilated; (2) unless there is no obstruction, bony or other—and this means that the head must be well down in the pelvis, and that the soft parts of the pelvic canal and outlet are soft and yielding, for nothing but harm can come of sending the fœtal head against the unyielding soft parts of the vagina.

One may lay it down as a good general rule that ergot is oftener required in multiparous cases than in first cases, unless the latter are long drawn out and the uterus exhausted.

(II) How Ergot acts in Labour, or what one may expect of Ergot.

(*a*) It collects the small irregular and non-effective pains into larger effective contractions, so that the labour is sooner ended and the uterus saved work.

(*b*) The drug improves the tone of the muscle-fibres, whereby the uterus gains an advantage in that its musculature is ready at once to respond to further contraction, and thus neither time nor energy is spent in bringing the fibres up to that point at which contraction begins.

(*c*) By acting on the abdominal muscles and on the muscular system generally, the tissues as a whole are improved in tone. This helps the uterus in its struggle.

The foregoing may be called the immediate benefits, but there is often a more remote beneficial action.

(*d*) The forcible contractions following the administration of ergot, by expelling uterine contents, such as after-birth, membrane or other foreign body, relieve the organ of its troubles and set it free to recover itself in the ordinary way of Nature.

(III) How soon do the Liquid Extract and the Infusion act when given orally, and Ergotoxine injected hypodermically?

The two first were given in doses equal to 30 gr. (nearly 2 grm.) of the dried drug. Thirty minims (1·7 c.c.) of the liquid extract were given in about 2½ fl. oz. (75 c.c.) of warm tea or in the same of cold water, and 30 gr. of the freshly powdered drug were infused in 2½ to 3 fl. oz. (75 to 90 c.c.) of boiling water, and given while warm without straining.

The hand was placed on the abdomen, and the first indication of ergot action observed and noted. Both acted in practically the same time, so that in the following table no distinction is made between the preparations. In twenty trials the times were as follows: One acted in four minutes, three in five, one in six, three in seven, five in ten, two in eleven, one in twelve, two in fifteen, one in nineteen, one in twenty minutes. This gives an average of ten minutes (*see* fig. 7). I did not try the time-action hypodermically, for after extensive use of the galenical preparations of ergot I am satisfied that when given by the mouth they act as rapidly as is necessary; besides, hypodermic injection of vegetable solutions is not unattended by danger.

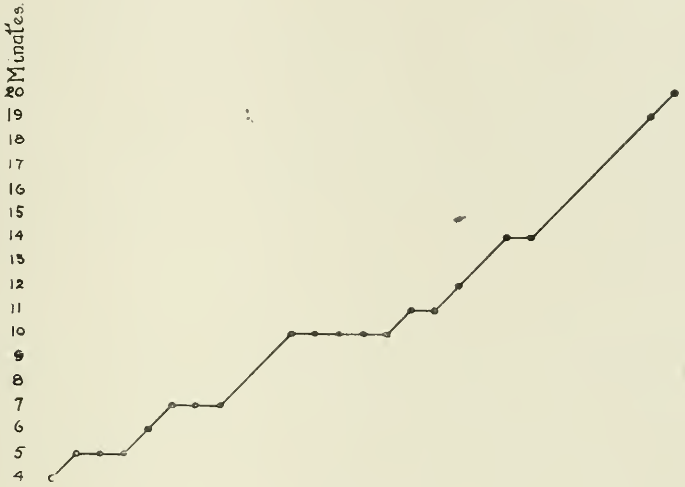


Fig. 7.

Shows at a glance the time in which infusion and liquid extract act when given by the mouth—an average of ten minutes—in doses equal to 30 gr. of the solid drug.

As might be expected, ergotoxine acts more promptly than any of the galenicals, and that for two reasons: (1) It is a pure active substance (alkaloid); (2) it is injected directly into the lymph spaces. Its average time is four minutes.

(I) Illustrative Cases: Liquid Extract.

Case I.—Woman, aged 22. O.L.A. Long labour. Inertness. Within two hours of delivery. I have said that ergot is not so often needed in a first labour, but here is one in which it was useful. This woman had been in labour many hours and was quite worn out, and there appeared to be true inertness or inertia of the uterus. The pains were feeble, although they returned with regularity every fifteen minutes. The uterus was soft and flabby, and at the height of the contraction its surface could be readily bulged inwards. After waiting for ninety minutes, and observing that no headway was being made, I gave 30 ♏ (1·7 c.c.) of the liquid extract by the mouth, and in twelve minutes the tone had improved, and the pains which previously had come on every fifteen minutes now came every three, two, five, two, one, and five minutes respectively, and the last brought the head on to the perineum, and the whole was ended in a little more than thirty minutes after the drug was given. Child alive.

Case II.—Woman, aged 41. Eleventh pregnancy. O.L.A. Within two hours of delivery. Paralysis of sphincter of anus and paresis of abdominal muscles. This woman had lax abdominal walls, and when the pains came on the uterus received no help from the abdominal muscles. The whole muscular system was in a state of hypotone, and the pulse was low in tension. The os was fully dilated, and the external parts were soft, flabby, and dilatable in a high degree; but although the pains appeared regularly every two and a half minutes, the uterus contracted very imperfectly, and the head made no progress. The external sphincter of the anus was completely paralysed, so that with each pain wind and rectal contents were passed. After waiting for forty minutes without any sign of improvement, I administered 45 ♏ (2·6 c.c.) of liquid extract of ergot by the mouth. In twenty minutes there was full ergot action. The uterus contracted firmly, and at the height of the contraction phase its surface, instead of being soft, smooth, and bulgable, was now firm, hard, and the surface irregular, as if bound with cords: the latter was due to the extreme tonic contraction of small bundles of fibres. The abdominal muscles, instead of being passive and uncontracted during the contraction of the uterus, now contracted firmly, and in unison with the uterus. The pulse also improved in tone. The head gradually came down; a living child was born forty minutes after the ergot was given. Long rest in bed cured the anal paralysis.

(II) Illustrative Cases: Fresh Infusion.

Case I.—Woman, aged 35. Sixth pregnancy. O.L.A. Within one and a half hours of delivery. Long labour. Feeble pains. I saw this woman at 4 in the afternoon, and she had been in labour since 10 in the morning. The membranes were unruptured, but the os was well dilated. The pains came on every five minutes, and the contraction phase lasted thirty seconds. I ruptured the membranes at 4.10. At 4.55 progress was not being made, and as I had the infusion ready in case of its being needed, I gave it by the mouth in dosage equal to 30 gr. of the dry drug. At 5.6, that is, eleven minutes later, there was evidence of ergot action with prolonged tonic contractions, and each pain brought the head down. A living child was born at 5.30.

Case II.—Woman, aged 36. Seventh pregnancy. O.L.A. Within two hours of delivery. Feeble pains. This patient was seen at 5 in the morning, when the presentation was made out. The head was below the brim, and the os was in medium dilatation, but the pains only came on every ten minutes. At 10.25 the os was fully dilated, but the head had not come down in a corresponding degree, and the pains, although now recurring every five minutes on the average, only lasted from twenty to thirty seconds. At 11.30 the conditions were much the same as at 10.25. All the outside parts and the vagina were soft and dilatable. At 11.45 she was given 30 gr. (about 2 grm.) of ergot infused as already detailed. In ten minutes there was ergot action, and at 12.12 a labour which looked as if it would last for hours was terminated by the birth of a living child.

It is unnecessary to quote any more cases, for the experience is ever the same—(1) that fresh infusion of a sound ergot produces rapid and effective contraction of the uterus, and (2) that although the infusion acts well it possesses no advantage over a carefully prepared liquid extract. (3) The action of a fresh infusion appears to be in labour at least identical with that of a liquid extract, whether this extract be one or thirteen months old.

(III) Illustrative Cases: Ergotoxine.

I have experimented with this alkaloid as to its action in labour. I employed the pure alkaloid supplied by Messrs. Burroughs Wellcome and Co., and injected it deeply into the gluteal muscle. A recital of the results in two cases will show that it acts in almost all respects like the liquid extract or infusion. The chief differences being (1) that it acts more quickly (in about four minutes, as against ten minutes for the liquid extract and infusion); (2) it acts more energetically as a rule; (3) its action is more evanescent: its contractor action on the uterus being over in twenty-five or thirty minutes, against about forty or fifty minutes for the galenicals. Speaking generally, it possesses no advantage over the latter, unless a very rapid action is desired, when it is to be preferred.

Case I.—Woman, aged 45. Twelfth pregnancy. Child born. Retained afterbirth. No after-pains in this or former labours. Dose $\frac{1}{100}$ gr. (0·00065 grm.). A healthy child was born at 5 (as I was informed). I saw her at 6.19, and the after-birth had not come away, and the patient said there had not been a single pain since the child was born. One curious feature in this woman's case was that she had never suffered from after-pains in any one of her confinements. Here, then, was a suitable case for trial of the alkaloid, and so as soon as I could get it ready I injected the dose mentioned, having previously satisfied myself that the uterus was not contracting. In two and a half minutes there was moderate tonic contraction, and three and a half minutes later, or six minutes after injection, powerful tonic contractions occurred and the after-birth was expelled, and the patient asserted that for the first time she knew what after-pains were.

Case II.—Woman, aged 34. Fourth pregnancy. Occiput anterior. Within two hours of delivery. Toneless uterus. I saw her at 6, when the occiput was in the anterior position. The os was well dilated. At 6.20 the membranes ruptured. The pains returned every two to three minutes, and lasted from thirty to forty-five seconds. They were not of great amplitude, but what was most important, between the end of the one pain and the beginning of the next the uterus was quite flaccid. At 7.25 ergotoxine was injected. At 7.34 signs of ergotoxine action—very late in onset. At 7.37 uterus now in tone between each pain, and not toneless as it was before. At 7.41 the amplitude of the contractions much increased. At 7.45 head almost at outlet. The labour was soon terminated by the birth of a living child, in contrast to the two previous confinements, when dead children were born, and no ergot in any shape or form had been employed. Here the ergotoxine did good by "gathering up small irregular and ineffective pains, and converting them into strong regular and effective pains.

(IV) Illustrative Cases: Tyramine (p-hydroxyphenylethylamine).

I had previously employed tyramine in doses of $\frac{1}{13}$ gr. (0·005 grm.), $\frac{2}{13}$ gr. (0·010 grm.), and $\frac{3}{13}$ gr. (0·015 grm.), but as these had no effect at all on the labour pains, I resolved in future to employ larger doses, carefully to note the action (if any) and, if no appreciable action followed then, to administer 30 ♏ of liquid extract of ergot to the same patient and to compare its action (if any) with the action (if any) of the large dose of tyramine. This was necessary in order that one might not do an injustice to tyramine; hence, in the next two experiments each was divided into *(a)* a period of observation of the pains before anything was administered; *(b)* a period during which tyramine had been given to the patient; and *(c)* a period when the woman was under the influence of liquid extract of ergot if this was necessary.

Case I.—Woman, aged 26. First labour. O.L.A. In active labour ten hours; two hours before delivery head well down, os fully dilated.

(*a*) *Observation for twenty-nine minutes before anything administered* (4.39 *till* 5.8).

No. 1 pain	... 4.39 ...	Lasts 30 seconds (feeble).	Uterine muscle soft during pain.		
,, 2 ,,	... 4.42 ...	,, 30 ,,	,,	,, ,,	,,
,, 3 ,,	... 4.45 ...	,, 30 ,,	,,	,, ,,	,,
,, 4 ,,	... 4.47 ...	,, 45 ,,	,,	,, ,,	,,
,, 5 ,,	... 4.51 ...	,, 30 ,,	,,	,, ,,	,,
,, 6 ,,	... 4.54 ...	,, 45 ,,	,,	,, ,,	,,
,, 7 ,,	... 4.57 ...	,, 30 ,,	,,	,, ,,	,,
,, 8 ,,	... 5.0 ...	,, 45 ,,	,,	,, ,,	,,

Average rate nearly three minutes; almost no progress has been made.

(b) At 5.8 injected into hip $\frac{4}{13}$ gr. (0·02 *grm.*) *tyramine. Observation for twenty-two minutes* (5.8 *till* 5.30).

No. 1	pain	...	5.10	...	Lasts	30	seconds	(feeble).	Uterine muscle	soft	during pain.
,, 2	,,	...	5.12	...	,,	30	,,	,,	,,	,,	,,
,, 3	,,	...	5.15	..	,,	30	,,	(stronger ?)	,,	(firmer ?)	,,
,, 4	,,	...	5.18	...	,,	30	,,	,,	,,	,,	,,
,, 5	,,	...	5.20	...	,,	30	,,	,,	,,	,,	,,
,, 6	,,	...	5.22·5	...	,,	25	,,	,,	,,	,,	,,
,, 7	,,	...	5.24·5	...	,,	30	,,	,,	,,	,,	,,
,, 8	,,	...	5.28	...	,,	25	,,	,,	,,	,,	,,

Average rate at which pains appear a little increased; last a shorter time. Pains doubtfully stronger; head advanced a little, as might be expected.

(c) *Observations for twenty-four minutes* (5.32 *till* 5.56) *when head on perineum. At* 5.30 30 *fl.* ♏ (1·7 *c.c.*) *liquid extract of ergot given orally.*

No. 1	pain	...	5.32	...	Lasts	30 seconds (feeble).	Uterine muscle	soft	during pain.
,, 2	,,	...	5.35	...	,,	over 30 secs. (stronger ?)	,,	firmer	,,
,, 3	,,	...	5.36	...	,,	a few seconds			
,, 4	,,	...	5.40	...	,,	60 seconds (stronger ?)	,,	soft	,,
,, 5	,,	...	5.42	...	,,	30 ,,	,,	,,	,,
,, 6	,,	...	5.44	...	,,	30 ,,	,,	,,	,,

Pain No. 6 appeared as if it was to pass off in thirty seconds, but quite suddenly another pain followed, and then another, with only a few seconds' intermission till 5.56, when the head was driven down on the perineum. That is, in twelve minutes after the ergot was given there was evidence of ergot action, and in twenty-four minutes after the drug was given by the mouth the head was in the perineum. At 6.30 a living child was born; the delay of thirty-four minutes between birth and the appearance of the head at the outlet being owing to a hard, unyielding perineum and soft parts at the outlet. During the whole course of the experiment the woman was lying on her left side, and only one vaginal examination was made in the course of each of the three observation periods. No doubt as to the superiority of the liquid extract of ergot over tyramine.

Case II.—Woman, aged 29. First case. O.L.A. Head extended; lingering labour, twenty-four hours; os fully dilated, head well down; pains feeble. Woman on left side.

(*a*) *Observation lasts thirty minutes* (8.12 *till* 8.51). *No drug.*

No. 1	pain	...	8.12	...	Lasts	30	seconds.
,, 2	,,	...	8.14	...	,,	50	,,
,, 3	,,	...	8.15·5	...	,,	40	,,
,, 4	,,	...	8.20·5	...	,,	50	,,
,, 5	,,	...	8.22·5	...	,,	40	,,
,, 6	,,	...	8.26·5	...	,,	50	,,
,, 7	,,	...	8.29	...	,,	40	,,
,, 8	,,	...	8.32·5	..	,,	40	,,
,, 9	,,	...	8.34·5	...	,,	40	,,
,, 10	,,	...	8.37	...	,,	60	,,
,, 11	,,	...	8.41	...	,,	30	,,

The course of the pains up till 8.51 was much as recorded. Average frequency of recurrence of pains, a little over 2·5 minutes. Average time the pains lasted, a little over 40 seconds.

(*b*) *Observation lasts thirty-six minutes* (8.51 *till* 9.27). *Tyramine. At* 8.51, $\frac{6}{13}$ *gr., or almost* $\frac{1}{2}$ *gr.* (0·03 *grm.*), *tyramine injected into hip and part well rubbed.*

No. 1 pain	...	8.54	...	Lasts 30 seconds.
,, 2 ,,	...	8.57·5	...	,, 40 ,,
,, 3 ,,	...	9.0	...	,, 60 ,,
,, 4 ,,	...	9.2·5	...	,, 30 ,,
,, 5 ,,	...	9.5	...	,, 20 ,,
,, 6 ,,	...	9.6·5	...	,, 50 ,,
,, 7 ,,	...	9.8·5	...	,, 45 ,,
,, 8 ,,	...	9.11	...	,, 40 ,,
,, 9 ,,	...	9.14	...	,, 35 ,,
,, 10 ,,	...	9.16·5	...	,, 55 ,,
,, 11 ,,	...	9.21	...	,, 40 ,,

At 9.10, 2 fl. oz. of cold water given. Average frequency of recurrence of pains a little over 2·5 minutes, and practically the same as under Observation (*a*). Average time pains last, a little over 40 seconds, and a trifle less than under Observation (*a*). Thus neither in force, frequency, nor amplitude had the pains increased under the drug. Tone perhaps a trifle improved.

(*c*) *Observation lasts thirty-three minutes* (9.27 *till* 10), *that is, from the time ergot was given till the head is on the perineum. At* 9.27 30 *fl.* ♏ *of liquid extract of ergot given orally.*

No. 1 pain	...	9.30	...	Lasts 40 seconds.	
,, 2 ,,	...	9.32·5	...	,, 50 ,,	
,, 3 ,,	...	9·35	...	,, 90 ,,	Evidence of ergot action eight minutes after given.
,, 4 ,,	...	9.38·5	...	,, 125 ,,	
,, 5 ,,	...	9.42			

After this the pains became almost continuous, and at 10, or thirty-three minutes after the ergot was given by the mouth, the head was in the perineum and the labour was speedily terminated by the birth of a living child.

One could hardly obtain better evidence of the efficacy of ergot and of its superiority to the amine with which it is compared.

Does Opium hinder the Ecbolic Action of Ergot?

I prefer opium to morphine in the present connexion, for it appears to me to give more comfort to the woman in labour than does an equivalent of morphine. This may be owing to a more localized as well as to a slower and more prolonged action. When given in conjunction with ergot my experience is that the effect opium had was to blunt the sensory centres, while leaving the ergot to act in its own special way and to produce the usual uterine action. Indeed, so far from retarding the stimulant action of ergot, opium often appears to stimulate the cord and so simulate the action of ergot. It is a common experience in midwifery practice that opium when given by itself for the first ten to fifteen minutes after exhibition, increases the force and frequency of the pains, while its analgesic action only comes on after this time. The narcosis produced by 25 to 30 ♏ of tincture of opium is often sufficient to render the woman incapable of pushing with the feet and pulling with the hands when a labour pain comes on, and this is so much lost, but, on the other hand, the patient is saved suffering. It appears as if opium delayed the onset of the ergot action, but, on the other hand, it also seemed to prolong the time during which it produced powerful contraction action. The recital of three cases will illustrate some of the points under discussion.

Case I.—Woman, aged 25. First pregnancy. O.L.A. The woman was first seen at 10.30, when the head was at the brim. The os was not dilated more than 1 in. (25 cm.). The other parts were all relaxed. The pains recurred every ten minutes and were non-effective, for the woman was tired out and it was evident she wanted rest. I gave by the mouth 30 ♏ (1·7 c.c.), of tincture of opium, equal to about 2 gr. (0·129 grm.) of opium. This was at 12.45. The pains continued much the same till about 4, but in between the pains the woman slept comfortably. The os was now fully dilated, but it was plain that something more was necessary to terminate the labour. At 5.15 she was given by the mouth 30 ♏ of fluid extract of ergot, and in twenty minutes the uterus contracts more forcibly than it has done in the past seven hours. The opium, too, is in full action; the hand on the abdomen detects the onset and passage of the pains, although the only outward evidence of their presence is a momentary attempt on the part of the woman to clutch the towel in order to pull, but in a few seconds she falls back into a slumber. Each of these attempts is coincident with the onset of a pain. Meanwhile the head gradually came down as if no opium had been given, and a few minutes before 6 the head was on the perineum, when the woman suddenly awoke from the slumber, and at 6 a living child was born.

Case II.—Woman, aged 28. Third pregnancy. O.L.A. Patient first seen at 9.25, when she was reported to have been twelve hours in labour. The os was fully dilated, and all the soft parts soft and relaxed. The pains were in abeyance, and the woman was despondent. I gave her by the mouth at one and the same time 25 ♏ of tincture of opium, equal to about 1·75 gr. of opium (0·113 grm.) and 30 ♏ of fluid extract of ergot (1·7 c.c.). In nineteen minutes the ergot was in full action, and in forty minutes after it was given the opium had begun to take effect, for although I could feel that tonicoclonic contractions took place at intervals, the woman did not feel them to any appreciable extent, and a few minutes later the labour terminated by the birth of a living child and with comparative comfort to the woman.

Case III.—A multiparous woman, aged 28, had been in labour for twelve hours. The whole of the parts were lacking in tone, and the woman was worn out. This was at 9.35. I administered 25 ♏ of tincture of opium (1·75 gr. or 0·113 grm. of opium) and 30 ♏ of fluid extract of ergot. Again the ergot did not act for eighteen to twenty minutes, and the opium did not produce marked analgesia for nearly three-quarters of an hour, and from this time till near the end the woman had only a hazy notion of pains being present, for she dozed in their absence, and only expressed some uneasiness while they were present till near the end of the labour. A living child was born at 11.

Medium Chloroform Anæsthesia. No Modification of Ergot Action.

A perusal of the following notes of a case will show that the action of ergot on the uterus in labour is in no way modified by chloroform anæsthesia. This may not be considered worthy of proving, for it is well known that the movements of the uterus are independent of nervous impulses; that involuntary muscle-fibre such as the intestinal and uterine are less susceptible to poisoning by chloroform than is the musculature of the heart, blood-vessels, and than are the voluntary muscles. Further, it is well known that the higher centres can be brought into play by the woman to inhibit or delay a pain, or succession of pains, for a time at least, so that one might argue that the giving of chloroform would remove the inhibiting power of the will. However, theory and experiment do not always agree with clinical experience.

How the Chloroform Anæsthesia was carried out.—The anæsthetic was exhibited by taking a piece of blotting-paper 8½ in. by 6 in. (22 cm. by 14·5 cm.), folding it in four and placing it at the bottom of a stout tumbler, saturating the paper with the anæsthetic, the tumbler being afterwards inverted to ensure that no chloroform lay at the bottom. When the pains came on the woman seized the tumbler and placed it over her mouth and nose and inhaled the vapour: the tumbler naturally did not fit accurately and so there was no danger of the patient receiving too large an admixture of chloroform vapour, even if this was possible from the small volume of chloroform held by the blotting-paper. Having no handle the tumbler fell from the patient's hand on the onset of the slightest anæsthesia, so there was no danger on that side. This method enables one to give chloroform safely, and in such quantity as to blunt the pains and no more. At the end of each pain the paper was again saturated with chloroform and turned with its mouth downwards on a clean towel, when it was ready against the start of the next pain. (*See* fig. 8.)

Illustrative Case.—Woman, aged 33. First labour. Occiput anterior. Was seen at noon, when she had been in labour for ten hours. The head was at the brim, the os dilated to a diameter of $1\frac{1}{2}$ in. (3·75 cm.), the bag of membranes protruding, and the occiput to the front. The pains recurred every fifteen minutes and only lasted twenty seconds. I saw her again at 3, when the pains had increased in force and frequency, and the woman appeared to feel so keenly that I had to administer chloroform, beginning at 3.30. At 3.45 it was plain that the pains, which were small, frequent in recurrence, irregular in rhythm, and non-effective, were not likely to alter, so I gave by the mouth 30 ℳ of fluid extract of ergot (1·7 c.c.). In ten minutes the pains became larger, more regular in rhythm, less frequent, but more effective. The chloroform was continued as before, and the only effect that the anæsthetic had on the course of the labour was that as the pains went on as before the woman was dull and apathetic, and made no effort at pulling the towel and pushing with the feet, so that their aids alone appeared to have been sacrificed, but much more was gained. The labour terminated at 4.30 by the birth of a healthy child.

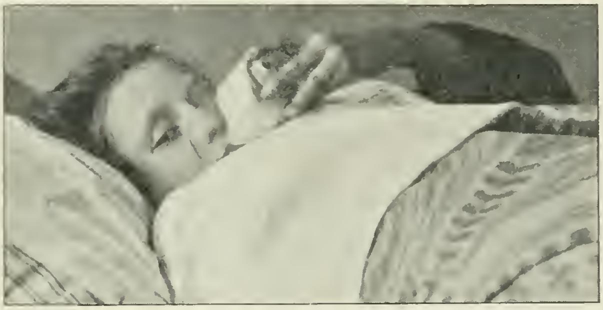


Fig. 8.

Shows method of giving chloroform to produce light anæsthesia. The glass cannot accurately fit over mouth and nose and hence the anæsthesia cannot be deep. The glass having no handle the fingers cannot become entangled, and so the vessel falls down as soon as the woman "goes over," if this ever really takes place. A stout glass should be employed to avoid breakage.

Deep chloroform anæsthesia would make no difference to the ecbolic action of ergot, but we are not here concerned with this condition, for deep anæsthesia is usually needed for operative treatment, in which ergot is not likely to be employed.

So-called Disadvantages of Ergot Examined.

(1) It occasionally fails to act for some unaccountable reason, but in this it only resembles other remedies of undoubted worth.

(2) It renders the whole uterine muscle more sensitive to a stimulus of any kind, so that touching the uterus through the skin or examination by way of the vagina, however carefully made, will set up a pain. This, however, is only an exaggeration of the normal state, for a strong stimulus applied to any uterus in labour will set up movement and contraction.

(3) *Inhibition with Pain; Value of Mechanical Stimulus; Kneading.*—I am now certain that I have seen one or two cases of this, but as I had not kept careful notes and had not carefully observed the various circumstances of the cases, I am unable to make any statement of value. The following I carefully observed, however:—

Inhibition with Pain.—This case was in a woman aged 25, and it occurred in the practice of another medical man who asked me to see the patient with him. The labour had come off without any difficulty, and a short time before its termination the practitioner had given by the mouth 45 ♏ (2·6 c.c.) of the liquid extract. After the birth of the child the uterus had not contracted, and the after-birth had not come away. When I saw the woman she complained of great pain in the abdomen. I passed my hand over the part, but could not make out the contour of the uterus, so great was the relaxation. Although the relaxation was so great there was no bleeding. Kneading the abdomen gradually brought about contraction of the uterus, expulsion of the after-birth, and relief of the pain, and no further trouble was experienced. I believe the comparatively large dose of ergot had induced inhibition of the uterus.

The important points are: (1) That mechanical stimulation is a valuable line of treatment when relaxation from whatever cause has resulted; (2) that a comparatively small dose of ergot will sometimes cause inhibition of the nervous mechanism and relaxation of the organ; and so in my experience 30 fl. ♏ of the liquid extract of ergot is the average maximum dose for women in labour.

(4) *Error of Judgment in administering Ergot.*—It will occasionally happen that, however careful one may be, ergot will be given when the parts are not ready to effect the birth. The error is not productive of anything more serious than disappointment, for the powerful effect of the drug is over within an hour, and the delivery can be brought about by waiting, opium, or forceps.

Illustrative Case.—Woman, aged 30, in labour, was judged to be suitable for the exhibition of ergot. All the soft parts were relaxed, the head was in the O.L.A. position, the pains were feeble and only recurred every twenty minutes, and all that appeared necessary was a few strong pains in order to bring about delivery. At 8.37 she was given 30 ♏ (1·7 c.c.) of the fluid extract. At 8.47 there was a powerful pain and at 8.49 another; another at 8.53, 8.56, and 9.3, and afterwards almost regularly every three minutes till 9.24, when the ergot action ceased entirely, and there followed feeble pains of no service, and at 9.45 the woman was delivered by forceps with the greatest possible ease, and a living child born.

The exhibition of ergot in no way interferes with the employment of forceps, for as soon as these are applied the contractor action of the drug ceases.

(5) Extreme ergot action.

(*a*) *Tonico-clonic and Fine Tetanic Contractions after Large Dose.*—Woman in her fifth pregnancy and within two hours of delivery. O.L.A. position and os fully dilated. The pains came on every two or two and a half minutes, but were lacking in force. The whole uterine muscle was in undertone, for at the height of a contraction the organ was so flaccid that it could always be pushed in. The condition was evidently one in which ergot was indicated, and I gave by the mouth 45 ♏ of the liquid extract (2·6 c.c.). In seven minutes there was true ergot action. The tone was improved, and at the height of the contraction the uterine muscle could not be indented with the knuckles as it could before. Ten minutes later the ergot action was extreme. I tried to analyse the various phases of the pain and found that the contraction phase was really a tonico-clonic one in which eight tonico-clonic contraction phases took place in one minute, then there followed rest phases

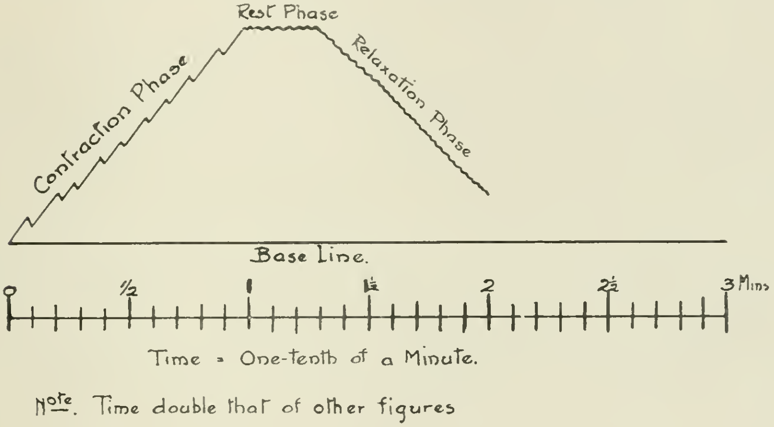

Rest Phase
Contraction Phase
Relaxation Phase
Base Line.
0
½
1
1½
2
2½
3 Mins
Time = One-tenth of a Minute.
Note. Time double that of other figures

FIG. 9.

This is somewhat like figs. 5 and 10 and it has been brought about by a large dose of ergot. The contraction phase is broken up by coarse tonico-clonic elements, but the rest phase and the relaxation phase show fine rapid tonic and clonic contractions almost tetanic in character.

lasting about three-tenths of a minute, during which small bundles of muscles were felt to be a state of rapid contraction; the relaxation phases lasted about seven-tenths of a minute, and the same rapid contractions were observed as in the rest phases. The relaxation phase was no true relaxation, for the relaxation seemed to be broken into by clonic spasms. The relaxation phase never appeared to be complete, for before it was evidently ended another contraction began and the cycle was repeated (*see* fig. 9). If this be compared with fig. 5 it will be remarked that the contraction phase in both are alike, so ergot only exaggerates the conditions often met with in a perfectly normal labour. The labour ended happily within one hour.

This case is quoted in order to show the extreme contractor effect of ergot. In my opinion, a few minims more would have produced only a trifling transient contractor action and a final inhibitory action in which the uterus would have relaxed.

(*b*) *Tonico-clonic and Tetanic Contractions after Large Dose.*—Woman, aged 30. Fifth pregnancy. Within two hours of delivery. The pains came on every two to two and a half minutes. The contraction phase was weak, although there was a steady rise till the maximum point was reached, but at this point, when the organ should have been firm it was soft and its surface could easily be bulged in; but more than this, there remained at the height of every contraction an arc of the uterus almost uncontracted. The abdominal walls were lax and flabby. The pulse was soft and low in tension so that the uterine muscle only shared in the general condition. I debated in my own mind whether I should apply forceps or give ergot. I decided on the latter alternative and gave by the mouth 45 ♏ (2·6 c.c.) of the liquid extract. In fifteen minutes there was full ergot action; one might almost say extreme ergot action. The contraction phase was made up of tonico-clonic elements lasting about sixteen-twentieths of a minute; of a rest phase tetanic in character lasting six-twentieths of a minute; and of a tonico-clonic relaxation phase lasting rather less time than the contraction phase. This last never seemed to be completed, or as I have represented in fig. 10 it did not reach the base line, for the cycle started at once and was repeated again and again till the head was on the perineum. When the ergot was in full action the uterus was so irritable that the slightest touch to the parts set up subsidiary or extra pains. I believe that I had here reached the maximum contractor dose of ergot and that an extra 10 ♏ (0·592 c.c.) would have produced inhibition after a short contractor action (fig. 10).

(*c*) *Extreme Tonic Contraction.*—It has been often asserted that the long-drawn-out tonic contraction induced by large doses of ergot may kill the child and do damage to the uterus. This is contrary to my experience, for I can remember many cases in which this extreme tonic contraction was present, and in none was damage done either to mother or child, for when the ergot acted in this particular manner it always spent itself, so to speak, very quickly. The following, of which I kept careful notes, will bear out this statement:—

Illustrative Case of above.—This was a woman, aged 30, in her fourth pregnancy, who had been long in labour. All the parts were flaccid and the abdominal muscles were so much relaxed that they rendered no aid to the tired-out uterus. The pains hardly advanced the head at all. The usual dose of 30 fl. ♏ was administered orally, and in five minutes there followed a pain which lasted exactly five minutes: two minutes were occupied by the contraction phase and three minutes by the resting phase, and the relaxation was accomplished by a sudden drop (*see* fig. 11). Smaller pains followed, and in half an hour a living child was born. Here, as in other cases in which the parts are toneless, ergot appears to act most energetically.

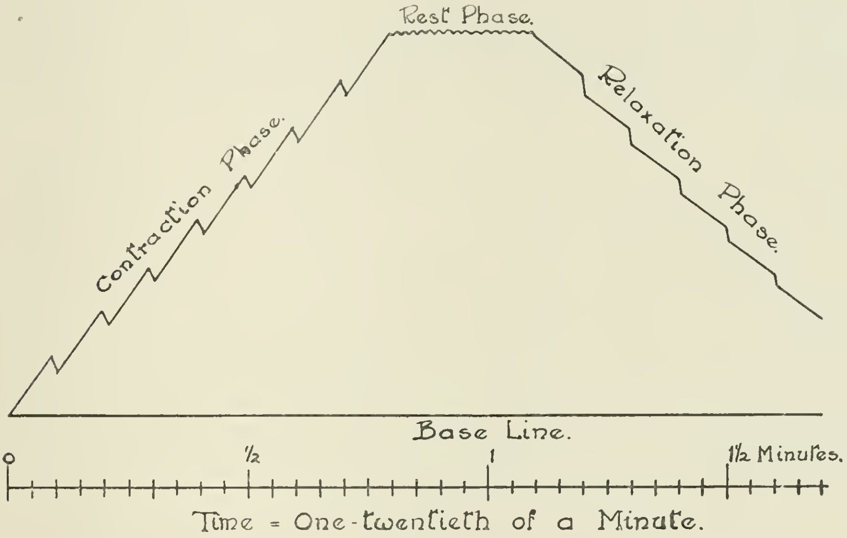


FIG. 10.

Resembles figs. 5 and 9, but here the ergot action is more extreme. The contraction phase of the pain shows coarse tonic and clonic elements, and the rest phase shows fine tetanic contractions, and the relaxation phase is broken up into tonic and coarse clonic contractions.

I now go on to describe one or two cases which appear to illustrate an abnormal action of ergot.

Abnormal Action of Ergot.

(*a*) *Tonic Spasm of Sphincter Vaginæ and Levator Ani delaying Delivery.*—Woman, aged 26, in her first pregnancy, had been in labour for many hours.

I first saw her at 6.30 and found the os fully dilated, the membranes ruptured, the head well down in the pelvis and in the O.L.A. position. The pains were feeble, and recurred no oftener than two in the hour. I saw her again at 11, and found the pains now recurring every two and a half minutes and lasting thirty seconds. The vagina and external parts were not so soft and relaxing as desirable, and the pains appeared to fail of their full effect, and I thought they could be rendered more effective if ergot were administered, and I gave 30 ♏ of the fluid extract at 11.30. In fifteen minutes ergot action was evident, and in another five minutes there were almost continuous alternating contractions and relaxations, but the most important circumstance was that although the head was now at the outlet it could not be born because of a tonic spasm of the *sphincter vaginæ* and of the *levator ani*, and each pain increased the spasm. Fortunately by 12.15 the effect of the ergot passed off and the pains ceased altogether. I now laid the woman on her back, and kneaded the abdomen and the uterus; the spasm became undone, and a living child was born at 12.30.

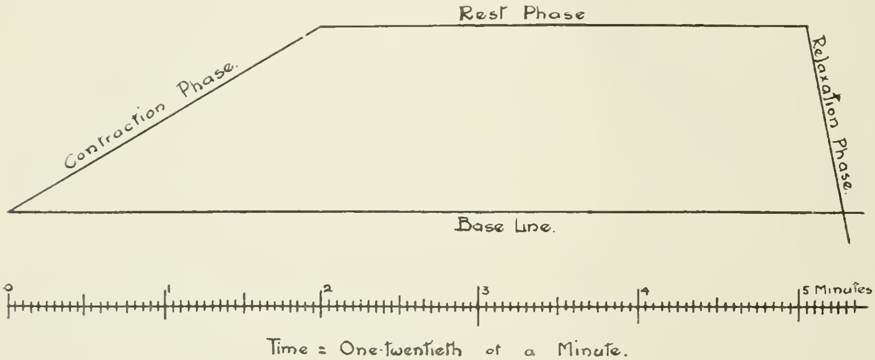


FIG. 11.

Extreme tonic contraction following a dose of ergot administered during labour. Compare with figs. 4, 5, 9, and 10.

(*b*) *Inco-ordinate Contraction of Uterus and Abdominal Muscles after Child Born.*—Woman, aged 30, in her fourth pregnancy, was delivered normally at 7.15. The uterus was felt to be in medium tonic contraction, but the after-birth was not expelled. At 7.20 I gave 30 ♏ (1·7 c.c.) of the liquid extract by the mouth. At 7.24, with the left hand on the abdomen, I could feel that ergot action had begun, and then followed rapid gross clonic contractions of the abdominal muscles, but while this was going on the uterus was felt to be still in medium tonic contraction. The clonic contractions of the abdominal muscles were repeated again and again without a single response on the part of the uterus. I inserted my hand and found the after-birth firmly grasped by the uterus, and had no difficulty in removing it. I have tried to represent the condition by a diagram (*see* fig. 12).

A consideration of the two foregoing histories raises the question: Does ergot act on the skeletal muscles? In (*a*) the *sphincter vaginæ* and muscles around the perineum before the drug was given were certainly not so yielding as is usual in a labour so far advanced, but as certainly did they pass into tonic spasm. Was this due to the ergot or was it reflex? In (*b*) the ergot appeared to make the abdominal muscles pass into clonic spasm while the uterus remained almost in the state it would have been had none been given. I am inclined to believe that ergot does act on the skeletal muscles through the cord.

(*c*) *Local Contractions in Uterine Muscle.*—Woman, aged 30, in her third pregnancy. Labour began at 4.30 in the morning, and I saw her at 10.30 and found all the parts moderately relaxed, the os in moderate dilatation and the head in the anterior position. I saw her again at 4.30, when the pains came on every twenty minutes, and little advance had been made since 10.30. I next saw her at 8.37, and although a little progress had been made there seemed to be little chance of a speedy delivery without aid. All seemed ready for ergot, and I administered the usual 30 ♏ of the fluid extract and there was a speedy response, the pains increasing in force, frequency and amplitude, but the interesting point is that during the periods when the uterus was free from the pains, small bundles of the uterine muscle were in tonic contraction,

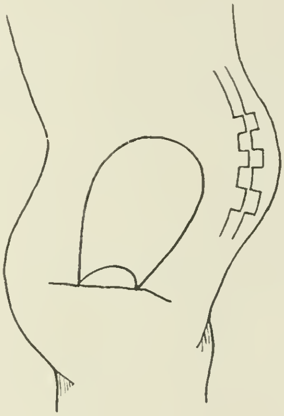


FIG. 12.

Shows how ergot may cause inco-ordinate action between the uterus and the abdominal muscles. In the figure the uterus is shown in medium tonic contraction, while the abdominal muscles are shown in coarse tonico-clonic contraction in which the clonic element predominates. Child born. After-birth in uterus.

sometimes bundles of one arc and sometimes of another. This went on for thirty minutes, when the energetic pains passed off and were replaced by pains in which the contractions were limited to areas. The contraction areas got smaller and smaller, and it was clear that delivery could not take place without some additional aid. The forceps were applied and a living child was speedily born.

Erysipelas of Buttocks and Thighs Fifteen Days after injection of $\frac{1}{50}$ gr. of Ergotoxine: Toneless Uterus during Labour.—A circumstance of great importance remains to be mentioned in one case. Whether it had anything to do with the ergotoxine or no I cannot say, but it is worthy of remark. A woman was delivered on the 7th and I never again handled her, because there was no need, for all went on as well as could be till the time about to be spoken of. The trained nurse who assisted me, washed, dressed and attended to the patient on the 7th and on the 8th, and then left. Another nurse came on the 9th, and the patient went on so well that I left her on the 15th. This second nurse attended till the 21st and she left the patient in perfect health, and the patient's mother came and did the little that was necessary. On the evening of the 22nd the patient was seized with shivering, and I was called to see her on the 23rd and was told that both her buttocks were red. The temperature was 102° F. (38·8° C.), the pulse rapid and the tongue coated, and she complained of pain and tingling on the parts affected. The buttocks were covered by a dark red sharply defined blush, having on its surface numerous small vesicles. The genitals were unaffected, but the redness extended down the thighs, chiefly behind. Day by day, despite active treatment, the redness extended till it reached both knees, and it did not get well till the end of five weeks and the whole of the epidermis was shed. Diligent search and inquiry failed to elicit any possible source of infection. The redness started near the point of injection (the part was well rubbed after injection) and spread downwards and over to the other side. There was no foul discharge, and as already mentioned the genitals were free from the redness, and appeared quite healthy.

It will have been observed that in the cases recorded in which clonic or tonic spasm followed, or in cases in which there was spasm of the muscles at the outlet, that these are minor disadvantages compared with the great advantage of having a drug which brings the head down to the outlet when kneading or application of forceps or other manœuvre speedily terminated the labour. As I have said, ergot does not kill the child when given in reasonable doses. I wish to emphasize the point that 30 fl. ♏ (1·7 c.c.) are a sufficient dose, and also that when this dose is much exceeded there may follow violent action, or what is more serious still, that the drug may inhibit or relax instead of stimulating or contracting the uterus in labour.

Post-partum Hæmorrhage.

Fortunately, since I began to make careful observations on the action of ergot, I have not had a genuine case, but if I had I should rely on rapidly acting agents, like ergotoxine or tyramine, which raise the arterial pressure, and on other measures to combat the collapse. (1) Liquid extract of ergot when given orally does not raise the arterial pressure; (2) *post-partum* hæmorrhage is not dependent on non-contraction of the uterus, and one often meets with a completely relaxed uterus after labour, in which there are none of the alarming symptoms of collapse which we associate with this condition, and often there is good contraction and yet bleeding is present; (3) the bleeding may be comparatively little and yet the symptoms of collapse are present, and sometimes there is very profuse bleeding with absence of collapse. It would seem as if the name is a misnomer, and a better designation would be either vasomotor collapse following labour or obstetrical shock.

Ergot in Abortions. Is it an Abortifacient?

Neither in ordinary nor in extraordinary doses will ergot induce abortion. No doubt the drug if continued over a long time in large doses so as to disturb the circulation and nervous system may cause abortion, but then so might digitalis, or even such an innocent medicine as Epsom salts, but here, as in the case of ergot, the abortion would only be part of a general toxic action. Indeed, ergot in medicinal doses will not alter the usual contractions and relaxations of a healthy pregnant uterus; it may act beneficially on bleedings at distant parts without disturbing pregnancy, and it may even stop bleedings from the uterus without disturbing the course of pregnancy, as the case about to be related will show.

Severe Bleeding at Third and Sixth Months. Ergot: no Abortion.—A woman, aged 33, whose first child was 2 years old, became pregnant for the second time; she suffered from sickness from the first, but at the end of the second month it was so severe that she had to keep her bed. At the end of the third month blood was passed by the vagina in large quantities, and one looked for a miscarriage. The os was not patent, nor were there symptoms of movements of the uterus, still these favourable signs did not allay my anxiety. Opium, rest and other means were employed to stop the bleeding, but without avail. I was adverse to employing ergot on account of the traditional belief that it caused abortion. The bleeding continued so severe that I felt certain there could be no hope of the fœtus coming to quickening, and I told the woman so, and I prescribed ergot in the usual manner, 20 ♏ doses (1·18 c.c.) of the fluid extract every three hours till some improvement took place. In twenty-four hours the bleeding completely stopped: quickening took place in due course. At the end of the sixth month the woman had another severe bleeding, and this was treated in the same way and with the same result. At the end of nine months the woman was delivered of a healthy boy, weighing 10 lb. (4,530·59 grm.).

While the ergot was being given careful examination was made by the hand to see if the movements of the uterus were in any way increased, such as one experiences when the drug is given in labour, but I am certain they were not. One may with safety say that if the fœtus has separated from its attachments, then ergot will increase the morbid contractions, and expel the contents much sooner than they would otherwise have been expelled. Ergot does not initiate uterine contractions, it only supplements them. One can quote experiments carried on in hundreds, or rather thousands, of cases during epidemics of ergotism; abortions were not commoner than in ordinary circumstances. More than this, ergot in large doses has been given during the menstrual flow to check bleedings in other parts, and the morbid bleeding has been checked while the physiological bleeding, that is, the menstrual flow, has gone on in its usual course, and the uterine movements have not been increased in any way. Some of these points will be supported by the two following cases.

The next most useful action of ergot, after its utility in labour, is without doubt in the removal of the separated but retained fœtus or membrane in abortions. Smellie (1697-1763), in his treatise on the "Theory and Practice of Midwifery,"[1] under the heading "Floodings—Abortion," says: "The os internum is kept open and irritated by the protruded part so as to occasion now and then a pain, which helps to separate and force down the others." This was written in 1741, before ergot had been introduced into British midwifery practice. The separated part sets up pathological contractions, and it is only when these have been initiated that ergot can act. As Smellie said, the protruding part (membrane, fœtus, or even afterbirth) acts as a peripheral, mechanical stimulant, setting up contractions, and in the large full-time uterus, with plenty of muscle ready to respond to a local stimulus, whereby strong contractions are set up, the offending body is likely to be soon expelled. In the small uterus of a two, three, or four months' pregnancy it is, however, different, for the muscle-fibre is so small in amount that a direct local stimulus may not be enough to induce contractions sufficiently powerful to empty the organ, and hence ergot is especially useful, for it acts not only directly on the muscle-fibres themselves, but also indirectly (where a peripheral stimulus cannot act) through the sympathetic system, and the ergot, by this double action, brings the foreign body sooner away and the uterus is saved work.

---

[1] New Sydenham Society, Lond., 1876.

Case I.—Woman, aged 36, who had gone to full term in seven previous pregnancies, became pregnant for the eighth time. At the end of the third month she began to bleed, without pain. This had gone on for three days before I saw her. On examination I found the os open and a mass protruding therefrom, and intermittent contractions could be detected. Here the uterus was trying to free itself of the offending dead material. I prescribed 15 ♏ (0·88 c.c.) of the fluid extract every four hours, and after the second dose had been given the whole of the uterine contents were expelled, and the woman experienced no further trouble. I sat down by the bedside and was able to satisfy myself that the force, amplitude, and frequency of the contractions were increased, not after the examination had been made, but thirty minutes after the dose of ergot had been administered.

What takes place when Ergot is given at the Third or Fourth Month: Difference from the Full-time Uterus in Labour.

I now endeavoured to find out in what way ergot acted in early abortions. First, there cannot be any doubt as to its usefulness, for cases in which abortion has taken place with continued bleedings at intervals, have cleared up when ergot has been administered. Secondly, as to the manner in which ergot acts. The contraction action is much slower in manifesting itself than in the full-time uterus, and takes from thirty to sixty minutes after exhibition; but then it lasts much longer, and a strong contraction action can be noted three hours after a dose of 15 ♏ of the liquid extract. The actual contraction lasts quite a long time, varying from ten minutes to half an hour, and the actual period during which relaxation is taking place may occupy a time equally long, and when the relaxation is complete there is often a large gush of bright blood, and with this the offending substances may be voided. The cycle may be repeated again and again till all foreign bodies are expelled, each succeeding cycle becoming shorter and shorter. If the foreign body be not removed in the earlier and longer cycles it may be detached by having its blood supply cut off in the improved tone of the uterus, which the ergot later on induces.

Case II.—Abortion at Third Month: Subsequent Intermittent Bleeding for Three Months.—Woman, aged 30, had four living children. Three years after the birth of the last child she missed three periods, but one day she was seized with pain in the abdomen and back with smart bleeding and the later expulsion of a substance "like an egg," and she thought all was well. But at frequent intervals during the next three months she had bleedings, and these weakened her so much and made her look so pale that her relatives became alarmed and I was called to see her. I examined and found an enlarged soft, boggy uterus with a patent os from which a bloody fluid was exuding in small amount. I prescribed 60 ♏ (3·5 c.c.) of the liquid extract in divided doses every twenty-four hours. At the end of a week a small piece of membrane was passed, the ergot was stopped, and the woman recovered without further trouble. The contractions and relaxations as detailed above.

I now go on to give short notes of one case typical of many to show that ergot often acts quite as well as curetting, so that in all like instances ergot should be tried before operation is resorted to.

Case III.—Expelled Abortion. Continued Intermittent Bleeding but no Passage of Membrane.—A woman, aged 32, who had borne five healthy children, missed three periods, when she had severe bleeding with the passage of a fœtus, for which no medical aid was sought. During the next three months she had irregular periods of intervals of five, two, three, and two weeks, with increasing, irregular, griping pains, breathlessness on exertion, and pallor of the face. At this stage I saw her. On examination I found the uterus soft and in size equal to a three months' pregnancy and undergoing irregular contractions and relaxations. The os was patent and a slightly disagreeable bloody fluid was issuing therefrom. The woman was sent to bed and prescribed 60 fl. ♏ (3·5 c.c.) of liquid extract of ergot in divided doses in the twenty-four hours. In three days the discharge had completely stopped and the dosage was reduced to half; in a week it was stopped and the patient allowed to get up. Coincident with this the uterus was reduced in size, the breathlessness had gone, and the colour of the face had improved. The discharge was carefully examined but no membrane was seen. At the end of three weeks she had another severe bleeding. The ergot was given at the rate of 30 fl. ♏ (1·7 c.c.) every twenty-four hours for a week and she was again well. After this the periods became regular, and the woman was restored to health. So far as could be detected no membrane was ever passed.

Here, probably, the membrane became broken down and was absorbed, but the uterus was left soft and flabby, and the ergot, by causing firm contraction of the muscular fibres, acted beneficially on the whole uterus. One can, in the same manner, account for the beneficial action of ergot in removing unhealthy membranous layers which line the uterus in many cases following an abortion. The ergot, by causing powerful contractions of the muscular coat, squeezes out fluids from all the coats, and thus the drug acts like a caustic by separating and expelling all substances foreign to the organ. It is by its contractor action that ergot expels many tumours; it deprives them of blood and they slough and are removed much in the same manner as if a caustic had been applied. Besides this, I am inclined to the belief that ergot has a specific tonic action on the uterine muscle.

PART II.—ACTION ON PARTS OTHER THAN THE UTERUS.

In bleedings from parts other than the uterus ergot is often of the highest service and I shall, as shortly as possible, set down the clinical evidence I have collected under various headings.

(1) *Alimentary Tract.*

I believe there is almost universal agreement on this point, hence I do not propose to take up time quoting cases.

(2) *Urinary Bladder.—Catarrhal Inflammation.*

A man, aged 21, began to complain of great pain over the region of the bladder, aggravated on passing urine, which was almost alkaline in reaction, flaky, and soon became ammoniacal. Benzoic acid internally and irrigation with boric lotion and demulcent drinks failed to remove the condition. A surgeon sounded him for stone with a negative result. In two days he passed blood-stained urine, and next day bright red blood containing epithelial cells from the bladder wall. He was placed on ergot (liquid extract, 15 ♏ every three hours) with 5 ♏ of tincture of opium. In twenty-four hours there was a diminution of the blood, and in forty-eight hours the bleeding had almost ceased. Twenty-four hours later all blood was absent, and the patient made an uninterrupted recovery. I had him under review for three years, and he had no return.

In other somewhat similar cases I have found it equally successful.

(3) *On the Arterial Pressure: Ergot given orally, practically no change.*

Within the past five years it has often been stated that ergot must be contra-indicated in hæmoptysis because it raises the arterial pressure. This is entirely contrary to my experience, for in medicinal doses I have never been able to raise the pressure by means of the drug. I quote one case as follows:—

A woman, aged 27, whom I have had under observation for some years, suffering from extreme Graves's disease or exophthalmic goitre, progressing towards recovery, has a *moderately high* systolic pressure ranging from 130 mm. to 150 mm., but with a *very low* diastolic pressure ranging from 60 to 70 mm. (observations made with a modified Riva-Rocci sphygmomanometer with broad armlet). The pulse ranges from 120 to 140.

	Systolic	Diastolic	
(1) Observation for one week before ergot was given	150	65	
(2) Observation for one week, 60 ♏ (3·5 c.c.) liquid extract given daily by mouth	150	65	No change
(3) Observation for second week, same dose of ergot given daily by mouth	130	65	Systolic 20 lower; diastolic same.
(4) Observation for another week; ergot stopped	130	70	No change from last week.
(5) Observation for another week; ergot stopped	125	65	Much as last week

The practical outcome is that the average of the systolic and diastolic pressures remains the same, but if the ergot did anything it lowered the systolic pressure.

(4) *Ergot in Pulmonary Hæmorrhage.*

The newest teaching is that ergot and its active principle should not be employed in pulmonary hæmorrhage, nor in cerebral hæmorrhage, because the blood-vessels supplying these parts have no or only a rudimentary sympathetic nerve supply, hence ergot by raising the general arterial blood-pressure will do harm by dilating the small arteries of the areas named, and so help to increase the mischief it is intended to remove. One might meet this statement with one equally strong, that the vessels of the areas named are entirely passive, and neither contract nor relax of themselves, but are simply ruled by the state of the vessels on their central side; if the vessels at a distance transmit less blood, less blood will reach the terminal vessels. Then there is the probable action of ergot on the blood tissue whereby a clot may the more readily form at the oozing points. Further, it is not wise to reason directly from a physiological experiment carried on in the laboratory to the morbid state of the sick room, more especially when experience gained in the sick room does not tell in favour of the laboratory teaching, for after all drugs may have a different action in diseased states of the tissues than in the normal state. Diseased tissues have different electrical reactions as well as possible different chemical combining qualities from normal tissues. Further, as we have just seen, the galenical preparations of ergot when given orally, at least do not raise the arterial tension. From the standpoint of the presence or absence of a vasomotor nerve supply, allowing for the moment that this is important, it is true that most histologists hold to the view that the pulmonary vessels have none or only a rudimentary nerve supply, but are they really certain of this? All they should lay claim to is, that such a supply has not been demonstrated. Besides, some histologists lay claim to have found such a nerve supply. Then there is the argument of experience. Before men knew how or on what tissue ergot acted, the drug was employed in bleeding from the pulmonary tract and with manifest benefit. I am not unmindful of the facts that Dixon and his fellow-worker found that the pulmonary arterioles dilated when they perfused them with ergot, but this is different from administering the drug by the mouth. Lastly, there has to be taken into account the fact that Dale found that β-iminazolethylamine, a constituent of the galenicals, lowered the arterial pressure as opposed to the other chief amine, so that the pressor and depressor action of the galenical preparations of ergot may be equally balanced.

Case I: Hæmoptysis.—A woman, aged 47, whose brother and sister died of phthisis, began to spit bright red blood in small quantities, but this increased till soon she had spit one pint (568 c.c.). The temperature was slightly raised and the pulse was 80. On examination soft râles were heard on the right side from the clavicle down to the third rib. The heart sounds were normal. She was prescribed 10 ♏ (0·59 c.c.) of the fluid extract every eight hours with the other usual hygienic means. After the first dose the profuse expectoration ceased, but the expectoration remained somewhat profuse and tinged with blood for four days, when the dose of ergot was doubled and given every four hours, and in a week from the onset of the bleeding the woman had taken 1 fl. oz. of the liquid extract (28 c.c.) and the bleeding was completely stopped. The woman requested that she might continue the ergot for some time longer, and she returned in a week to report herself, and I found that within this time she had taken another fluid ounce and had had no more bleeding. I told her to stop the ergot and I prescribed Begbie's mixture for her. I saw her at the end of another week and found that there had been no more bleeding and the area over which the râles were heard had now diminished.

Case II: Hæmoptysis; Tuberculous.—History: A man, aged 23, was seen for the first time in October, 1906. He had a tuberculous history, and he himself had a latent angular curvature. Soft mucous râles could be heart at his right apex. In December he spat up one pint or more of bright red blood, and thereafter there went on for some hours the coughing up of mouthfuls of blood at intervals of half an hour or thereby, when he was prescribed, by one of my friends, 20 ♏ of the ammoniated tincture of ergot, equal to 5 gr. (0·324 grm.) of crude ergot, every four hours. The bleeding gradually stopped, till in twenty-four hours not a trace remained. He continued the medicine till he took altogether, I found, 14 fl. dr. of the tincture, equal to 220 gr. (14·26 grm.) of crude ergot. In October, 1908, he had another bleeding, this time losing more than half a pint, followed by the spitting up of mouthfuls at intervals. He insisted on having the same medicine, and all traces of bleeding had stopped well within a week, that is to say, by that time not a trace could be found in the sputum, and blood as blood was not spat after the second dose of medicine had been taken. Despite all remonstrances to the contrary, the man continued the ergot for three weeks after he was quite well. In April, 1909, the man had another bleeding, and in this instance, as in the others, the area of the lung from which the blood came could be mapped out by auscultation. He repeated his old prescription, but it failed to act, and this was thought to be owing to inertness on the part of the ergot preparation; he was prescribed 10 ♏ (0·592 c.c.) of the fluid extract, and it acted at once and the bleeding stopped. He continued the liquid extract for three weeks, and I noted that he took altogether 1½ fl. oz. in that time (42·6 c.c.). On November 30, 1910, he had another bleeding, this time, however, only losing half a pint in all. The liquid extract was again prescribed, in the same doses as in the hæmorrhage of 1909. In all the bleedings the characteristic bright red blood, intermixed with frothy sputum, was remarked, showing that the seat of leakage was the terminal vessels in the lung. The man was seen on May 3, 1911, and he has had no more bleedings since the one just recorded. He presents all the appearances of one suffering from chronic pulmonary tuberculosis. I believe I am right in claiming that, in his case at least, there is sufficient evidence to show that a preparation of ergot, toxic enough to cause powerful contractions of the uterus in labour, has produced none of the disasters sometimes feared.

DISCUSSION.

Dr. H. H. DALE said that the paper had been of the greatest interest, and gave a clear indication that Dr. Sharp had been doing what many had been wishing for years that some one would do—namely, making clinical observations as nearly as possible in the manner in which laboratory experiments were conducted. He had been making observations on the action of ergot on the uterus, which were as nearly quantitative as conditions allowed. That was the kind of observation specially needed. Dr. Sharp asked him for his opinion as to what was a good ergot. That was a difficult question, and one which clinicians must help to settle. At least three active principles, highly potent, were present in ergot, including the specific alkaloid, ergotoxine, and two bases not peculiar to ergot, but formed from any protein by putrefaction, being derived from amino-acids by the action of putrefactive organisms. Was ergot good in itself? And should one speak of good ergot or a good ergot extract? There was no doubt that some extracts of ergot were made in such a way that the quantity of one or other of these bases was greatly increased, owing to the putrefactive changes taking place in the process of manufacture of the extract. Therefore the question must remain unsettled until there was more information available, even more definite than that which Dr. Sharp had given, as to which one wanted—ergotoxine or one of the putrefaction bases. Dr. Sharp said that ergotoxine was useful by itself. He was glad to hear that, because some years ago he was rash enough to prophesy that ergotoxine should have therapeutic value from laboratory results. He had waited for some one to give clear clinical support to that prophecy. He was prepared to hear that p-hydroxyphenylethylamine had comparatively little effect on the uterus. Its effect varied considerably on the uteri of animals in the laboratory, in accordance with the functional condition of the uterus. On the cat's uterus, in the non-pregnant condition, relaxation was produced: but if the uterus was pregnant it contracted in response to the base. He had seen instances, late in pregnancy, when the reaction seemed reversed again. The third active principle Dr. Sharp had not had the opportunity of investigating by itself; but he would be glad to put Dr. Sharp in the way of doing so, because in iminazolethylamine one had a most powerful uterine stimulant. Its action on the uterine muscle was very intense. The liquid extract of ergot contained very little ergotoxine. The contrast between the prolonged action of the liquid extract and the more rapid and evanescent ection of ergotoxine, as described by Dr. Sharp, probably indicated the difference between the action of the base iminazolethylamine, which occurred in all liquid extracts, and that of ergotoxine. Another point alluded to was that ergot did not initiate pains or cause abortion. That agreed with his experience in the laboratory. There seemed to be very few drugs which would produce abortion. He had tried with the most powerful physiological uterine stimulants, as distinguished from generally toxic ones, including suprerenal extracts and pituitary body, also with ergot in all its forms, yet he had not succeeded unless he gave sufficient to cause poisoning in the parent and death of the fœtus. He had not produced the initiation of normal labour by a drug. With regard to hæmoptysis, when clinical observers said that hæmoptysis could be stopped by ergot, and laboratory observers said hæmoptysis would be aggravated by ergot, it was because laboratory observation was incomplete. Iminazolethylamine lowered the systemic blood-pressure by vaso-dilatation, and constricted the branches of the pulmonary artery, and thus produced what ought to be ideal conditions for checking hæmoptysis.

Dr. BEZLY THORNE said he was struck by Dr. Sharp's observations that liquid extract of ergot produced a contraction of the sphincter vaginæ in a certain number of cases. He would ask whether Dr. Sharp's observations led him to a conclusion as to what was the range of action of ergot on muscular tissue. If it did not act on the muscular tissues of arteries and blood-vessels generally in raising tone, and did act on the sphincter vaginæ, had Dr. Sharp formed an opinion as to what was its range of action?

The PRESIDENT (Professor A. R. Cushny) congratulated Dr. Sharp on his observations, which were of the character the Section desired to encourage. The clinical action of ergot was the sort of thing one would expect a practitioner to investigate, and the wonder was that more had not been done already.

Dr. SHARP, in reply, said that one and half hours before delivery, when the woman had not been given anything, in an experiment lasting 27 minutes, the average rate of the pains was 2·6 minutes, and the average length 44 seconds. Next was an experiment lasting 30 minutes, with tyramine; the average time was 2·7 minutes while the pain lasted, on the average $41\frac{1}{2}$ seconds. Ergot was given at 9.27, and at 9.30 she had a pain lasting 40 seconds. At $9.32\frac{1}{2}$ there was a pain lasting 50 seconds. At 9.35 the uterus was very much firmer. At 9.38 there was a pain lasting 125 seconds. The pains were increased in time and amplitude by the ergot. At 9.42 the pains were continuous until the head was sent down ten minutes later on to the perineum, and the child was born 33 minutes after the liquid extract was given. It was said that ergot did not act on the skeletal muscles; but, be that as it may, ergot made the uterus extremely irritable: even a slight examination of the vagina would cause increased pains. Previously to Dr. Dale's work on the subject, it was thought that ergot contained two substances, one of which inhibited the uterus, and the other which contracted it; but now it was known that in small doses it would stimulate, and in large doses it would inhibit. The first action of poisons was to stimulate, and the later action was to kill. He had employed the fresh infusion in 10-dr. doses. The liquid extract he used had been found to keep twelve months.

Therapeutical and Pharmacological Section.

May 16, 1911.

Professor A. R. Cushny, F.R.S., President of the Section, in the Chair.

An Investigation of the Action of Aconite on the Pulse-rate.

By Frederick W. Price, M.D.[1]

Mr. President,—In your striking and very suggestive Presidential Address you referred to the lamentable lack of accurately observed and recorded results of the effects of drugs in man, the reason being that the physician is content with what Lord Kelvin described as "knowledge of a meagre and unsatisfactory kind," since his inquiries are limited to the most general features, largely depending upon the subjective statements of the patients, and without trying to accurately estimate other factors than drugs. You referred to the fact that this absence of accurate clinical records prevents the findings of the experimental pharmacologist being substantiated, and that this is seriously hindering the advance of therapeutics. You made an eloquent plea that by the adoption of the methods of exact analysis, practical therapeutics should be brought to the higher state of cultivation which has been reached by experimental science.

This short paper is the outcome of an attempt to work on the lines you suggested. It embodies the results of a series of investigations dealing with the simple question of the action of aconite on the pulse-rate in the sick-bed. There was no inquiry into the other actions of the drug. The object of the investigation was to see if aconite could be substituted for digitalis, strophanthus or squill, without the attending disadvantages of these drugs, such as loss of appetite, nausea, vomiting, diarrhœa, or headache; and to substantiate or disprove the prevailing belief both among experimental pharmacologists and practical therapeutists, that the internal administration of aconite directly diminishes the pulse-rate. Incidentally, however, the blood-pressure was recorded in some of the cases, and the subjective phenomena were noted in almost all. As far as my observations have gone, there have been no results to support the current view. Indeed, what action, if any, the drug has on the pulse-rate is to increase it.

---

[1] From the Cardiac Wards of Mount Vernon Hospital.

It was Baron Störck who, in the year 1762, first introduced aconite into practice. He stated it was a diaphoretic and diuretic, and employed it in chronic rheumatism, gout, paralysis, exostosis, and scirrhus. His observations led to its employment in other diseases, such as syphilis, goitre, glandular swellings, convulsive disorders, intermittent fever, ulceration, amaurosis, phthisis, dropsies, some skin diseases, uterine affections, and hypertrophy of the heart, so that the drug quickly gained a great reputation, especially among German and Italian physicians, as a narcotic (anodyne), sedative, sudorific, resolvent, and diuretic. Later experience, however, did not confirm the earlier favourable accounts, and within a few decades the drug had almost entirely fallen into disuse.

I will deal very briefly and with the main steps only of the further history of the drug, and mostly only in so far as its action on the circulation is concerned.

In 1796 and later Hahnemann experimented with the drug upon himself and his disciples. He described it as a specific in those acute inflammatory fevers in which allopathy used to recommend venesection. He seldom used it in such fevers as typhoid, but in measles and scarlet fever, when not of an asthenic type.

In 1811, Brodie was the first in this country to examine into the effects of the drug on animals. He inferred that aconite and some other drugs act as a poison, simply by destroying the functions of the brain.

About the year 1835, Lombard stated that the drug was beneficial in diseases of the heart attended by over-action of the organ, because of its sedative properties. It was he who also first prominently brought the drug into use in the treatment of rheumatic fever.

In the *Lancet* of March 25, 1837,[1] Sherwin published an account of a case of a female who had been poisoned by the alcoholic tincture of the root, in whom five hours after the poison the pulse was 58 per minute, and intermittent. The woman eventually recovered.

On March 31, 1837, at the London Hospital, Pereira introduced a small quantity of the soft alcholic extract of the root into the peritoneal cavity of a strong dog, which had been kept fasting for some hours. He says that at one period afterwards the action of the heart was slower than usual, and the first and second sounds unusually distinct. Subsequently the circulation was quickened. The animal died.

---

[1] *Lancet*, 1836-37, ii, p. 13.

In 1845, Alexander Fleming published a very important treatise entitled "An Inquiry into the Physiological and Medicinal Properties of the Aconitum Napellus," which was an Inaugural Dissertation which obtained from the Senatus Academicus of Edinburgh a Gold Medal at the graduation of 1844. It was based on a series of experiments on animals, and on observations of the physiological and therapeutic action of aconite on man carried on in the Royal Infirmary of Edinburgh during his residence there. He also made observations on vegetables. In regard to man, he states that he had every facility for hourly and continual observations of its effects, which were noted with scrupulous care and accuracy. He came to the conclusion that the advantages resulting from the employment of the drug were great, and he expressed his strong hope that its use would be greatly extended. I will deal only with his observations on the pulse-rate.

First of all, in regard to animals, he believed that a sedative action is exerted on the heart, and that the pulse becomes rapidly weaker and less frequent. He based this particular belief on two experiments: (1) $\frac{1}{4}$ gr. aconitina was introduced into the stomach of a dog. In twenty minutes the pulse was 144, regular, and of good strength. In an hour it had fallen to 68 and was weak and very irregular. In two and a half hours it was 76, weaker, and more irregular. Death ensued in four hours. (2) $\frac{1}{2}$ gr. aconitina was introduced into the stomach of a dog, which had not tasted food for twenty-four hours. At first the pulse was 200 per minute, in twenty-two minutes it was 144, and in forty minutes it was 92, and very weak. It will be noted that in both cases the doses were large, and that it is stated that in the first case the pulse became irregular, while in the second case it was not noted whether the pulse was regular or not.

In regard to man, he came to the conclusion that with small or medicinal doses there were four degrees of operation.

First degree: After the exhibition of 5 ♏ of the tincture, in three hours and a half the pulse is diminished in strength, and in another hour it becomes less frequent. Thus a pulse which in the normal state beats 72 in the minute, will by that time have fallen to about 64. Second degree: Should a dose of 10 ♏ be given at first, or the first dose of 5 ♏ be succeeded in two hours by another of equal amount, in an hour and a half the pulse will probably have fallen to about 56 beats in the minute, and become smaller and weaker than before, still maintaining, however, perfect regularity. Third degree: On the administration of 5 ♏ more, two hours subsequently to the last dose, the pulse is reduced still further in strength and frequency, perhaps falling to 40 or even 36 beats per minute, but still maintaining its regularity. More frequently, however, it rises to 70 or 80, and becomes small, weak, and probably more or less irregular. Fourth degree: On the administration of a fourth dose of 5 ♏, two hours after the third, the pulse becomes still smaller, weaker, and more irregular. When the action of the drug is carried to a fatal extent the pulse becomes imperceptible, both at the wrist and heart.

He believed that aconite exerts a direct sedative influence on the vascular system, reducing, more or less, according to the dose administered, the strength, volume, and, in the first instance, the frequency of the pulse. The diminution in frequency varies much, *caeteris paribus*, with the individual, the pulse, in some cases, not falling below 60, and in others sinking so low as 48, 40, and even 36. As a general rule it maintains its regularity as long as it continues to become slower. On the sedative action being carried further it rises in frequency, becomes irregular and intermittent, and still smaller and weaker. Its character is then, frequently, most remarkable. It may present, first, irregularity in point of strength and volume. An ordinary beat of moderate size may alternate with a small, imperceptible pulsation, such as is observed in some cases of heart disease; the pulse, at the same time, not presenting any irregularity in point of rhythm. Secondly, it may be simply intermittent, the pulsations not differing from each other in strength and volume. One of these intermissions may last for several seconds. Thirdly, more frequently the pulse is irregular, both in point of rhythm and strength. Thus a pulse which, for three, four, or more beats, is weak, but regular, of moderate size, and beating at the rate of 36 or 40 per minute, may suddenly become much smaller and weaker, and rise to 120. After fifteen or twenty pulsations more, its character may again change, and for the next few seconds a full and soft beat may alternate with a nearly imperceptible one. In a short time it may become intermittent, only to resume in another minute. The pulse may continue in this anomalous condition for one or two hours after every other symptom of the action of the drug has disappeared. It sometimes presents, in a very marked degree, the character described by the term *labouring*, in which case its beats suggest very forcibly the idea that the heart is suffering from some depressing influence. Each contraction appears to be performed slowly and with difficulty, and the artery may positively be felt distending tardily under the finger. In such cases the cardiac sounds, as heard by the stethoscope, are weak and indistinct. When, on the other hand, the pulse is quick, irregular, and intermittent, they are confused and interrupted, as if many of the contractions of the heart were imperfectly performed.

To prove beyond doubt that the sedative effect exerted on the circulation is direct, he frequently examined the pulse, every five or ten minutes, for one or two hours after the exhibition of a dose. In general, the first perceptible change was a diminution of strength, and, in a few cases, of frequency; but in no instance did he observe the slightest tendency to primary excitement. He regarded the rising of the pulse, after it has fallen to a certain standard, merely as an indication of increasing debility, and as the effect of effort on the part of the heart to compensate for diminished power by increased frequency; while the irregularity and intermissions which follow are evidently the result of the inability of the organ to maintain steadily this augmented frequency.

If two or three doses have been given, the heart recovers itself in a period varying from twelve to twenty-four hours. If, on the other hand, the administration of the drug has been continued for a week or more, several days elapse before it does so; shortly after which event it is generally observed that the pulse becomes somewhat quicker and fuller than natural. In short, a slight degree of reaction is established—an occurrence which we know almost invariably succeeds depression of the circulation from other causes, as loss of blood, cold, shock to the nervous system, &c.

The effect of change of posture on the pulse of individuals, influenced by aconite, may be stated as follows: Supposing the pulse, under the first degree of operation, to be 64 while the patient is in bed, it will rise, on his assuming the erect posture, to 70 or 80, becoming at the same time smaller and weaker. Should the second degree of operation have been induced, lowering it to 56, it will rise, on a similar change of posture being made, to 80 or 90, become much smaller and weaker, and perhaps present an irregular character. If, in the third degree of operation, the patient attempt to rise, he will probably fall back in a fainting state. The influence of change of position thus seems to increase in the ratio of the depression which has been induced.

The practical inferences which Dr. Fleming deduced from a consideration of the action of aconite on the circulation were as follows:—

(1) That it is a powerful antiphlogistic.

(2) That it is calculated to be of great value in all cases where there is inordinate activity of the circulation.

(3) That it is contra-indicated when there is obvious mechanical impediment to the passage of the blood, particularly through the heart or lungs. It is requisite, therefore, in every case to ascertain that no such obstruction exists before commencing its use.

(4) That it is contra-indicated whenever there is irritability of the circulation, with great diminution of power, such as occurs after severe venous hæmorrhage.

We find that the opinions in regard to the action of the drug in man were based on his observations of five cases of acute rheumatism, a case of chronic rheumatism, a case of lumbago, a case of abdominal aneurysm, a case of erysipelas of the foot, and a case of sprained ankle. After a careful perusal of the records of these cases, his conclusions are, in my opinion, in large measure unwarrantable. As an example, it is merely necessary to state that in regard to the cases of rheumatism, the exact temperature was not taken (it was merely stated whether the skin was hot, warm, or cold), and therefore the normal fluctuation of the pulse-rate with the temperature was not sufficiently taken into account; the pulse-rate was taken only once a day in the first four cases; there appears to have been a close relationship between the pulse-rate and the varying conditions of the symptoms and physical signs of the joints; there were not always controls; and the pulse was never counted at the apex beat.

Pereira, in the third edition of his book published in 1853, stated that Redfern communicated to him a case of poisoning by tincture of aconite, which was probably that known as Fleming's tincture. It was the case of a woman, aged 21, who took five drops three times a day for two days, which was increased to six drops every two hours on the third day, when after the second dose symptoms of poisoning set in. The pulse, which was at first 150 and fell to between 50 and 60 in a few minutes, was so small and compressible as scarcely to be felt at the wrist. The woman recovered.

In 1855, M. Scroff stated that the drug exerts a strongly depressive influence on the action of the heart and large vessels, which was observed either immediately or after a short acceleration of the heart's action—which depressing action persists.

Pereira, in the fourth edition of his book published in 1857, stated that the heart appears to be weakened or paralysed by aconite, and a state approaching to asphyxia is produced.

In 1866, Aschscharumow published his experimental researches upon the physiological effects of aconite both upon cold- and warm-blooded animals.[1] He stated that full doses of aconite in all classes of animals are followed first of all by a lowering of the frequency and force of the heart's action, due to a stimulation of the cardiac inhibitory nerves in the medulla oblongata, followed by increased rapidity and irregularity due to exhaustion and paralysis of the nerves.

In 1872, Boehm and Wartmann, from experiments, came to the conclusion that the drug paralyses the terminations of the vagus.

In a dissertation on aconite, C. Ewers, in 1873, stated that aconite slowed the pulse by stimulating the vagus roots.

In 1874, A. G. Burness and F. F. Mavor experimented on horses. The conclusion was formed that the drug was indicated in all febrile states not depending on blood-poisoning or local inflammation, and in hypertrophy of the heart and vascular excitement.

In 1879, Ringer and Murrell, from experiments on frogs, were led to conclude that aconitina paralyses all nitrogenous tissues, and suggested that it affects all the structures of the heart—first its ganglia, next its nerves, and lastly its muscular substance.

Ringer, through the wide circulation of the various editions of his book, had a large share in popularizing the use of this drug in most acute inflammations, because of its supposed action on the circulatory system. In his undoubted opinion, moderate doses of the drug greatly reduce the number of the heart-beats, even to 40 or 36 in a minute; but after a larger and dangerous dose the pulse beats faster, and may become irregular. Sometimes even a small quantity excites irregular action. Whether increased or lessened in frequency, the pulse always loses strength, showing retardation of the circulation. He believed that, if given sufficiently early and in constantly repeated minute doses, it can cut short and limit the intensity of most acute inflammations, especially if the inflammation is not extensive, or not very severe. He had great faith in it in the treatment of tonsillitis and pericarditis. It was he who introduced the method of administering the drug in very small doses, frequently repeated.

In regard to my investigation the cases chosen were: Three cases of mitral stenosis, two with completely irregular rhythm (that is, with auricular fibrillation) and one with normal rhythm; one case of double aortic disease and mitral incompetence, with normal rhythm; two cases of pulmonary tuberculosis, both with normal rhythm, one with slight and the other with a fair degree of pyrexia; seven cases of scarlet fever, two in adults and five in children, all with normal rhythm; one case of malignant endocarditis, with normal rhythm and a high temperature. In all there was increased rapidity of the pulse.

---

[1] *Arch. f. Anat. Physiol. u. wissensch. Med.*, Leipz., 1866, pp. 255-283.

Plan of investigation: All the patients were kept in bed. The drug was usually given three times a day. The hours of administration, in all excepting the case of malignant endocarditis, were at nine, one, and five o'clock. The pulse was counted nine times a day in all excepting the case of malignant endocarditis—viz., a few moments before each dose, one hour after, and three hours after. In some cases, indeed in most, the pulse-rate was not only taken on the days of administration, but at exactly corresponding hours for some days before, or after, or both before and after the days the patient was taking the drug, in order to compare the rates of identically the same hours when the patient was taking the drug and when he was not. The blood-pressure was taken at the same time as the pulse-rate in the two heart cases with normal rhythm, in one of the heart cases with completely irregular rhythm, and the cases of pulmonary tuberculosis. I do not attach any certain value to the blood-pressure readings in the case with irregular rhythm. In some cases, on the completion of a course of the drug, the pulse-rate was counted by means of the ink polygraph every two minutes for an hour or so after the last dose. Whenever there was pyrexia the temperature was taken at the same times as the pulse-rate. The subjective phenomena were noted whenever present.

Preparations used: At the commencement of the investigations samples of tincture of aconite obtained from several firms of chemists were used. No action at all in the sick-bed was observed, and on being tested at the University College Pharmacological Laboratory these specimens were found to be inert. Afterwards specimens of aconitina prepared in the above-named laboratory were used, and, with the exception of one case, my findings are concerned only with these.

CASES.

Case I.—A clerk, aged 36, was admitted to Mount Vernon Hospital on August 4, 1910, complaining of shortness of breath and pain in the præcordia. There was a history of four attacks of rheumatic fever.

The apex beat was within the nipple line, and there was a mitral presystolic bruit present. The ink polygraph revealed the presence of an auricular wave in the jugular tracing. The pulse was regular, varied from 74 to 110, being usually about 90 per minute, and the blood-pressure ranged from 110 to 122.

On August 18, that is after a fortnight without any drug, patient was put on $\frac{1}{600}$ gr. aconitina, twice a day for two days and three times a day for one day. The medicine was then stopped for two days, and recommenced on August 23. For two days the dose was $\frac{1}{600}$ gr., for three days $\frac{1}{450}$ gr., for five days $\frac{1}{300}$ gr., and for four days $\frac{1}{200}$ gr. The pulse-rate and blood-pressure were taken for four days after the drug was stopped, at the same hours as when the drug was being taken. From the very commencement of the administration of the drug there were thirst, dryness and burning of the throat, feelings of prickling and numbness of the extremities, and within a few days there was præcordial pain and "thumping" of the heart. Later there was vomiting. The subjective phenomena continued for some days after the drug was stopped.

An analysis of the readings of the pulse-rate and blood-pressure, which I may remind you were taken nine times a day, shows that while the patient was taking the drug the average daily pulse-rate was not diminished, while the blood-pressure was, if anything, slightly increased.

Taking the averages of the hour of administration, of one hour afterwards and three hours afterwards, there was no diminution. A study of the reading of a continuous observation for an hour by means of the polygraph and mercurial manometer reveals the same.

On September 10, patient was put on to 15 ♏ digitalis, four times a day, till September 16, when he had taken 5½ dr. On September 15 he vomited, but there was no effect on the heart-rate or blood-pressure. On September 16 the pulse was occasionally intermittent, which simultaneous tracings of the jugular and radial showed to be due to a partial heart-block. It was not detected after that date.

Case II.—A barman, aged 22, was admitted to Mount Vernon Hospital on August 19, 1910, complaining of weakness, shortness of breath, and loss of power in the left arm and leg. There was a history of chorea at the age of 6 and 8, and of typhoid fever in 1897; none of rheumatism. There was a history pointing to cerebral embolism on June 12. On admission there was left-sided partial hemiplegia, dyspnœa on the slightest exertion, the apex beat was in the fifth space, $5\frac{5}{8}$ in. from the middle line, there was an apical diastolic thrill, and a systolic and occasionally a diastolic bruit. The ink polygraph revealed the absence of an auricular wave in the jugular tracing. The pulse was completely irregular, and varied from 100 to 110 beats per minute.

On August 23 patient was put on to $\frac{1}{600}$ gr. aconitina for two days, and then to $\frac{1}{450}$ gr. for three days, and during the whole of this time there was dryness of the throat. The dose was then increased to $\frac{1}{300}$ gr. for five days, in the middle of which there was frontal headache and vomiting for one night. So far, unfortunately, the pulse-rate had been taken at the wrist, and as it was completely irregular, I have not considered its countings absolutely reliable. Apparently, however, there was no diminition of its rate. The pulse was then counted at the apex beat, and the dose was increased to $\frac{1}{200}$ gr., and continued for five days. During this time there were much the same subjective phenomena as noted in the last patient, excepting that there was frontal headache and no vomiting. They ceased the day after the medicine was stopped.

An analysis of the readings shows that the average daily pulse-rate was not diminished; indeed, if anything, it was higher than formerly; the blood-pressure was unaltered.

Taking the averages of the hour of administration, of one hour afterwards, and of three hours afterwards, there seem to have been drops of eight beats and 5 mm. of mercury one hour after, and of seven beats and 3 mm. of mercury after three hours. On the other hand, a study of the reading of a continuous observation, forty minutes, by means of the polygraph shows the pulse to have remained the same.

After a lapse of two full days the patient was placed on 15 ♏ tincture of digitalis four times a day. This dosage was continued for a week. On that day the patient vomited. The average pulse was then 70—that is, a drop of about 50 beats per minute since before the digitalis was commenced. It fell to 65 the next day. The patient was now much better, and could breathe with greater comfort after exertion.

Case III.—A young woman, aged 20, was admitted to Mount Vernon Hospital on June 30, 1910, complaining of pain in the left chest and shortness of breath. There was a history of several attacks of rheumatic fever. On admission there was orthopnœa, the apex beat was in the sixth space, forcible, 6 in. from the middle line, and there was a double aortic and mitral systolic bruit. The ink polygraph revealed the presence of an auricular wave in the jugular tracing. The pulse was normal in rhythm, collapsing in type, and during the first week, when the patient was not taking any medicine, its rate varied from 96 to 128, the average being 114.

Patient was then put on to 10 ♏ tincture of aconite every two hours during the day, and usually twice during the night, when the average pulse-rate was 107, and the blood-pressure increased from 132 to 144. After nine days the dose was increased to 15 ♏, and continued for six days, during which the average pulse-rate was 112, and the blood-pressure increased from 144 to 154. There were no subjective phenomena. The day after the drug was discontinued the average pulse-rate slightly dropped.

About four weeks later patient was given $\frac{1}{600}$ gr. aconitina three times a day for two days, and no change was detected in the heart. Five days later the patient was given $\frac{1}{1200}$ gr. three times a day for two days, then $\frac{1}{600}$ gr. for three days, then $\frac{1}{450}$ gr. for four days, and then $\frac{1}{300}$ gr. for three days. The drug was then stopped, but the pulse-rate and blood-pressure were taken at the same hours for four days more. During the whole time the drug was administered there was a feeling of "burning" of throat and nausea. When the dose was $\frac{1}{450}$ gr. there were a "choking" feeling and a consciousness of the heart beating rapidly and forcibly. The subjective phenomena passed away the day after the drug was stopped. An analysis of the readings shows that the average daily pulse-rate was not diminished, and the blood-pressure increased.

Taking the averages of the hour of administration, of one hour afterwards and of three hours afterwards, there is nothing definite to note. A continuous observation for one hour after taking aconitina compared with a continuous observation for forty-five minutes without taking the drug reveals very much the same in both instances.

During the stay in the hospital patient had two courses of large doses of tincture of digitalis until vomiting supervened, and there was no appreciable result on the heart-rate or blood-pressure.

Case IV.—A dressmaker, aged 35, was admitted to the Mount Vernon Hospital on July 26, 1910, complaining of shortness of breath, throbbing of the heart, and præcordial pain. There was a history of scarlet fever; none of rheumatism. On admission the patient was orthopnœic, the apex beat was in the fifth space, $5\frac{1}{4}$ in. from the middle line, where a diastolic bruit was audible, and the liver was palpable for three fingerbreadths below the costal margin. The ink polygraph revealed the absence of an auricular wave in the jugular tracing. The pulse was completely irregular, and the patient being in great distress, she was put on to 15 ♏ tincture of digitalis three times a day. After taking $3\frac{1}{2}$ dr., as it caused headache and nausea, it was discontinued. By this time the patient's breathing was greatly relieved, and the pulse-rate fell from 125 to 61 beats per minute. (No digitalis on August 3.)

Sixteen days later (August 18) patient was put on to $\frac{1}{600}$ gr. aconitina twice a day. She seemed to have a remarkable susceptibility to the drug. It was only given on one day, but during that day and the following day the following subjective phenomena appeared: Burning and "swelling" of the throat, occipital headache, flatulence, nausea, vomiting, sensation of choking, and a feeling of numbness of the legs. If anything, the pulse-rate was slightly increased. On August 23, $\frac{1}{1200}$ gr. of aconitina was given three times a day, and the pulse-rate counted nine times a day at the times already described. This dose was given for two days, during which time there were burning and swelling of the throat, occipital headache, and flatulence. On August 25 and August 26, $\frac{1}{600}$ gr. was given three times a day. During this time the headache was intense, and, besides the symptoms enumerated, there were præcordial pain, dyspnœa, and retching. The patient looked so ill that it was thought advisable to discontinue the drugs. Some of the subjective phenomena, however, continued the following day.

An analysis of the readings shows that the average daily pulse-rate was practically the same when the patient was on $\frac{1}{1200}$ gr. dose, or $\frac{1}{600}$ gr. dose, or during four days following when no drug was being administered.

The average pulse-rate at the time of administration, one hour afterwards, and three hours afterwards, was also unaltered.

It may be thought that although aconite apparently has no action in reducing the pulse-rate in cardiac non-febrile cases it may succeed in pyrexial cases. Accordingly in order to settle this point I made a series of investigations in four cases of pulmonary tuberculosis, one case of malignant endocarditis, and seven cases of scarlet fever, in all of whom there was pyrexia present.

I wish to very cordially express my indebtedness to Dr. Foord Caiger for allowing me entrance to the South-Western Fever Hospital, and to Dr. E. F. Coghlan for helping me in the observations on the cases of scarlet fever.

Case V.—A woman, aged 35, was admitted to the Mount Vernon Hospital on October 6, 1910, with signs of a vomica in the left upper lobe and of partial consolidation of the right apex, both active, and of tuberculous laryngitis. Patient was anæmic, there were pain in the left shoulder region, cough, and purulent expectoration containing tubercle bacilli. The temperature was irregular in type, and ranged up to 101° F.

On October 22, $\frac{1}{600}$ gr. aconitina was given three times a day for three days, then $\frac{1}{450}$ gr. for three days, and then $\frac{1}{300}$ gr. for three days. For a few days after the drug was stopped there was frontal headache.

The pulse-rate and blood-pressure were taken for three days before the drug was commenced, and for three days after it was discontinued. An analysis of the readings, with those of the temperature, shows that the daily averages and those of the hour of administration, of one hour afterwards, and three hours afterwards, were unaffected by the drug.

Case VI.—A young woman, aged 19, was admitted to Mount Vernon Hospital on October 15, 1910, with signs of vomication and consolidation of the upper two-thirds of the left lung, and partial consolidation of the right apex, active, and of chronic non-tuberculous laryngitis. The temperature was irregular in type, and while the patient was taking the drug did not rise above 99·4° F., except on one occasion when it rose to 102·4° F. Aconitina was given for the same time and in the same doses, and similar observations were made as in the last case. A study of the readings shows a negative result.

The two other cases of pulmonary tuberculosis — males — were similarly treated. Unfortunately the records were lost. The drug had apparently no effect.

Case VII.—A woman, aged 22, was admitted to the Great Northern Central Hospital on May 28, 1910, with a history of pain in the left chest, and of two previous attacks of rheumatic fever. On admission the apex beat was in the fifth space in the nipple line, and there was an aortic diastolic and a mitral systolic bruit. The ink polygraph revealed the presence of an auricular wave in the jugular tracing. The temperature on admission was 101° F., and pursued an irregular character, usually highest in the evening between 101½° F. and 103° F., on one date (June 18) reaching 104° F. and falling to near the normal; later in July it became more irregular in character. The pulse was regular and varied in rate from 84 to 120. Antistreptococcic serum, sodium salicylate, and quinine were tried at various times with no appreciable effect on the temperature. Aconitina was given for twelve days, commencing at $\frac{1}{1200}$ gr. twice a day and increasing gradually to $\frac{1}{250}$ gr. three times a day. During the course of administration there were thirst, dryness of throat, slight headache, and vomiting. The pulse-rate was taken for three days before and for three days after the drug was taken. An analysis of the readings, as in the other cases, shows a negative result. A continuous observation by means of the polygraph and manometer after $\frac{1}{200}$ gr. reveals the same.

The remaining cases are seven cases of scarlet fever. The general plan of investigation was the same as in the other cases, that is, the pulse was counted nine times a day, at the same hours each day, both when the drug was being administered and when it was not. The temperature was also taken, but the blood-pressure was not. It is not necessary to describe the cases in detail, because all the records, as well as all the records of the other cases, will be published with this paper. If you study these records you will undoubtedly come to the conclusion that the administration of the drug had no effect in diminishing the pulse-rate. Also the impression made on Dr. Coghlan's mind by comparing the cases with a large number of other cases admitted to the South-Western Fever Hospital was that the drug had no influence on the course of the temperature. I will only mention the ages, the dosage, and the condition of the throat, the last because aconite has a considerable reputation for affections of the fauces.

Case VIII.—Aged 32. Dose: $\frac{1}{600}$ gr., increased to $\frac{1}{450}$ gr., and finally to $\frac{1}{300}$ gr. Fauces painful, red and swollen.

Case IX.—Aged 28. Dose: $\frac{1}{600}$ gr., increased to $\frac{1}{450}$ gr. Fauces injected, œdematous and ulcerated; tonsils swollen and ulcerated.

Case X.—Aged $13\frac{1}{2}$. Dose : $\frac{1}{600}$ gr., increased to $\frac{1}{450}$ gr. Fauces injected, swollen and œdematous.

Case XI.—Aged $10\frac{1}{2}$. Dose: $\frac{1}{1200}$ gr., increased to $\frac{1}{900}$ gr., and finally to $\frac{1}{600}$ gr. Fauces injected and follicles enlarged.

Case XII.—Aged 9. Dose : $\frac{1}{1200}$ gr., increased to $\frac{1}{600}$ gr. Fauces slightly reddened and follicles enlarged.

Case XIII.—Aged 9, Dose: $\frac{1}{1200}$ gr., increased to $\frac{1}{900}$ gr., and finally to $\frac{1}{600}$ gr. Fauces *nil*, abnormal.

Case XIV.—Aged $7\frac{3}{4}$. Dose: $\frac{1}{1200}$ gr., increased to $\frac{1}{900}$ gr., and finally to $\frac{1}{600}$ gr. Fauces ulcerated.

Mr. President, I submit that, judging from these cases, there is no ground for believing that the internal administration of aconite, either in very small, small, or larger doses, even in doses which produce the full physiological effect, reduces the pulse-rate or blood-pressure. On the contrary, if it has any influence at all, its effect is to increase them.

RECORDS OF CASES.

Case I.—J. N., male, aged 36, clerk. Admitted to Mount Vernon Hospital on August 5, 1911. Complaint : Shortness of breath and pain over left chest. History: The patient had acute rheumatism at 8, 13, 22, and 32 years of age; after each attack for some time pain in the left chest; since February, 1910, has suffered from shortness of breath and pain, especially on exertion.

State on admission : Healthy-looking man; pain localized to small area, fifth interspace, 1 in. or so internal to nipple line; pulse of normal rhythm ; left border of heart $3\frac{1}{8}$ in. from middle line; mitral presystolic bruit and short sharp first sound (at times this murmur represented by a distinct sound of a soft character). The ink polygraph reveals the presence of auricular wave in the jugular tracing. (*See* Tables I to IV.)

TABLE I.

Drug and dose	Date		Hour of adminis-tration	Pulse-rate			Blood-pressure			Sensations
				On adminis-tration	One hour after	Three hours after	On adminis-tration	One hour after	Three hours after	
										Aug. 17.—Normally slight phasic variation, not exaggerated by exertion. Went up one flight of stairs without distress
Aconitina, $\frac{1}{600}$ gr. ...	August	18	9.20	100	96	94	110	95 (2 hours after)	98	10.20, slight pricking of left arm; 12.20, dryness of throat; 6.50, legs numb; 7.15, burning of throat, thirst and pricking sensation; 8, burning of throat, numbness gone, slight thirst and dryness of throat
,, ,, ...	,,	,,	5.45	98	92	88	110	112 (1½ hours after)	108 (at 9.30)	
,, ,, ...	,,	19	9.30	90	82	97	116	112	108	10.30, throat dry; 11, ditto; 6, no symptom but dry throat
,, ,, ...	,,	,,	5	102	93	92	122	118	118	
,, ,, ...	,,	20	9.30	102	96	96	—	—	—	—
,, ,, ...	,,	,,	1.30	110	96	92	—	—	126	
,, ,, ...	,,	,,	5.30	98	92	91	—	—	—	
Drug discontinued ...	,,	21 & 22	—	—	—	—	—	—	—	—
Aconitina, $\frac{1}{600}$ gr. ...	,,	23	9.30	88	90 (at 11.30)	90	136	120 (at 11.30)	122	
,, ,, ...	,,	,,	1.30	92	100	—	122	118	—	
,, ,, ...	,,	,,	5.45	100	100	—	114	116	—	
,, ,, ...	,,	24	9.30	88	90	90	122	126	118	10.30, throat dry and præcordial pain (thumping of heart during night)
,, ,, ...	,,	,,	1.30	94	90	90	132	122	116	
,, ,, ...	,,	,,	5.30	86	94	—	110	128	--	
,, $\frac{1}{450}$ gr. ...	,,	25	9.15	96	90	90 (at 12.40)	126	118	124 (at 12.40)	Thumping of heart during night; vomited at 1.30 a.m.; much præcordial pain; 11.30, severe pain
,, ,, ...	,,	,,	1.30	102	102	98	128	130	126	
,, ,, ...	,,	,,	5.30	106	102	—	132	122	—	
,, ,, ...	,,	26	9.20	90	88	—	134	136	—	Thumping of heart at night
,, ,, ...	,,	,,	1.30	86	86	100	130	132	142	
,, ,, ...	,,	,,	5.30	102	102	—	144	142	—	
,, ,, ...	,,	27	9.30	90	88	84	140	130	130	Thumping of heart at night; throat dry (begins 1 hour after dose and lasts 1 hour)
,, ,, ...	,,	,,	1.30	92	88	90	134	122	128	
,, ,, ...	,,	,,	5.30	94	86	—	140	126	—	
,, $\frac{1}{300}$ gr. ...	,,	28	9.30	94	86	88	134	126	130	—
,, ,, ...	,,	,,	1.45	94	98	116	126	134	136	
,, ,, ...	,,	,,	5.30	96	92	—	124	136	—	

Aconitina, $\frac{1}{300}$ gr. ...	August	29	9.30	108	94	92	134	132	142	—
,, ,, ...	,,	,,	1.30	92	96	84	126	130	126	
,, ,, ...	,,	,,	5.30	94	100	—	140	138	—	
,, ,, ...	,,	30	9.30	98	86	86	142	130	134	Vomited between 1 and 2; heart still thumping
,, ,, ...	,,	,,	1.30	80	88	86	120	126	122	
,, ,, ...	,,	,,	5.30	88	94	—	126	126	—	
,, ,, ...	,,	31	9.30	98	94	94	144	138	136	Slight dragging pain during night; also thumping; no nausea
,, ,, ...	,,	,,	1.30	92	96	88	138	128	130	
,, ,, ...	,,	,,	5.30	102	94	—	142	124	—	
,, ,, ...	September	1	9·30	106	90	92	140	120	118	—
,, ,, ...	,,	,,	1.30	92	88	96	126	132	126	
,, ,, ...	,,	,,	5.30	96	102	—	140	134	—	
,, $\frac{1}{200}$ gr. ...	,,	2	9.30	92	80	88	136	120	126	—
,, ,, ...	,,	,,	1.30	86	100	90	126	122	128	
,, ,, ...	,,	,,	5.30	92	102	—	126	130	—	
,, ,, ...	,,	3	9.30	92	88	90	128	118	128	Pain, some feeling of numbness, and pins and needles of lower and upper extremities throughout night; vomited twice during night
,, ,, ...	,,	,,	1.30	92	88	82	118	120	128	
,, ,, ...	,,	,,	5.30	92	88	—	132	138	—	
Drug not given ...	,,	4	9.30	92	—	—	130	—	—	Good night; slight numbness (no pins and needles); no nausea; slight pain and thumping; during day no symptoms
Aconitina, $\frac{1}{200}$ gr. ..	,,	,,	1.30	98	100 (at 3)	102	114	136 (at 3)	130	
,, ,, ...	,,	,,	5.30	102	90	—	136	120	—	
,, ,, ...	,,	,,	9.30	—	—	—	—	—	—	
,, ,, ...	,,	5	9.30	98	86	98	126	118	126	Numbness, pins and needles, dragging pain, thumping heart—all considerable; slight numbness to-day, but no pain or nausea
,, ,, ...	,,	,,	1.30	100	—	—	120	—	—	
,, ,, ...	,,	,,	3.42	—	—	—	—	—	—	
Aconitina discontinued	,,	,,	5.30	92	86	—	124	114	—	Slight numbness (no pins and needles) only of lower extremities; pain; no thumping or nausea
,, ,, ...	,,	6	9.30	84	88	84	110	130	126	No symptoms during day
,, ,, ...	,,	,,	1.30	100	98	—	130	118	130 (at 5)	
,, ,, ...	,,	7	9.30	90	86	86	114	120	124	Last night only dragging pain (considerable) and thumping; no symptoms during day
,, ,, ...	,,	,,	1.30	104	92	96	124	110	132	
,, ,, ...	,,	8	9.30	104	104	90	126	130	130	—
,, ,, ...	,,	,,	1.30	96	—	104	128	—	118	
,, ,, ...	,,	9	9.30	100	—	94	116	—	114	No numbness; dragging pain; less thumping; palpitation severe three-quarters of an hour this morning
,, ,, ...	,,	,,	2.30	102	—	106 (at 6.30)	116	—	134 (at 6.30)	

TABLE II.—CONTINUOUS OBSERVATION FOR ONE HOUR AFTER $\frac{1}{200}$ GR. OF ACONITINA, MEDICINE GIVEN AT 3.42 P.M. ON SEPTEMBER 5.

Time	Pulse-rate	Blood-pressure
3.42	88	114
3.44	100	108
3.46	88	114
3.48	95	120
3.50	90	116
3.52	90	120
3.54	86	122
3.56	90	126
3.58	90	130
4	90	128
4.2	90	128
4.4	90	126
4.6	88	130
4.8	88	132
4.11	86	132
4.12	90	132
4.14	88	—
4.16	92	122
4.18	90	130
4.20	94	120
4.22	92	120
4.24	94	126
4.26	92	132
4.28	—	126
4.30	—	124
4.32	—	—
4.38	92	120
4.40	92	120

TABLE III.

Drug and dose	Date	Pulse-rate, daily average			Blood-pressure, daily average			Average of pulse-rate	Average of blood-pressure
		Hour of administration	One hour after	Three hours after	Hour of administration	One hour after	Three hours after		
Aconitina, $\frac{1}{600}$ gr.	August 18	99	94	91	110	103	103	95	105
,, ,,	,, 19	96	87	94	119	115	113	92	116
,, ,,	,, 20	103	95	93	—	—	126	97	126
,, ,,	,, 23	93	97	90	124	118	122	93	121
,, ,,	,, 24	89	91	90	121	125	117	90	121
,, $\frac{1}{450}$ gr.	,, 25	101	98	94	128	123	125	97	125
,, ,,	,, 26	93	92	100	136	133	142	95	137
,, ,,	,, 27	92	87	90	138	126	128	89	127
,, $\frac{1}{300}$ gr.	,, 28	95	92	102	128	132	133	96	131
,, ,,	,, 29	98	97	88	133	133	134	91	130
,, ,,	,, 30	89	89	86	129	127	128	88	125
,, ,,	,, 31	97	95	91	141	133	133	94	136
,, ,,	Sept. 1	98	93	94	135	128	122	95	128
,, $\frac{1}{200}$ gr.	,, 2	90	94	89	129	124	127	91	127
,, ,,	,, 3	92	88	86	126	125	128	89	126
,, ,,	,, 4	100	95	102	125	128	130	92	128
	,, 5	99	86	98	123	118	126	91	123
,, discontinued	,, 6	92	83	84	120	124	128	86	124
,, ,,	,, 7	97	89	91	119	115	138	92	121
,, ,,	,, 8	100	104	97	127	130	124	100	127
,, ,,	,, 9	101	—	100	116	—	124	100	120

Table IV.

Drug and dose	Date	Pulse-rate, average at			Blood-pressure, average at			Average of pulse-rate	Average of blood-pressure
		Hour of administration	One hour after	Three hours after	Hour of administration	One hour after	Three hours after		
Aconitina, $\frac{1}{600}$ gr.	Aug. 18—24	95	93	92	119	117	114	93	117
,, $\frac{1}{450}$ gr.	,, 25—27	95	92	95	134	127	132	94	131
,, $\frac{1}{300}$ gr.	,, 28 to Sept. 1	95	93	92	134	131	130	93	132
,, $\frac{1}{200}$ gr.	Sept. 2—5	90	91	90	126	124	127	90	126
,, discontinued	,, 6—9	97	92	94	121	120	126	94	122

Case II.—S. E., male, aged 32, barman. Admitted to Mount Vernon Hospital on August 19, 1910. Complaint: Weakness, shortness of breath, and loss of power of left arm and leg. History: No history of rheumatism; chorea at 6 and 8 years of age; typhoid fever in 1897. For some years has complained of shortness of breath and palpitation: Sudden attack of loss of consciousness on June 12, 1910; recovered with loss of power of left arm and leg.

State on admission: Left-sided partial hemiplegia; very short of breath on slightest exertion; pulse completely irregular; apex beat in fifth space; right border of heart $1\frac{5}{8}$ in., and left border $5\frac{5}{8}$ in., from middle line; diastolic thrill at the apex, where a systolic and occasionally a diastolic bruit audible. The ink polygraph reveals an absence of auricular wave in the jugular tracing. (*See* Tables V to VII.)

Table V.—Pulse-rate from August 19 to August 23 (Patient Resting and no Drug) = 100-110.

Drug and dose	Date	Pulse-rate	Sensations
Aconitina, $\frac{1}{600}$ gr., three times daily	Aug. 23—24	97	Dryness of throat, which continues whole time during which drug is being taken
Aconitina, $\frac{1}{450}$ gr.	Aug. 25—27	102	—
Aconitina, $\frac{1}{300}$ gr.	Aug. 28—Sept. 1	89	Aug. 30, frontal headache and vomiting during night; Sept. 1, passed off

TABLE VI.

Drug and dose	Date	Hour of adminis-tration	Pulse-rate			Blood-pressure			Sensation
			On adminis-tration	One hour after	Three hours after	On adminis-tration	One hour after	Three hours after	
Aconitina, $\frac{1}{200}$ gr. ...	September 2	9.30	140	156	126	126	114	110	Numbness and "pins and needles" of right arm and leg, noticed at first slightly
,, ,, ...	,, ,,	1.30	126	132	—	110	108	—	
,, ,, ...	,, ,,	3.43	—	114	—	—	98	—	
,, ,, ...	,, ,,	5.30	116	—	—	120	—	—	
,, ,, ...	,, 3	9.30	118	100	108	122	120	128	Thumping of heart last night; noise during night, slight to-day; nausea; frontal headache
,, ,, ...	,, ,,	1.30	102	104	96	118	118	124	
,, ,, ...	,, ,,	5.30	108	84	—	120	120	—	
,, not given ...	,, 4	9.30	124	—	—	118	—	—	All symptoms, but less, last night; none to-day
,, $\frac{1}{200}$ gr. ...	,, ,,	1.30	128	118 (at 3)	116	116	116 (at 3)	122	
,, ,, ...	,, ,,	5.30	114	116	—	124	110	—	
,, ,, ...	,, ,,	9.30	—	—	—	—	—	—	
,, ,, ...	,, 5	9.30	124	116	120	120	102	110	Numbness, &c., and frontal headache, and occasional thumpings last night; no nausea; no subjective phenomena to-day
,, ,, ...	,, ,,	1.30	130	—	—	116	—	—	
,, ,, ...	,, ,,	5.30	116	108	—	114	120	—	
,, ,, ...	,, 6	9.30	130	96	112	120	120	120	Same symptoms during night; none to-day
,, not given ...	,, ,,	1.30	132	118	—	128	110	—	
,, $\frac{1}{200}$ gr. ...	,, ,,	3.52	—	—	120 (at 6.30)	—	—	106 (at 6.30)	
,, not given ...	—	—	—	—	—	—	..	—	Numbness less last night; thumping less; no headache; no symptoms to-day

Continuous observation for forty minutes after taking $\frac{1}{200}$ gr. aconitina. Pulse-rate continuously 110 per minute.

TABLE VII.

Drug and dose	Date	Pulse-rate, average at			Blood-pressure, average at			Pulse-rate average for whole day	Blood-pressure average for whole day
		Hour of administration	One hour after	Three hours after	Hour of administration	One hour after	Three hours after		
Aconitina, $\frac{1}{200}$ gr.	Sept. 2	127	134	126	119	107	110	129	112
,, ,,	,, 3	109	96	102	120	119	126	102	118
,, ,,	,, 4	121	117	116	120	113	122	118	118
,, ,,	,, 5	123	112	120	117	111	110	118	113
,, ,,	,, 6	130	96	116	120	120	113	114	118

TABLE VIII.

Drug and Dose	Date	Pulse-rate		Blood-pressure
		Morning	Evening	
	July 1	110	118	—
	,, 2	120	128	132
	,, 3	96	114	—
Tincture aconite, 10 ♏ every two hours during day and usually twice during night	,, 4	110	132	—
,, ,, ,,	,, 5	76	124	—
,, ,, ,,	,, 6	98	138	—
,, ,, ,,	,, 7	100	106	132
,, ,, ,,	,, 8	120	108	—
,, ,, ,,	,, 9	96	124	—
,, ,, ,,	,, 10	100	119	—
,, ,, ,,	,, 11	104	95	144
,, ,, ,,	,, 12	90	92	—
Tincture aconite, 15 ♏ every two hours during day and usually twice during night	,, 13	108	108	—
,, ,, ,,	,, 14	92	112	—
,, ,, ,,	,, 15	98	124	144
,, ,, ,,	,, 16	106	119	—
,, ,, ,,	,, 17	104	144	—
,, ,, ,,	,, 18	100	126	154
Discontinued	,, 19	100	117	—

From July 27 to August 17 patient had intermittent attacks of pain, and took aspirin from July 31 to August 3; her pulse continued to vary from 92 to 120 in the morning, and from 92 to 144 in the evening. On August 17 and 18 three doses of aconitina, $\frac{1}{600}$ gr., t.i.d. No change was detected in heart.

TABLE IX.

Drug and dose	Date	Hour of administration	PULSE-RATE			BLOOD-PRESSURE			Sensations
			On administration	One hour after	Three hours after	On administration	One hour after	Three hours after	
Aconitina, $\frac{1}{1200}$ gr. ...	August 23	9.30	124	106	100	140	134	120	—
,, ,, ...	,, ,,	1.35	110	106 (at 3.15)	—	120	135 (at 3.15)	—	
,, ,, ...	,, ,,	5.30	116	100	—	125	115	—	
,, ,, ...	,, 24	9	116	100	120	120	110	116	" Burning " of throat, and nausea
,, ,, ...	,, ,,	1	118	110	116	116	112	120	
,, ,, ...	,, ,,	5.30	116	106	106	116	108	106	
,, $\frac{1}{600}$ gr. ...	,, 25	9	118	120	100	120	120	108	—
,, ,, ...	,, ,,	1	116	106	100	104	104	116	
,, ,, ...	,, ,,	5	106	100	116	116	106	108	
,, ,, ...	,, 26	9	110	104	105	116	96	100	—
,, ,, ...	,, ,,	1	108	100	96	118	110	118	
,, ,, ...	,, ,,	5	112	105	110	110	100	120	
,, ,, ...	,, 27	9	112	115	112	116	120	106	—
,, ,, ...	,, ,,	1	106	106 (at 2.30)	100	100	120 (at 2.30)	114	
,, ,, ...	,, ,,	5	106	114	108	120	130	124	
,, $\frac{1}{450}$ gr. ...	,, 28	9	110	116	96	130	136	130	—
,, ,, ...	,, ,,	1	94	100	110	126	136	138	
,, ,, ...	,, ,,	5	116	120	110	140	130	126	
,, ,, ...	,, 29	9	100	96	112	140	130	120	—
,, ,, ...	,, ,,	1	100	104	95	126	120	126	
,, ,, ...	,, ,,	5	96	100	112	126	136	155	

Aconitina, $\frac{1}{450}$ gr. ...	August	30	9	126	120	116	156	150	134	After each dose feeling as though going to choke, followed by "burning," nausea; conscious of heart beating rapidly and forcibly
,, ,, ...	,,	,,	1	118	110	112	136	128	122	
,, ,, ...	,,	,,	5	100	104	100	118	120	120	
,, ,, ...	,,	31	9	120	130	110	140	140	112	—
,, ,, ...	,,	,,	1	100	104	96	120	118	114	
,, $\frac{1}{1200}$ gr. ...	,,	,,	5	102	108	110	118	130	120	
,, $\frac{1}{300}$ gr. ...	September	1	9	104	100	96	120	120	116	—
,, ,, ...	,,	,,	1	104	98	96	124	120	125	
,, ,, ...	,,	,,	5	106	116	102	128	112	114	
,, ,, ...	,,	2	9	100	96	100	146	126	130	—
,, ,, ...	,,	,,	1	100	104	114	120	130	134	
,, ,, ...	,,	,,	5	100	116	100	136	120	120	
,, ,, ...	,,	3	9	120	118	110	120	120	116	—
,, ,, ...	,,	,,	1	120	112	—	130	118	—	
,, ,, ...	,,	,,	4.16	—	126 (at 6)	120 (at 8)	140	130 (at 6)	136 (at 8)	
No drug	,,	4	9	118	120	120	130	120	140	No subjective phenomena
,, ...	,,	,,	1	116	110	106	140	120	120	
,, ...	,,	,,	5	108	110	116	124	120	120	
,, ...	,,	5	9	120	112	96	136	130	128	—
,, ...	,,	,,	1	100	104	96	120	130	120	
,, ...	,,	,,	5	96	96	100	130	130	128	
,, ...	,,	6	9	120	112	100	130	130	124	—
,, ...	,,	,,	1	106	108	118	128	126	128	
,, ...	,,	,,	5	112	118	100	126	116	114	
,, ...	,,	7	9	112	106	110	118	118	116	—
,, ...	,,	,,	1	110	—	—	118	—	—	

Case III.—E. A. J., female, aged 20. Admitted to Mount Vernon Hospital on June 30, 1910. Complaint: Pain in the left chest and shortness of breath. History: Rheumatic fever at the age of 13, and several attacks since, the last being in February of this year. Has had shortness of breath on exertion since the age of 15.

State on admission: A spare, pale, nervous-looking girl; lies in bed propped up; pulse of normal rhythm, collapsing in type; apex beat in sixth space, diffuse and forcible; right border of heart $1\frac{3}{4}$ in., and left border 6 in., from middle line; double aortic and mitral systolic bruit audible. The ink polygraph reveals the presence of auricular wave in the jugular tracing. (*See* Tables VIII to XIII.)

TABLE X.—SEPTEMBER 3: CONTINUOUS OBSERVATIONS FOR ONE HOUR AFTER TAKING ACONITINA, $\frac{1}{300}$ GR.

Time	Pulse-rate	Blood-pressure	Sensations
4.16	—	—	—
4.18	115	140	—
4.20	125	140	—
4.22	120	160	—
4.23	120	150	—
4.25	127	140	—
4.26	120	140	—
4.30	128	140	—
4.34	125	140	—
4.35	124	140	—
4.39	145	160	—
4.40	128	120	—
4.41	128	140	—
4.43	128	120	—
4.44	—	144	Beads of perspiration standing out on nose
4.45	135	136	—
4.47	130	138	—
4.50	130	150	—
4.51	—	140	Dyspnœa began a few minutes ago
4.52	140	156	—
4.53	150	150	—
4.54	157	156	—
4.55	153	158	Breathing easier; burning sensation of throat and nose just now
4.58	140	110	—
4.59	135	130	—
5.2	133	130	—
5.4	128	130	—
5.7	130	130	—
5.8	132	136	—
5.11	128	112	—
5.13	128	115	—
5.14	125	130	—
5.16	130	118	—
5.17	131	130	—
5.18	140	130	—

TABLE XI.—SEPTEMBER 4: CONTINUOUS OBSERVATIONS FOR ONE HOUR, WITHOUT DRUG.

Time	Pulse-rate	Blood-pressure	Sensations
10.45	120	140	—
10.47	132	145	—
10.49	120	142	—
10.52	120	140	—
10.55	115	150	—
10.57	117	138	—
11	120	140	—
11.2	120	140	—
11.4	115	140	—
11.6	117	140	Left arm moved for two minutes
11.8	140	150	Left arm moved
11.10	130	156	—
11.12	132	156	—
11.14	125	148	—
11.19	135	142	—
11.20	125	150	—
11.22	128	140	Left arm moved
11.24	150	160	—
11.24½	142	160	—
11.25	142	160	—
11.27	125	146	—
11.28	130	138	—
11.30	125	138	—

TABLE XII.

Drug and dose	Date	Pulse-rate (daily average)	Blood-pressure (daily average)
No drug given	July 1	114	—
,, ,,	,, 2	124	132
,, ,,	,, 3	105	—
Aconitina, 10 ♏, every two hours during day and usually twice during night	,, 4	121	—
,, ,, ,,	,, 5	100	—
,, ,, ,,	,, 6	113	—
,, ,, ,,	,, 7	103	132
,, ,, ,,	,, 8	114	—
,, ,, ,,	,, 9	110	—
,, ,, ,,	,, 10	109	—
,, ,, ,,	,, 11	99	144
,, ,, ,,	,, 12	91	—
Aconitina, 15 ♏, every two hours during day and usually twice during night	,, 13	108	—
,, ,, ,,	,, 14	102	—
,, ,, ,,	,, 15	111	144
,, ,, ,,	,, 16	112	—
,, ,, ,,	,, 17	124	—
,, ,, ,,	,, 18	113	154
Discontinued	,, 19	108	—

TABLE XIII.

Drug and dose	Date	PULSE-RATE, DAILY AVERAGE			BLOOD-PRESSURE, DAILY AVERAGE			Pulse-rate, average for whole day	Blood-pressure, average for whole day
		Hour of administration	One hour after	Three hours after	Hour of administration	One hour after	Three hours after		
Aconitina, $\frac{1}{1200}$ gr.	Aug. 23	117	104	100	128	128	120	107	125
,, ,,	,, 24	117	105	114	117	110	114	112	114
,, $\frac{1}{600}$ gr.	,, 25	113	109	105	113	110	111	109	111
,, ,,	,, 26	110	103	104	115	102	113	105	110
,, ,,	,, 27	108	112	107	112	123	115	109	117
,, $\frac{1}{450}$ gr.	,, 28	107	112	105	132	134	131	108	132
,, ,,	,, 29	99	100	106	131	129	134	101	132
,, ,,	,, 30	115	111	109	137	133	125	112	131
,, ,,	,, 31	110	117	103	130	129	113	110	126
,, $\frac{1}{300}$ gr.	Sept. 1	105	105	98	124	117	118	102	120
,, ,,	,, 2	100	105	105	134	125	128	103	129
,, ,,	,, 3	120	119	115	130	123	126	118	125
No drug	,, 4	114	113	114	131	120	127	114	126
,,	,, 5	105	104	97	129	130	125	102	128
,,	,, 6	113	113	106	128	124	122	110	125
Aconitina, $\frac{1}{1200}$ gr.	Aug. 23—24	117	104	107	122	119	117	109	119
,, $\frac{1}{600}$ gr.	,, 25—27	110	108	105	113	108	113	108	111
,, $\frac{1}{450}$ gr.	,, 28—31	108	110	106	133	131	126	108	130
,, $\frac{1}{300}$ gr.	Sept. 1—3	108	110	106	129	122	124	108	125
Discontinued	,, 4—7	110	110	106	129	125	128	109	127

Case IV.—S. W., female, aged 35, dressmaker. Admitted to Mount Vernon Hospital on July 26, 1910. Complaint: Shortness of breath and "throbbing" of heart. History: Measles, whooping-cough, and scarlet fever during childhood; no rheumatism, chorea, or tonsillitis; at the age of 25, bronchitis, followed by palpitation on and off, worse last two years; dyspnœa, præcordial pain and occasional swellings of feet last four years.

State on admission: Lies in bed propped up; breathing rapid and laboured and aggravated by the slightest movement; pulse completely irregular; apex beat, fifth space; right border of heart 1 in., and left border 5¼ in., from the middle line; at the apex second sound followed by a murmur, which fills up whole diastolic period when the heart is rapid, but when a pause occurs there is a clear interval between the diastolic murmur and the first sound; liver three finger-breadths below costal margin. The ink polygraph reveals an absence of auricular wave in the jugular tracing. (*See* Tables XIV to XVI.)

TABLE XIV.

Pulse-rate from August 10 to 17 varied from 65 to 82 (patient resting and no drug), as follows: August 10, 75; August 13, 72; August 15, 65; August 17, 82.

Drug and dose	Date	Hour of administration	Times of observation	Pulse-rate	Sensations
Aconitina, $\frac{1}{600}$ gr.	August 18	9.20	—	—	Nausea, and swelling and burning of throat
			10.20	96	
			10.50	84	
			11.20	74	
			11.50	86	
			2.20	84	
			1.20	84	
,, ,,	August 18	5.25	—	70	
			5.50	83	
			6.35	81	
			7.10	74	Headache
			7.45	86	
			8.15	88	
			8.45	80	
			9.30	78	Numbness of legs during night
			10	76	
Drug discontinued	August 19	—	9.30	92	Flatulence, burning and swelling of throat, headache
			11.45	90	Nausea and vomiting this morning, which continued, and in afternoon trembling and choking
			2.45	92	
			3.30	121	
Aconitina, $\frac{1}{1200}$ gr.	August 20	9.30	—	96	—
			11.45	94	
Drug discontinued	August 21	—	—	—	—
,, ,,	August 22	—	—	—	—

TABLE XV.

Drug and dose	Date	Hour of administration	PULSE-RATE Hour of administration	One hour after	Three hours after	Sensations
Aconitina, $\frac{1}{1200}$ gr.	August 23	9.30	87	80	—	Nausea, burning and swelling of throat after medicine; flatulence; occipital headache
,, ,,	,,	1.30	86	82	—	
,, ,,	,,	5.30	84	82	—	
,, ,,	August 24	9	84	82	95	Symptoms as before, including headache
,, ,,	,,	1	86	82	86	
,, $\frac{1}{600}$ gr.	,,	5.30	88	88	88	
,, ,,	August 25	9	88	80	80	Symptoms as before, but also retching and præcordial pain
,, ,,	,,	1	84	80	82	
,, ,,	,,	5	—	88	99	"Dreadful night"; occipital headache, so severe that could not sleep; dyspnœa; otherwise symptoms as before
,, ,,	August 26	9	82	78	82	Symptoms continue
,, ,,	,,	1	82	78	84	
,, ,,	,,	5	92	80	94	

TABLE XV—(*continued*).

Drug and dose	Date	Hour of observation	Pulse-rate			Sensations
			At hour of observation	One hour after	Three hours after	
Drug discontinued	August 27	9	84	76	84	Better; less headache, nausea, burning in throat, no præcordial pain, slight flatulence; "lumps" in throat almost gone
,, ,,	,,	1	84	86	84	
,, ,,	,,	5	80	96	80	
,, ,,	August 28	9	86	82	80	—
,, ,,	,,	1	90	86	96	
,, ,,	,,	5	96	94	96	
,, ,,	August 29	9	80	84	80	—
,, ,,	,,	1	76	82	76	
,, ,,	,,	5	84	84	92	
,, ,,	August 30	9	92	88	90	—
,, ,,	,,	1	84	84	80	
,, ,,	,,	5	80	96	88	

TABLE XVI.

Drug and dose	Date	Pulse-rate, average at			Pulse-rate, average of whole day
		Hour of administration	One hour after	Three hours after	
Aconitina, $\frac{1}{1200}$ gr.	August 23	86	81	—	83
,, ,,	,, 24	85	82	90	86
,, $\frac{1}{600}$ gr.	,, 24	88	88	88	18
,, ,,	,, 25	86	83	87	85
,, ,,	,, 26	85	79	87	84
Discontinued	,, 27	83	86	83	83
,,	,, 28	91	87	91	89
,,	,, 29	80	83	83	82
,,	,, 30	84	89	86	86
Aconitina, $\frac{1}{1200}$ gr.	August 23—24	85	81	90	85
,, $\frac{1}{600}$ gr.	,, 24—26	86	83	87	85
Discontinued	,, 27—30	85	86	86	86

Case V.—K. D., female, aged 35. Admitted to Mount Vernon Hospital on October 6, 1910. History: Measles three times, whooping-cough, two years ago "run down," and anæmia. March 10: Hoarseness, cough, night-sweats, loss of flesh.

State on admission: Anæmic; tongue clean; pain in region of left shoulder; cough; expectoration purulent and contains tubercle bacilli; dyspnœa; night-sweats; signs of vomica, active, left upper lobe, and of partial consolidation right apex; signs of infiltration of arytænoids (cords not seen). (*See* Table XVII.)

TABLE XVII.

Drug and dose	Date	Hour of observation	TEMPERATURE			PULSE-RATE			BLOOD-PRESSURE		
			At hour of observation	One hour after	Three hours after	At hour of observation	One hour after	Three hours after	At hour of observation	One hour after	Three hours after
			° F.	° F.	° F.						
None	Oct. 19	9	N.	N.	100	106	106	113	100	100	108
,, ...	,, ,,	1	100·2	100·4	100·6	112	116	110	108	108	102
,, ...	,, ,,	5	101·4	101	100	110	112	116	108	106	106
,, ...	,, 20	9	97·2	97·2	97·4	100	112	116	96	100	96
,, ...	,, ,,	1	98	98	98·6	100	112	116	96	102	106
,, ...	,, ,,	5	99	99	98·6	110	100	110	110	112	102
,, ...	,, 21	9	96·6	96·6	98	100	96	112	100	98	98
,, ...	,, ,,	1	99	99·8	99·4	116	116	112	100	102	106
,, ...	,, ,,	5	99	99	100	106	116	110	102	110	110
		Hour of administration	At hour of administration			At hour of administration			At hour of administration		
Aconitina, $\frac{1}{600}$ gr.	,, 22	9	96·6	97	N.	106	106	104	100	100	94
,, ,,	,, ,,	1	97·4	100·2	99	125	120	130	102	98	94
,, ,,	,, ,,	5	97·2	98	99	126	115	120	102	106	96
,, ,,	,, 23	9	97	97	100·2	120	115	128	106	102	94
,, ,,	,, ,,	1	100	99	99	120	120	118	94	98	104
,, ,,	,, ,,	5	99	98	100	120	118	112	108	100	106
,, ,,	,, 24	9	97	98	100	100	120	106	104	100	94
,, ,,	,, ,,	1	100	101	101	106	120	118	106	100	100
,, ,,	,, ,,	5	99	N.	98·8	112	120	120	98	106	100
,, $\frac{1}{450}$ gr.	,, 25	9	98	99·8	100	120	122	122	110	102	90
,, ,,	,, ,,	1	100·4	101	100·8	120	120	120	100	100	100
,, ,,	,, ,,	5	101	100	100	120	120	106	100	94	94
,, ,,	,, 26	9	27	97·2	100·6	100	112	112	110	106	100
,, ,,	,, ,,	1	99	101	98·6	108	112	110	100	106	90
,, ,,	,, ,,	5	99·4	99	99	107	113	120	100	100	100
,, ,,	,, 27	9	97·2	98·6	99·6	118	116	112	100	96	100
,, ,,	,, ,,	1	99·6	100·4	99·6	118	112	115	100	100	90
,, ,,	,, ,,	5	99·6	99·8	100·4	116	120	120	100	106	96
,, $\frac{1}{300}$ gr.	,, 28	9	97	97	99·8	120	120	115	106	106	102
,, ,,	,, ,,	1	98	100·8	—	120	122	—	104	100	—
,, ,,	,, ,,	4	98	100	101·2 (at 8)	120	120	118 (at 8)	90	104	100 (at 8)
,, ,,	,, 29	9	97	97	98	112	106	104	104	104	100
,, ,,	,, ,,	1	98·6	99·2	98·8	120	118	100	98	96	100
,, ,,	,, ,,	5	100·4 98·6	98·6	99	100	104	104	104	104	102
,, ,,	,, 30	9	97	97	98	100	108	100	106	100	94
,, ,,	,, ,,	1	98	99	97	106	100	120	90	96	98
,, ,,	,, ,,	5	97·4	97·6	100	120	110	120	102	100	90
		Hour of observation	At hour of observation			At hour of observation			At hour of observation		
Drug discontinued ...	,, 31	9*	96·6	97	100	100	100	112	94	98	104
,, ,,	,, ,,	1	99	—	99	112	110	116	104	104	104
,, ,,	,, ,,	5	99	99	N.	106	108	118	110	110	106
,, ,,	Nov. 1	9	N.	97	99	112	118	120	108	104	102
,, ,,	,, ,,	1	98·6	99	99·8	115	115	120	90	104	112
,, ,,	,, ,,	5	99·4	N.	98·6	124	112	110	112	100	108
,, ,,	,, 2	9	97	97	99	100	100	100	106	100	100
,, ,,	,, ,,	1	97	97·4	98	100	110	120	100	100	106
,, ,,	,, ,,	5	98·2	97·4	98	120	100	100	106	108	112

* Oct. 31 : Frontal headache. Oct. 31 to Nov. 2 : No symptoms, apart from frontal headache, noted.

TABLE XVIII.

Drug and dose	Date	Hour of observation	TEMPERATURE			PULSE-RATE			BLOOD-PRESSURE		
			At hour of observation	One hour after	Three hours after	At hour of observation	One hour after	Three hours after	At hour of observation	One hour after	Three hours after
			° F.	° F.	° F.						
None	Oct. 19	9	97·6	97·6	97	120	120	110	90	90	90
,, ...	,, ,,	1	97·8	98	98	120	122	120	100	100	96
,, ...	,, ,,	5	N.	N.	98·8	115	112	120	100	100	100
,, ...	,, 20	9	97·2	97·2	97·6	120	118	116	100	90	94
,, ...	,, ,,	1	98	98·2	98·8	120	120	120	100	104	96
,, ...	,, ,,	5	98·8	98·8	98·6	122	120	123	100	100	90
,, ...	,, 21	9	97	97	N.	120	118	120	100	94	98
,, ...	,, ,,	1	N.	98·2	N.	120	120	120	98	100	102
,, ...	,, ,,	5	N.	98·2	99·2	116	120	118	102	96	102
		Hour of administration	At hour of administration			At hour of administration			At hour of administration		
Aconitina, $\frac{1}{600}$ gr.	,, 22	9	96·8	97·8	98·6	120	120	116	100	98	98
,, ,,	,, ,,	1	N.	99·4	N.	118	120	122	100	98	92
,, ,,	,, ,,	5	N.	102·4	101·8	125	130	142	90	94	94
,, ,,	,, 23	9	97	97	98	128	130	124	96	90	90
,, ,,	,, ,,	1	98·6	N.	N.	128	140	120	94	94	96
,, ,,	,, ,,	5	N.	98·2	98	120	120	120	100	100	100
,, ,,	,, 24	9	97	97	97·2	120	120	122	100	90	90
,, ,,	,, ,,	1	98	98	98	120	122	120	90	90	90
,, ,,	,, ,,	5	98	98	98	120	122	122	92	96	86
,, $\frac{1}{450}$ gr.	,, 25	9	97·4	98	98	130	130	115	90	92	86
,, ,,	,, ,,	1	98·2	97·6	98·6	126	122	120	90	90	90
,, ,,	,, ,,	5	98·2	98·4	99	120	112	106	96	86	86
,, ,,	,, 26	9	97·4	97·4	97·6	120	120	120	90	90	96
,, ,,	,, ,,	1	97·6	98	98·8	116	110	115	90	86	92
,, ,,	,, ,,	5	98	98·2	99	124	130	128	92	94	94
,, ,,	,, 27	9	97	97	99	120	122	120	98	94	94
,, ,,	,, ,,	1	N.	N.	98·6	118	120	120	90	80	90
,, ,,	,, ,,	5	98	99	N.	118	120	130	88	86	90
,, $\frac{1}{300}$ gr.	,, 28	9	97·4	97·2	97	132	130	132	90	90	86
,, ,,	,, ,,	1	98	98	—	122	130	—	98	94	—
,, ,,	,, ,,	5	98	98	98·2	124	130	132	90	86	104
,, ,,	,, 29	9	97	97	97·4	124	122	120	100	96	92
,, ,,	,, ,,	1	N.	N.	97	106	120	120	90	94	90
,, ,,	,, ,,	5	97·8	97	97·4	118	120	120	86	88	90
,, ,,	,, 30	9	97	97	97·6	120	120	112	86	84	82
,, ,,	,, ,,	1	98	98	97	118	112	108	86	90	90
,, ,,	,, ,,	5	97·6	97	N.	120	120	124	90	90	90
		Hour of observation	At hour of observation			At hour of observation			At hour of observation		
None	,, 31	9	97	97	98·8	120	120	120	86	86	84
,, ...	,, ,,	1	98	—	98·6	120	—	120	86	94	94
,, ...	,, ,,	5	98·2	98	98·2	120	120	120	94	94	94
,, ...	Nov. 1	9	N.	97	97	124	120	120	94	90	86
,, ...	,, ,,	1	N.	98·2	—	120	120	—	90	88	—
,, ...	,, ,,	5	98·6	98	98·2	124	120	126	102	90	100
,, ...	,, 2	9	97	96·8	97·4	116	120	108	90	86	100
,, ...	,, ,,	1	98	98	98·2	115	120	120	96	98	100
,, ...	,, ,,	5	98	97	98	122	115	120	96	94	100

TABLE XIX.

NOTE.—Drug administered at 9 a.m., 1 p.m., and 5 p.m. each day. Temperature and pulse-rate taken at hour of administration and one and three hours after.

Drug and dose	Date	Temperature			Pulse-rate.			Conclusions
		Hour of administration	One hour after	Three hours after	Hour of administration	One hour after	Three hours after	
		° F.	° F.	° F.				
None	October 19	97·9*	98	97·9	118*	118	117	Temperature: Risen at first hour on Oct. 19; risen at third hour on Oct. 21. Pulse-rate approximately level
,, ...	,, 20	98	98	98	121	119	119	
,, ...	,, 21	97·9	97·8	98·7	118	119	119	
Aconitina, $\frac{1}{600}$ gr. ...	,, 22	97·8	99·8	99·6	121	123	127	Temperature: Risen in one case, lowered in another, unaltered in the third after first hour; risen in two cases after third hour. Pulse-rate risen in all cases after first and third hours
,, ,, ...	,, 23	98	97·8	98·1	125	130	121	
,, ,, ...	,, 24	97·7	97·7	97·3	120	121	121	
,, $\frac{1}{450}$ gr. ...	,, 25	97·9	98	98·2	125	121	114	Temperature: Risen in all cases after first and third hour. Pulse-rate lowered 4 beats and 11 beats after first and third hours on Oct. 25; risen on succeeding days
,, ,, ...	,, 26	97·7	97·8	98·4	120	120	121	
,, ,, ...	,, 27	97·8	98·1	98·7	119	121	123	
,, $\frac{1}{300}$ gr. ...	,, 28	97·8	97·7	97·6	126	120	132	Temperature lowered fractionally except after third hour on third day. Pulse-rate lowered 6 beats and raised 6 beats at first and third hours on Oct. 28; risen in each case on Oct. 29 and Oct. 30
,, ,, ...	,, 29	97·8	97·8	97·3	116	120	120	
,, ,, ...	,, 30	97·5	97·3	97·7	119	117	115	
None	,, 31	97·7	97·5	98·5	120	120	120	Temperature lower at first hour and higher at third hour on Oct. 31; lower at first and third hours on Nov. 1; lower at first hour and rising at third hour on Nov. 2. Pulse-rate approximately level
,, ...	Nov. 1	N.	97·7	97·6	123	120	123	
,, ...	,, 2	98	97·3	97·8	118	118	116	

Temperature rose on five days after first hour, eight days after third hour; was unaltered two days after first hour, two days after third hour; fell on eight days after first hour, five days after third hour. Pulse-rate rose five days after first hour, seven days after third hour; unaltered four days after first hour, one day after third hour; fell on five days after first hour, seven days after third hour.

* The figures are respectively the daily averages of temperature and pulse-rate.

TABLE XX.

Drug and dose	Date	Temperature, taken at following times						Pulse-rate, taken at following times						Blood-pressure	Symptoms
		2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a.m.	10 a.m.	2 p.m.	6 p.m.	10 p.m.		
		° F.	° F.	° F.	° F.	° F.	° F.								
None	Aug. 24	101·5	99	98·5	102·8	102	104·5	120	92	96	120	120	120	—	—
,, ...	,, 25	98·5	98·5	98	100	101	104·5	120	88	92	100	104	120	—	—
,, ...	,, 26	101	98	98	99	101	104	120	112	90	100	108	160	—	—
Aconitina, $\frac{1}{600}$ gr. bis die (10 a.m. and 2 p.m.)	,, 27	100	99	99·5	98	100	104	156	120	100	88	96	120	105 (10 a.m.) 95 (2 p.m.) 98 (6 p.m.)	—
Aconitina, $\frac{1}{600}$ gr. t.i.d.	,, 28	102·5	100	98	98	98	104	120	116	96	98	96	96	—	—
,, ,,	,, 29	103	100	98	98·5	100	103	120	100	96	—	100	100	—	—
,, ,,	,, 30	102	101·5	98	98	99	103	100	96	100	96	100	108	100 (10 a.m.) 95 (2 p.m.) 108 (6 p.m.)	Great thirst during following night
,, ,,	,, 31	100	98·5	103·5	103	101	102	96	96	126[1]	96[2]	92[3]	120	[1]100 [2]104 [3]115	Slight dryness of throat
Aconitina, $\frac{1}{450}$ gr. t.i.d.	Sept. 1	103	99	98	98·5	98·5	104	—	98	88[1]	84[2]	100[3]	124	[1]105 [2]105 [3]100	Vomited early next morning, after an aperient
,, ,,	,, 2	103	99	98	98·5	98·5	102·5	120	100	80[1]	96[2]	96[3]	112	[1]100 [2]84 [3]102	Dryness of throat; vomited 10.30 p.m.
Aconitina, $\frac{1}{300}$ gr. t.i.d.	,, 3	102·5	98	98	99	100·5	102·5	120	—	91[1]	100[2]	104[3]	124	[1]110 [2]110 [3]95	Dryness of throat; felt sick during following night, but did not vomit
Aconitina, $\frac{1}{300}$ gr. bis die	,, 4	103	99	98	99	99	103	124	100	100[1]	96[2]	104[3]	116	[1]100 [2]102 [3]125	Slight headache; rather depressed in spirits: sore throat following night
None	,, 5	103	100	98	100	98	102·5	116	124	92	100	100	132	—	Comfortable day & night
Aconitina, $\frac{1}{250}$ gr. t.i.d.	,, 6	102·5	100	98	98	100	103	132	128	92	104	88	116	—	Dryness of throat; slight headache
,, ,,	,, 7	103	100·5	—	98·5	98	103·5	128	112	—	108	104	140	—	Dryness of throat; headache during following night
Aconitina, $\frac{1}{250}$ gr. and $\frac{1}{200}$ gr. at 4.2 p.m.	,, 8	101·5	99·5	99	98	100·5	104	136	128	124	100	104	124	—	—
None	,, 9	101·5	99	98	99·5	102	—	124	106	100[1]	104[2]	140[3]	—	[1]110 [2]112 [3]130	Vomited at 7 p.m.; throat very dry; headache; pulse feeble
,, ...	,, 10	—	99	99	100	101	104	—	106	116	112	100	128	—	—
,, ...	,, 11	101·5	99	99·5	100	101	103	116	104	—	104	116	125	—	—

Case VI.—F. M. O., female, aged 19. Admitted to Mount Vernon Hospital on October 15, 1910. History: Morbilli; gradual onset with cough and expectoration, and hoarseness, July 1909; hæmoptysis one month ago; loss of weight; formerly night-sweats.

State on admission: Height, 5 ft. 4 in.; weight, 8 st. 1 lb. (former average weight, 9 st. 2 lb. 8 oz.); appetite good; tongue clean; cough; purulent blood-stained expectoration, containing tubercle bacilli; night-sweats; signs of vomication and consolidation, with râles, upper two-thirds left chest; slight impairment, harsh breath sounds, and increased vocal resonance right apex; signs of chronic non-tuberculous laryngitis; pulse of small volume and low tension. (*See* Tables XVIII and XIX.)

Case VII.—E. C., female, aged 22. Admitted to the Great Northern Hospital on May 28, 1910. Complaint: Pain in lower left chest and armpit for past fourteen days. Previous illnesses: Acute rheumatism at 8 and 16 years of age.

State on admission: Apex beat fifth space; dullness from right border of sternum to left nipple line; aortic diastolic and mitral systolic bruit. Ink polygraph reveals the presence of an auricular wave in the jugular tracing. (*See* Tables XX and XXI.)

TABLE XXI.—CONTINUOUS OBSERVATION FOR ONE HOUR. ACONITINA, $\frac{1}{200}$ GR. GIVEN.

Time p.m.	Pulse-rate (100 at 3.53)	Blood-pressure (112 at 4)
4.2	—	—
4.4	92	108
4.6	100	112
4.8	102	108
4.10	104	112
4.12	102	112
4.14	100	112
4.16	100	114
4.18	104	114
4.20	102	114
4.22	106	108
4.24	102	110
4.26	100	114
4.28	100	108
4.30	104	114
4.32	98	113
4.34	106	112
4.36	102	116
4.38	102	114
4.40	104	108
4.42	100	—
4.44	102	114
4.46	—	—
4.48	104	108
4.52	102	108
4.56	104	114
5.2	110	—

Case VIII.—M. R. M., female, aged 32. Admitted to the South-Western Fever Hospital on November 2, 1910. Previous illnesses: Measles, diphtheria. History of present attack: October 31: Quite well. November 1: Vomiting, sore throat, glands enlarged, diarrhœa. November 2: Headache, pain in joints, general rash.

State on admission: Throat congested, a few specks of greyish-white exudation on both tonsils; glands palpable and tender; no Koplik's spots; no discharges; pain in knees, feet and fingers; no swelling of joints; punctate scarlet rash all over; tongue peeling in patches, papillæ enlarged.

November 3: Fauces injected, follicles swollen; no adenitis; punctate erythema all over body. November 4: Fauces injected; no joint pains, rash fading. November 5: Fauces red and swollen, but less painful; rash fading, but some punctate, still persists on arms; neuralgic pains at back of neck. November 6: Less pain in neck; feeling more comfortable. (*See* Table XXII.)

TABLE XXII.

Drug and dose	Date	Hour of observation	Temperature: At hour of observation	Temperature: One hour after	Temperature: Three hours after	Pulse-rate: At hour of observation	Pulse-rate: One hour after	Pulse-rate: Three hours after
			° F.	° F.	° F.			
None	Nov. 3	9	102	103	101	136	136	128
,, ...	,, ,,	1	100	102	101	120	120	120
,, ...	,, ,,	5	103	102·6	103	136	136	136
		Hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{600}$ gr.	,, 4	9	102	101	101·2	128	128	128
,, ,,	,, ,,	1	101	101	100	126	128	116
,, ,,	,, ,,	5	99·8	100	101·2	112	112	116
,, $\frac{1}{450}$ gr.	,, 5	9	99·8	99·2	100·2	112	88	90
,, ,,	,, ,,	1	99·7	99·8	99·5	96	92	92
,, ,,	,, ,,	5	100	100·2	100·2	96	100	90
,, $\frac{1}{300}$ gr.	,, 6	9	99·2	N.	98·75	92	88	88
,, ,,	,, ,,	1	N.	98·75	N.	80	96	76
,, ,,	,, ,,	5	N.	N.	98·75	72	72	88
Drug discontinued	,, 7	9	N.	N.	98·25	88	88	88
,, ,,	,, ,,	1	98	98	N.	80	80	80
,, ,,	,, ,,	5	98	99	N.	80	72	80
,, ,,	,, 8	9	98·5	N.	98·75	80	80	80
,, ,,	,, ,,	1	N.	98	N.	N.	98	N.

Case IX.—I. J., female, aged 28. Admitted to the South-Western Fever Hospital on November 22, 1910. Previous illnesses: Pertussis. History of present attack: November 20: Well. November 21: Vomited, sore-throat, rash on chest. November 22: Adenitis.

State on admission: Fauces injected; tonsils swollen and ulcerated; no Koplik's spots; no adenitis; *nil* abnormal on examination of heart; vivid punctate erythema all over.

November 23: Swelling of fauces a little less; tongue coated; considerable discharge; some adenitis; rash persists and is hæmorrhagic in places; some aching of limbs; no sleep. November 24: Fauces little ulcerated about uvula and considerable œdema yet, but less swelling; cervical glands still enlarged; aches all over though no actual joint pains; rash still well-marked; hæmorrhagic papillæ on limbs in places; slept well yesterday afternoon and evening. November 25: Fauces improved, some injection and œdema; tongue peeled in centre; adenitis remains; punctate erythema remains; sleeping well. (*See* Table XXIII.)

TABLE XXIII.

Drug and dose	Date	Hour of observation	TEMPERATURE			PULSE-RATE		
			At hour of observation	One hour after	Three hours after	At hour of observation	One hour after	Three hours after
			° F.	° F.	° F.			
None	Nov. 23	9	100	102	103	116	120	132
		Hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{600}$ gr.	,, ,,	1	103·5	103	104*	136	136	136
,, ,,	,, ,,	5	102	103	102·4	130	128	186
,, ,,	,, 24	9	101	101	100	120	116	112
,, $\frac{1}{450}$ gr.	,, ,,	1	100	100·5	100	112	112	112
,, ,,	,, ,,	5	100·5	100·5	100·25	116	108	108
,, ,,	,, 25	9	97·25	98	N.	100	96	96
,, ,,	,, ,,	1	N.	N.	98	96	92	90
,, ,,	,, ,,	5	N.	N.	N.	80	83	80

* Hot sponge.

Case X.—V. B., female, aged 13½. Admitted to the South-Western Fever Hospital on November 24, 1910. Previous illnesses: Morbilli, pertussis, diphtheria. History of present attack: November 19: Well. November 20: Sore throat. November 21: Nausea. November 22:

Headache. November 23: Rash on legs. November 24: Swollen cervical glands.

State on admission: Fauces vividly injected and swollen; no Koplik's spots; tongue peeled; no adenitis; no discharges; *nil* abnormal on examination of the heart; vivid punctate erythema all over, some staining.

November 26: Fauces injected and œdematous; tongue peeled; apex beat fifth interspace in left nipple line; mitral systolic bruit, not well propagated; pulmonary second not accentuated; punctate erythema all over. November 27: Tongue peeled; rash faded. (*See* Table XXIV.)

TABLE XXIV.

Drug and dose	Date	Hour of observation	TEMPERATURE			PULSE-RATE		
			At hour of observation	One hour after	Three hours after	At hour of observation	One hour after	Three hours after
			° F.	° F.	° F.			
None	Nov. 24	5	101	103	103	128	116	136
		At hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{600}$ gr.	Nov. 25	9	101	100·4	100·8	112	100	100
,, ,,	,,	1	101	100·8	101	120	112	104
,, ,,	,,	5	100	99·4	99·8	92	92	90
,, ,,	Nov. 26	9	99	100	99	90	92	90
,, ,,	,,	1	99	99·75	99	92	92	92
,, ,,	,,	5	99	100	100·4	100	96	96
,, $\frac{1}{450}$ gr.	Nov. 27	9	100	100	99	92	90	88
,, ,,	,,	1	99	99·75	99	90	90	100
,, ,,	,,	5	99·8	99·4	99	88	92	96
Drug discontinued	Nov. 28	9	N.	—	—	88	—	—

Case XI.—J. R., female, aged 10½. Admitted to the South-Western Fever Hospital on November 1, 1910. Previous illnesses: Morbilli, chicken-pox, pertussis. History of present attack: October 28: Well. October 29: Headache, sore throat, and diarrhœa. October 31: Rash on chest.

State on admission: Fauces injected and follicles enlarged; no Koplik's spots, tongue peeled; no adenitis or discharges; *nil* abnormal on examination of heart; punctate erythema all over.

November 2: Rash fading. November 3: Rash faded. (*See* Table XXV.)

TABLE XXV.

Drug and dose	Date	Hour of observation	Temperature			Pulse-rate		
			Hour of observation	One hour after	Three hours after	Hour of observation	One hour after	Three hours after
			°F.	°F.	°F.			
None	Nov. 2	9	99	N.	98·75	100	92	92
,, ...	,,	1	98	N.	98	88	90	80
,, ...	,,	5	98	N.	98	80	80	72
		Hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{1200}$ gr.	Nov. 3	9	N.	N.	98	80	80	80
,, ,,	,,	1	98	N.	N.	80	80	80
,, ,,	,,	5	N.	97·75	98·25	72	80	80
,, $\frac{1}{900}$ gr.	Nov. 4	9	N.	98·25	N.	80	80	80
,, ,,	,,	1	98	N.	98	82	80	80
,, ,,	,,	5	97·75	97·75	98	80	80	80
,, $\frac{1}{600}$ gr.	Nov. 5	9	97·75	97	97·75	80	78	88
,, ,,	,,	1	98	97·6	97	90	80	96
,, ,,	,,	5	98	98·2	N.	96	94	88
Drug discontinued	Nov. 6	9	N.	98	N.	80	80	80
,, ,,	,,	1	98	N.	98	80	92	92
,, ,,	,,	5	98	98	98	92	88	88

Case XII.—E. L., female, aged 9. Admitted to the South-Western Fever Hospital on November 12, 1910. Previous illnesses: Morbilli, scarlet fever. History of present attack: November 9: Well. November 10: Sudden onset, sore throat. November 12: Rash all over.

State on admission: Fauces injected; follicles slightly enlarged; tongue negative; no adenitis; no discharges; punctate erythema all over.

November 12: Fauces slightly reddened and follicles enlarged; no adenitis or discharges; rash still present. November 14: Fauces still slightly injected; tongue peeled; rash vivid; circumoral pallor. November 15: Fauces *nil;* tongue peeled; desquamating all over. (*See* Table XXVI.)

Case XIII.—M. T., female, aged 9. Admitted to the South-Western Fever Hospital on November 1, 1910. Previous illnesses: Morbilli, chicken-pox. History of present attack: October 30: Well. October 31: Sudden onset, sore throat. November 1: Rash all over.

State on admission: Fauces a little injected along free border of palate; no Koplik's spots; no adenitis; tongue negative; punctate erythema all over trunk, some on limbs; circumoral pallor.

November 2: Fauces injected; tongue coated, papillæ not enlarged; faint punctate erythema all over. November 3: Fauces *nil;* rash faded; tongue negative. November 4: Rash almost faded. (*See* Table XXVII.)

TABLE XXVI.

Drug and dose	Date	Hour of observation	Temperature: At hour of observation	Temperature: One hour after	Temperature: Three hours after	Pulse-rate: At hour of observation	Pulse-rate: One hour after	Pulse-rate: Three hours after
			° F.	° F.	° F.			
None	Nov. 13	9	100	100·5	102	112	112	120
,, ...	,, ,,	1	101·5	101·75	101	116	120	136
,, ...	,, ,,	5	101·5	102	101·5	112	118	130
		Hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{1200}$ gr.	,, 14	9	101	101·5	100	112	112	116
,, ,,	,, ,,	1	100·25	100	100·25	116	118	116
,, $\frac{1}{600}$ gr.	,, ,,	5	100	100·25	100	116	118	116
,, ,,	,, 15	9	N.	N.	N.	100	96	96
,, ,,	,, ,,	1	98·6	N.	N.	92	96	90
,, ,,	,, ,,	5	N.	N.	N.	92	92	90

TABLE XXVII.

Drug and dose	Date	Hour of observation	Temperature: At hour of observation	Temperature: One hour after	Temperature: Three hours after	Pulse-rate: At hour of observation	Pulse-rate: One hour after	Pulse-rate: Three hours after
			° F.	° F.	° F.			
None	Nov. 2	9	100	100·5	100	116	112	108
,, ...	,,	1	100·4	100	100·4	108	112	108
,, ...	,,	5	100	100·4	100	112	112	116
		Hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{1200}$ gr.	Nov. 3	9	99	98·8	N.	100	100	98
,, ,,	,,	1	N.	98	N.	98	96	99
,, ,,	,,	5	N.	N.	N.	96	92	92
,, $\frac{1}{900}$ gr.	Nov. 4	9	99	99	N.	90	82	80
,, ,,	,,	1	N.	N.	98	80	80	84
,, ,,	,,	5	98	98	98·5	80	80	88
,, $\frac{1}{600}$ gr.	Nov. 5	9	98·5	97·75	97·25	84	98	92
,, ,,	,,	1	98·5	97·8	97·5	100	96	80
,, ,,	,,	5	98·25	98·75	97·75	102	100	84
Drug discontinued	Nov. 6	9	98·75	N.	N.	88	90	92
,, ,,	,,	1	N.	98	98·25	96	94	94
,, ,,	,,	5	98	N.	98	80	80	80

Case XIV.—A. W., female, aged 7¾. Admitted to the South-Western Fever Hospital on November 1, 1910. Previous illnesses: Morbilli, pertussis. History of present attack: October 29: Well. October 31: Headache, vomiting, sore throat, swollen cervical glands, rash on chest.

State on admission: Fauces injected; left tonsil enlarged and soft exudate on inner surface; tongue coated, papillæ not enlarged; no adenitis or discharges, or abnormality on examination of heart; punctate erythema all over, painful.

November 2: Fauces injected; exudate on tonsils; tongue peeled; no adenitis, rhinorrhœa, or otorrhœa; restless night. November 3: Fauces ulcerated; tongue peeled; rash fairly bright. November 5: Pulse slightly irregular; apex beat within nipple line and of good force. (*See* Table XXVIII.)

TABLE XXVIII.

Drug and dose	Date	Hour of observation	Temperature: At hour of observation	Temperature: One hour after	Temperature: Three hours after	Pulse-rate: At hour of observation	Pulse-rate: One hour after	Pulse-rate: Three hours after
			° F.	° F.	° F.			
None	Nov. 2	9	101	103	102·2	133	136	132
,, ...	,, ,,	1	101	102	101	128	136	128
,, ...	,, ,,	5	101·25	101·75	101	128	130	130
		Hour of administration	At hour of administration			At hour of administration		
Aconitina, $\frac{1}{1200}$ gr.	,, 3	9	99	99·2	100	112	118	116
,, ,,	,, ,,	1	99	99·2	100	116	116	112
,, ,,	,, ,,	5	100	100	99·5	112	108	116
,, $\frac{1}{900}$ gr.	,, 4	9	97·75	N.	98	96	96	96
,, ,,	,, ,,	1	N.	N.	98	96	92	90
,, ,,	,, ,,	5	N.	98·3	N.	90	92	90
,, $\frac{1}{600}$ gr.	,, 5	9	97·25	97·75	97	80	80	100
,, ,,	,, ,,	1	98	97·75	97·5	84	82	80
,, ,,	,, ,,	5	97·75	98·5	N.	78	92	84
Drug discontinued	,, 6	9	N.	N.	N.	90	90	88
,, ,,	,, ,,	1	98·75	N.	97·75	84	80	74
,, ,,	,, ,,	5	98	98	98·25	74	72	88
,, ,,	,, 7	9	97·8	97·8	98	72	72	80
,, ,,	,, ,,	1	N.	N.	98	80	80	78
,, ,,	,, ,,	5	N.	N.	N.	80	80	84

DISCUSSION.

Dr. Berdoe remarked that he had used aconite as a general practitioner in a great number of cases of pneumonia with high temperature, giving 5 ♏ doses of the tincture, and always with excellent results. The same applied to subacute rheumatism. He had never had occasion to give more than 5 ♏ doses, and within two days the pulse-rate was reduced and the temperature fell. The paper of Dr. Price seemed to show that it did not much matter what was given. In the cases where he considered aconite did good it might have been that the illness was naturally tending towards a favourable termination: possibly the attention of the nurse or the cold sponging had an effect for good. While listening to the paper he would not have been greatly surprised to hear that morphia had no effect in relieving pain or abating cough. His faith in pharmacology had been much shaken by the paper. When a student at the London Hospital, he was told that aconite was the proper thing to give for the conditions he had mentioned. But it now seemed that there was not a good scientific reason.

Dr. Robert Capes remarked that there were two sides to every question, and his experience as a general practitioner did not coincide with that of the last speaker. His belief was that the conclusion drawn in the paper—viz., that aconite as a drug was useless—was accurate, and his only surprise was that it should have been sufficiently important to bring this conclusion before the Society. So far back as 1886 he was taught to use aconite, but he had not prescribed it since the year 1888 and he was not aware it was at present used in general practice. If 5 ♏ doses would bring down the temperature and the pulse in pneumonia within two days, all he could say was that that was a type of pneumonia which had not come his way. He believed homœopaths still employed it. He thought the statement that aconite lowered the pulse and temperature in pneumonia was likely to prove misleading, and should not go forth unchallenged.

Dr. Johnston-Lavis said so little was known about the alkaloids of aconite that before any value was attributed to experiments of the kind, one should know definitely the chemical composition of a definite crystalline form which had been carefully studied by the crystallographer, and one in which even the optical properties had been studied. Many researches had been carried out on the alkaloids of the aconite plant. Even at the present time in France (where he practised) the tincture of the root and the leaf were credited with different properties. Knowledge in regard to digitalis and similar bodies was in a very chaotic state. When he was a pupil of Dr. Ringer that therapeutist was very enthusiastic about the use of aconite, and he believed that all his (the speaker's) contemporaries, when they went into practice, used aconite extensively. Before long he was impressed with the fact that there was much imagination in the results supposed to be derived from its use. At present he only used it on the rare occasions when his patients asked for it. He thought there should be further experiments with regard to the use of aconite in sore throats, especially those not associated with scarlet fever. He once assisted in treating a nearly fatal case of aconite poisoning, and there the peculiar effect was on the throat. Possibly by stimulating the phagocytosis or increasing the circulation it might help Nature in eliminating the invading micro-organisms. The investigation should be on sore throats having a defined bacterial origin. It was all very well to speak of follicular tonsillitis, or granular or membranous tonsillitis ; but that was not enough. There might be on the one hand simply a stimulation of the vital processes localized in the throat by the therapeutic effect of aconite : or aconite might have a specific action on certain microbes or their products and not on others.

Dr. PRICE, in reply, reminded the meeting that the only object of the paper was to deal with the effect of aconite on the pulse-rate ; he purposely omitted other observations. His reason for bringing the matter forward was that aconite had a distinct reputation for reducing the pulse-rate. This might be found in any standard book on pharmacology. He was sorry to destroy any belief which the first speaker held, but would point out that the reduction of the pulse-rate and temperature in pneumonia following the administration of a certain drug did not necessarily prove that the drug was the cause of it. In the cases under discussion he counted the pulse-rate before the administration of the drug, during it, and afterwards, at exactly corresponding times, and as far as he could gather, there was no diminution in the pulse-rate at all. With regard to tonsillitis, that was a disease in regard to which one might easily fall into error, because in a few hours the temperature might go down even though no drug was being taken. He believed it was in cases of tonsillitis that the drug largely derived its reputation. With regard to the specimens sold at ordinary chemists' shops, samples were obtained from three or four of them, all well known, and they were all found to be inert.

The Effect of Atropine on the Pulse-rate in Cases under the Influence of Digitalis.

By M. D. Silberberg, M.D.

The observations on which this paper is based were carried out while working under Dr. James Mackenzie in the Heart Wards of the Mount Vernon Hospital for Diseases of the Chest.

The clinical group of cases of heart disease which most readily and typically shows speedy improvement under digitalis treatment is that class of mitral cases of rheumatic origin which have developed fibrillation of the auricle. It is found that, generally speaking, patients with senile (degenerative) cardio-sclerosis, associated with fibrillation of the auricle, do not show nearly as marked a reaction to this drug, neither do the various conditions which have a regular rhythm. In the healthy heart it is rarely possible to reduce the rate to any marked degree (i.e., a fall of twenty beats per minute), neither is it in the tachycardia of febrile cases. In some cases with a regular rhythm a condition of marked slowing is produced, but here a more or less complete heart block is generally the result, or a marked sinus slowing. In these cases there is generally a co-existing impairment of the conduction tissue, as evidenced by the wide a—c interval in the jugular curve, or a widened P—R interval in the electro-cardiographic tracing.

The question as to the manner in which digitalis slows the rate in these post-rheumatic cases with auricular fibrillation is not yet definitely settled.

In a recent paper, Dr. Lewis [3] discusses the evidence in favour of digitalis slowing being due to heart-block produced by the drug. He is of the opinion that those cases of fibrillation which react markedly to digitalis will be shown to have had a defect in conductivity, evidenced by a wide a—c (P—R) interval. Experimentally, slowing of the ventricle when the auricle is fibrillating may be induced by stimulation of the vagus, the production of asphyxia [4]—both of which produce heart-block—or by section of the bundle [3]. But it is difficult to reconcile Lewis's view with the fact that in the great majority of cases of auricular fibrillation digitalis causes marked slowing. But with those in which the pulse is rapid, indicating ready conduction, it acts much more efficiently than in cases with slow pulse, in which the conduction may probably be more impaired. On Lewis's assumption the latter should react more readily.

Professor Cushny has suggested the view that under digitalis increased inhibitory activity lessens the number of impulses reaching the ventricle from the disorderly auricle. [1] If digitalis slowing is due to partial heart-block, which is the more important factor—the increased vagal activity or the direct action on the conducting tissues, or is the heart-block due to a combination of both factors, the degree of each varying in different groups of cases? A third factor to be considered is the general lowering of the irritability of the heart muscle by the improved nutrition, brought about by the improved circulation. It may be that the first step in the lowering of irritability follows on the exaltation of the inhibitory centre, the heart-rate is reined in, and the force of the muscle contraction improved and rendered more effective, and the general circulation is thus brought to a higher level of efficiency. This helps the elimination of toxic material through the increased output of urine, especially in dropsical cases, and the heart muscle shares in the general improved circulation.

In the aged the activity of the inhibitory centres is relatively low, and atropine, as will be shown later, has very little effect on the heart-rate, and the degeneration of the cardiac muscle is such that the nutrition cannot be much improved. These cases are, therefore, not in a favourable position to respond to the action of digitalis, and clinically it is found that very few do respond to this drug.

The measure of improvement under digitalis in these fibrillating cases is of great importance as a guide to the continuation of the treatment, and also as to the future prospects of the patient after leaving the hospital, and it was suggested by Professor Cushny that some light might be thrown on this question by the reaction of the patient to the removal of the cardiac inhibition by atropine. In this way the effects of the vagus action might be differentiated from those due to the direct action on the muscle and the improved nutrition. The following account is based on twelve cases.

By thus removing the inhibitory action one chief factor could be eliminated. Cases were selected which had been under observation in hospital for some time, and the pulse-rate was fairly stable under conditions of rest only. One-fiftieth of a grain of atropine sulphate (1·3 mg.)[1] was injected hypodermically and the pulse-rate recorded. This was done graphically by the use of a Mackenzie polygraph. The radial tracing was allowed to run through for about ten minutes to obtain the average rate, then the atropine was injected, and the tracing continued for an hour after this. By marking out the length of tracing in half minutes from the time marker, the rise in rate was obtained and the figures plotted on the curves shown.

---

[1] $\frac{1}{25}$ gr. and $\frac{1}{33}$ gr. were used on two occasions (Cases II and XII).

The next observation on the patients was taken while they were under the influence of full doses of digitalis, that is to say, until nausea and headache were complained of. The same dose of atropine was given and the rise in rate again recorded. The comparison of the two curves gives the relative difference in the rise of rate, and of the maximum rates produced by atropine—i.e., removing the vagal inhibition before and during digitalis administration.

CASES.

Case I.—S. W., aged 35, female, was admitted on July 26, 1910. This was a case of mitral stenosis and auricular fibrillation. She was admitted complaining of shortness of breath and throbbing of the heart. She had suffered from palpitation at intervals for the last ten years, worse during the last two years. She had a severe attack of breathlessness four years ago. There is no definite history of rheumatism.

Physical examination on admission: She lies in bed propped up with pillows, breathing rapid and laboured, and aggravated by the slightest movement. Pulse rapid and very irregular, 125 per minute. Heart, 1 in. to $5\frac{1}{4}$ in. transverse measurements from mid-line. There is a diastolic murmur at the apex filling the interval between second and first sound when the heart is rapid, but not reaching the first sound when there is a pause. Liver, three finger-breadths below the costal margin.

The polygraph tracing showed the usual characters associated with fibrillation.

Progress: She responded to 15 ♏ doses of tincture of digitalis, t.d.s. After $3\frac{1}{2}$ dr. were taken the pulse dropped to 66 per minute. (I will omit full details of the cases as they are to be dealt with more fully by Dr. Mackenzie.) During her stay in hospital she always responded in this manner to digitalis; when she was not on the drug or one of the group her pulse would rise to over 100, and her general comfort was not nearly as great. The main drawback to the digitalis with her was the easy production of headache and nausea. When she left the hospital early this year she was able to walk about the room with comfort.

On December 9 (Chart I, A) she had been off all drugs for eight days, and the pulse-rate had risen to between 90 and 100 per minute. A tracing was taken, and atropine sulphate, $\frac{1}{50}$ gr., was injected, and the pulse-rate recorded by the polygraph. It began to rise in twelve minutes after the injection, and from thence maintained a rate varying from 134 to 144 per minute. Dryness of the throat was complained of in eighteen minutes. Increase of rate, 44 per minute.

She was then placed on digitalis, and had taken 5 dr. by December 16 (Chart 1, B), and this amount had produced nausea and severe headache. Pulse-rate had fallen to 60—64 per minute. The tracing was taken as before, and the same dose of atropine sulphate, $\frac{1}{50}$ gr., given. The pulse-rate began to rise in nine minutes, and reached a rate of 76—84 per minute. Increase, 18 per minute. Throat dry in twenty-two minutes.

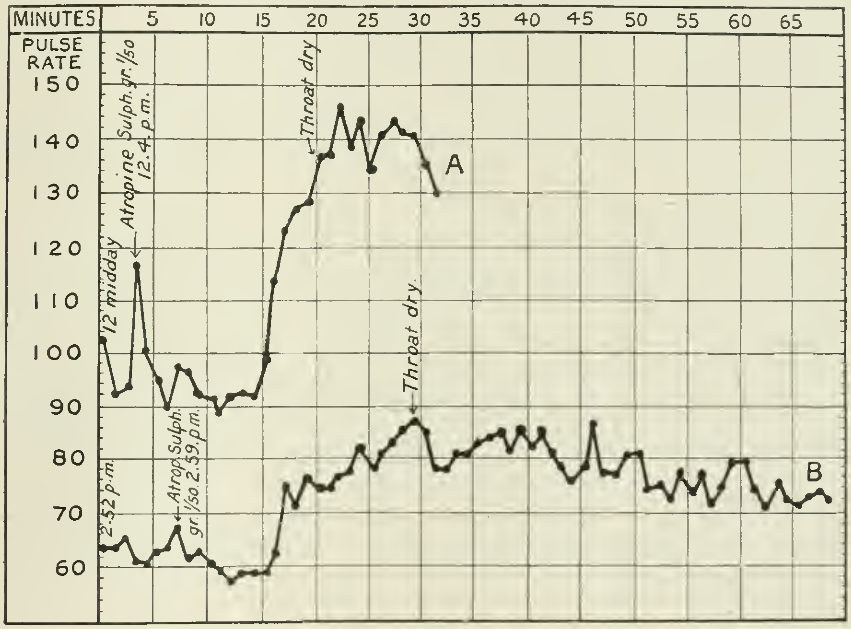


CHART I.

Case I.—Mitral stenosis and auricular fibrillation.

Curve A shows the rise of pulse-rate produced by $\frac{1}{50}$ gr. atropine before the patient is on digitalis (December 9, 1910).

Curve B shows the rise of pulse-rate produced by $\frac{1}{50}$ gr. atropine after the patient is on digitalis (5 dr. taken, December 16, 1910).

Case II.—H. G., aged 24, male, carman, was admitted on January 23, 1911. A case of mitral stenosis and auricular fibrillation. Complains of shortness of breath, palpitation, and cough. Had rheumatic fever at 10 years of age. No venereal diseases. Worked as a carman till three years ago. He had had hæmoptysis at intervals till five years ago. Shortness of breath for three years. Feet were swollen three years ago and again four months ago.

Physical examination: Heart dullness 1 in. to 6½ in., sixth space. There is a mitral systolic and diastolic bruit, the pulse is irregular, and shows the characteristics associated with a fibrillating auricle. Under rest alone his pulse was 92 per minute. Liver enlarged to 3 in below costal margin. He was breathless on slight exertion.

On January 31 (Chart II, A). atropine, $\frac{1}{50}$ gr., was given, and a pulse tracing taken. Pulse-rate before atropine, 76 to 86 per minute. He had been off drugs some days. It commenced to rise in sixteen minutes, and reached an average rate of 130—140, though touching 150. Increase, 53 per minute.

On February 6 he commenced taking digitalis, and his pulse-rate was 60 after taking 7 dr.

On February 21 (Chart II, B), 15 dr. of digitalis had been taken, and his pulse was about 60 per minute. Atropine, $\frac{1}{50}$ gr., was given and produced a rise of 74—86 from 66—72. Increase, 11. Throat dry in twenty-three minutes.

On February 24 (Chart II, C) he had taken 18 dr. of digitalis; pulse-rate 54—60. This time atropine, $\frac{1}{25}$ gr., was administered. This brought the rate to 76—84, practically the same figure as on February 21. Increase, 23. Throat dry in twenty minutes.

This patient was singularly free from nausea and headache on long-continued digitalis therapy, and felt stronger and more equal to exertion under it. He can now climb two flights of stairs without much distress.

The $\frac{1}{25}$ gr. atropine in this second observation is in the nature of a control, and as the maximum pulse-rate is no higher than with $\frac{1}{50}$ gr., one may consider that $\frac{1}{50}$ gr. is a sufficient quantity to fully remove vagal inhibition.

Case III.—C. W., female, aged 42, acrobatic dancer, was admitted on March 5, 1911. A case of mitral stenosis and auricular fibrillation. Complains of shortness of breath on exertion. This dates from an attack of acute rheumatism two years ago. Six weeks ago had an attack of violent palpitation and præcordial pain. There has been no œdema. Family history of phthisis.

Physical examination: Patient is a thin woman. Heart dullness 1 in. to 5¼ in. There is a mitral systolic and diastolic murmur and a diastolic thrill. The pulse is irregular and characteristic of auricular fibrillation. Rate, 90—112. Treated without drugs her pulse did not slow down.

On March 15 it was 120—130 per minute. She was given atropine sulphate ($\frac{1}{50}$ gr.,) and the tracing recorded a rate from 180—190 per minute. The radial tracing for many lengths was not reliable enough to be counted. The above figures are largely those from the apex tracing. A jugular tracing and the same type of irregularity show that

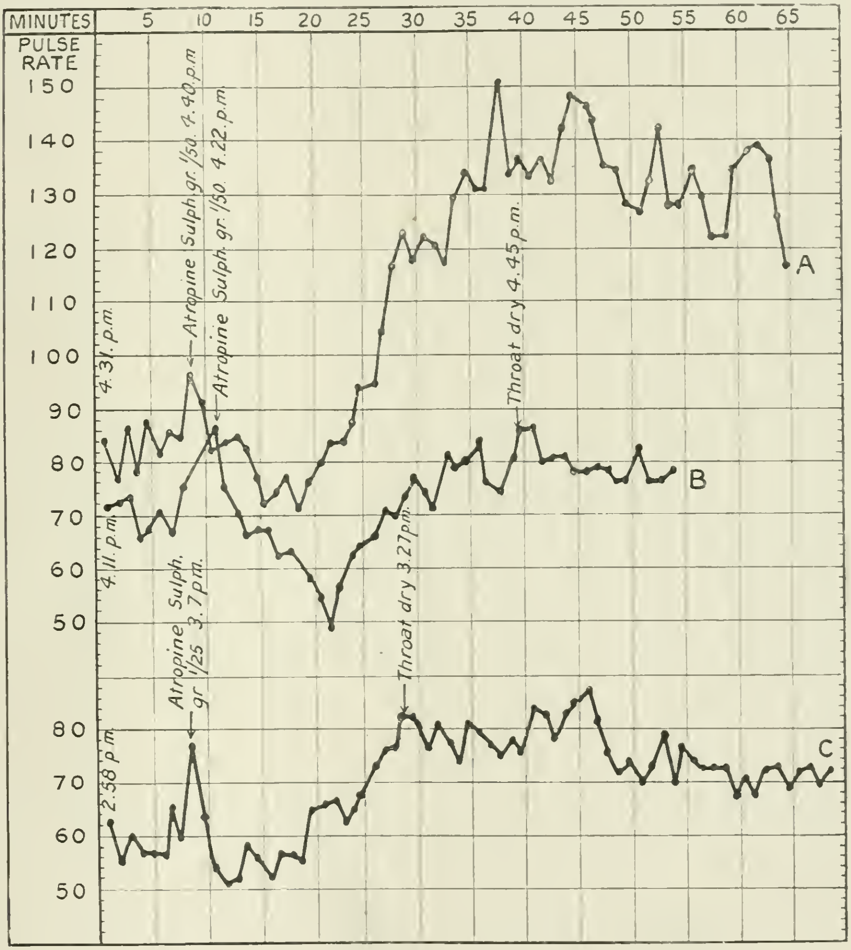


CHART II.

Case II.—Mitral stenosis and auricular fibrillation.

Curve A shows the rise in the pulse-rate produced by $\frac{1}{50}$ gr. atropine before the patient is on digitalis (January 31, 1911).

Curve B shows the rise in the pulse-rate produced by $\frac{1}{50}$ gr. atropine after the patient is on digitalis (15 dr. taken, February 21, 1911).

Curve C shows the rise in pulse-rate produced by $\frac{1}{25}$ gr. atropine after the patient is on digitalis (18 dr. taken, February 24, 1911).

the auricle was still in a state of fibrillation. Increase of rate, 60 per minute.

She began taking tincture of digitalis 20 ♏, t.d.s., on March 16. Nausea was produced by March 22 (7 dr. taken). Pulse 68. The medicine was stopped owing to increased nausea. Pulse-rate, 54.

On March 29 there was still slight nausea present. Pulse-rate, 70 per minute. She was given atropine $\frac{1}{50}$ gr., and the pulse rose to 90—96. Increase, 23. Throat dry in thirteen minutes.

After this date, without further drugs rate rose to 80, and she was put on tincture of strophanthus, 20 ♏, t.d.s., and this keeps the rate at 56—60. She is at present very comfortable and walks about without distress.

Case IV.—D. W., female, aged 17, was admitted on October 26, 1910; discharged on December 23. Mitral stenosis and auricular fibrillation. She has had chorea seven years ago and rheumatic fever nine months ago; she had never been well since, suffering from great difficulty in breathing.

Physical examination: She looked very ill and emaciated, face pallid. Orthopnœic. Respiration 50; pulse 144. Heart apex in sixth interspace. Dullness 1 in. to 6½ in. Rough mitral systolic murmur at the apex, and a systolic and diastolic murmur at the aortic area. Considerable œdema of the lungs. Her pulse tracings showed the characteristics of fibrillation. She responded rapidly to digitalis, and throughout her pulse was one of the most typical in this respect, always showing prompt and marked slowing under its influence. On several occasions there were long periods of coupled beats, 36—40 at the wrist, and the heart-rate double that number.

On December 1 the heart-rate was 76—78, coupled rhythm. With the polygraph receiver a continuous tracing was taken from the apex beat. Atropine sulphate, $\frac{1}{50}$ gr., was administered hypodermically. She complained of dryness in the throat in eleven minutes, the pulse-rate rose in nineteen minutes, and the coupled rhythm began to break from this time onwards, so that there were phases of non-coupled beats mixed with occasional couples. The continuous coupled rhythm returned in about three hours after the injection. The rate rose from 74—78 (coupled) to 86—92, mostly non-coupled. Increase of 12.

The injection was repeated the following day and gave a similar result. She was allowed to come out of the influence of digitalis, and on December 14 (twelve days later) her heart-rate varied from

110—130 per minute, irregular, and just an occasional coupled beat present.

Atropine $\frac{1}{50}$ gr. was given, and in ten minutes the apex tracing recorded from 170—176 per minute for an hour, when the atropine effect began to wear off and the rate slowed to about 120. It is interesting to note that there was an occasional coupled beat present even at this very rapid rate. The character of the irregularity was unaltered. She was then brought rapidly under the influence of digitalis, and the rate fell to about 60 till she left hospital. Increase in rate, 53 per minute, as opposed to 12 while under digitalis.

Case V.—W. W., male, aged 35, was admitted on February 18, 1910; discharged in January, 1911. Case of mitral stenosis and regurgitation and auricular fibrillation. On admission complained of shortness of breath and swelling of the legs. He had rheumatic fever twice at the age of 16. Worked hard as a carman till four years ago, when he noticed his heart beating rapidly at times. Shortly after this, when carrying a sack of coals, he was suddenly seized with shortness of breath and tightness across the chest and the pit of the stomach. He had to stop work after this and was unable to continue work for long. He has since been frequently compelled to seek hospital treatment.

Physical examination: Patient lies propped up in bed and his breathing is very distressed. His legs are swollen. Pulse rapid and irregular, about 90 per minute; marked pulsation in the jugular veins of the ventricular type. Apex is diffuse in the sixth space. Dullness, 2 in. to 7½ in. There is rough systolic murmur heard loudest at the apex and a short murmur following the second sound. The liver edge reaches to the umbilicus and it can be felt pulsating. He always gave a prompt reaction to digitalis as regards slowing of the pulse, though it produced severe headache. At the time he was discharged he could climb a flight of thirteen stairs without much discomfort or breathlessness.

On December 9 he had been without drugs for six full days. He was feeling fairly well and could climb a flight of stairs without much distress. His pulse-rate was 70—80 per minute. An injection of $\frac{1}{50}$ gr. atropine was given hypodermically, and the continuous tracing taken as before. The pulse began to rise in twelve minutes and gradually quickened to a rate varying between 90 and 100. Dryness of the throat came on in about twenty minutes. Increase in pulse-rate, 20 per minute.

On December 11 he was put on tincture of digitalis, at first 20 ♏, t.d.s., and after three days on 10 ♏, t.d.s. On December 21 he complained of severe headache and had vomited; he had then taken 3½ dr. A tracing was again taken, with the same dose of atropine. Pulse-rate was 58—60 before injection; it began to rise in sixteen minutes, and reached a rate varying from 84—92, an increase of 29 per minute.

Case VI.—A. M., male, aged 67. Cardio-sclerosis and auricular fibrillation.

On February 27, 1911, his pulse-rate was 118—122 per minute. Atropine, $\frac{1}{50}$ gr., was given, and the pulse reached a rate of 120—132, an increase of 6 per minute.

On March 4 he was put on tincture of digitalis, 20 ♏, t.d.s., and after 8½ dr. it produced nausea and was stopped on March 13. The rate then was 64—74. Atropine, $\frac{1}{50}$ gr., was given and produced a rise of 68—74; average increase, 2 per minute. (The rise shown towards the end of the curve is extraneous, and due to strong desire to empty the bladder: the pulse immediately settled down when this was relieved.)

Case VII.—C. W., male, aged 61, carman, was admitted on March 25, 1911. Cardio-sclerosis and auricular fibrillation at intervals. He complains of shortness of breath on exertion, and this has been present for the last twelve months. Has at times some sense of constriction and pain over the left side of the chest. Feet were swollen about one month before admission. Does not know when his heart is regular or irregular. There is no history of rheumatic fever or other illnesses.

Physical examination: Heart dullness 1 in. to 5¼ in. Has a mitral systolic murmur. Pulse, 64, and irregular (fibrillation). His pulse became regular on April 5, and remained so for three days. Polygraph and electro-cardiograph curves show a normal rhythm in contrast to previous fibrillation records. The irregularity returned on April 8 and remained so till May 3, when it again became regular, and has remained so till the present time. While regular the rate is 60—64, when irregular it is between 70 and 86.

On March 29 his pulse was irregular at a rate of 66—72. Atropine, $\frac{1}{50}$ gr., was given, but practically no rise in the pulse-rate was produced.

Digitalis reduced the rate to 56. He had then taken 15 dr., which amount induced vomiting; it was then discontinued.

A second observation with atropine was not obtained.

Case VIII.—C. O., male, aged 16. Mitral (and aortic) disease. Regular rhythm. Heart-block under digitalis. Admitted on October 20,

1910. He complained of shortness of breath, pain in the left side of the chest, swelling of the legs and abdomen. He was quite well till 11 years of age, then he began to be short of breath. Since that time has been in hospitals five times. No definite history of rheumatism.

Physical examination: Lies propped up in bed, respiration laboured. Face pale and anxious expression. Pulse regular, full, collapsing type, rate 138 per minute. Apex beat large and diffuse outside the nipple line in the fifth and seventh interspaces. Dullness 3½ in. to 5¾ in. At the apex there is a blowing systolic murmur and a diastolic murmur. There is a diastolic thrill over the apex. At the aortic area there is a faint diastolic murmur. Bases of the lungs dull on percussion and there are numerous fine crepitations on taking a deep breath. Jugular veins full and pulsating. Liver dullness 3 in. below costal margin; pulsates. Both liver and jugular tracings were of the ventricular form. There is some fluid in the abdominal cavity and the legs and thighs are œdematous. There is a slight trace of albumin in the urine.

This patient was very ill on admission, and the following day (October 21) was found in a state of collapse. He responded to immediate treatment. On the following day he was put on tincture of digitalis, 20 ♏, t.d.s., and he gradually improved, the urine increasing and the dropsy disappearing. It produced heart-block after 13 dr. had been taken. He felt very well till two days later, when after 15 dr. he vomited and it was stopped. After a week's cessation it seemed as if his heart was increasing in size, so he was again put on tincture of digitalis, 20 ♏, t.d.s., till October 24, when he had taken 9 dr. His pulse again became irregular from heart-block. He is now very well and can manage considerable amounts of exertion without any distress. His P—R interval is not wider than normal.

On December 7 he had been without drugs for thirteen days. His pulse was 120—124 and regular. Atropine, $\frac{1}{50}$ gr., was administered and it produced a rise in pulse-rate to 130—133, an increase of 9 per minute.

On December 19 he had taken 9 dr. of tincture of digitalis, 20 ♏, t.d.s., in nine days. This amount had produced digitalis heart-block. There were also some phases of pulsus alternans. The ventricular rate varied from 72—84 per minute. Atropine, $\frac{1}{50}$ gr., was given, and in seven minutes the regular rhythm returned, the block and pulsus alternans disappeared, and the rate increased to 112—116 per minute. The rhythm remained regular for two and a quarter hours and then the slowing due to digitalis heart-block returned. Increase in rate, 36 per minute.

Case IX.—L. W., female, aged 21, waitress. Aortic and mitral disease, digitalis heart-block. Complains of pain over the heart and shortness of breath. Had rheumatic fever at 13 years of age and again at 17. She has not been strong since the first attack. For the past five weeks she had to discontinue work owing to the pain over her heart and shortness of breath. She had occasional attacks of dyspnœa at night. Has had no œdema of legs, &c.

Physical examination: Heart dullness, 1¾ in. to 5½ in. sixth space. Mitral systolic and diastolic murmurs, latter runs right up to the first sound. There are aortic systolic and diastolic murmurs also. Pulse, 99—100, regular. Blood-pressure, 108—112. Her pulse remained at 84—90 under conditions of rest, and her breathing was somewhat distressed. On March 24, 1911, tincture of digitalis, 20 ♏, t.d.s., was given. After 10 dr. were taken nausea was produced. On March 4 the pulse fell to 60—70 and was irregular. The polygraph and electrocardiograph curves showed this to be due to heart-block. The pulse was 56 per minute on April 8, after 16 dr., and as there was severe headache the drug was stopped. Apart from this she felt much better and was able to get about with comfort. The irregularity remained till March 17. After this date the pulse rose to 96—100. Her P—R interval is 0·28 second (normal 0·12—0·17 second).

On April 5, in the stage of digitalis heart-block (12 dr. taken), atropine ($\frac{1}{50}$ gr.) was administered. Eight minutes after there were phases of regular mixed with irregular phases of pulse-beats, rate 79—86. In twelve minutes the rhythm was regular, and for eight minutes remained so at a rate varying from 104—110, and thence after till the end of the observation there were again phases of regular and irregular rhythms. I have no measurement of the a—c interval in these periods. After atropine, dryness of throat complained of in twenty minutes. Increase in pulse-rate from 60—64 to 110—114 = 50.

Case X.—F. P., female, aged 26, was admitted on January 16, 1911; discharged on April 4. This was a case of mitral stenosis and regular rhythm. Complains of pain over the left breast and occasional fainting attacks. She had chorea at 13 years of age and suffers from joint pains nearly every winter. On exertion she gets palpitation of the heart and her feet swell.

Physical examination: Fairly well nourished; teeth carious. Pulse 62 per minute and regular. Heart dullness, 1¼ in. to 5 in. fifth space. There is a mitral systolic and diastolic bruit at the apex. Digitalis in this patient reduced the pulse from 80 per minute to 68, when vomiting was induced, and it was discontinued.

On March 3, on conditions of rest alone her pulse-rate was 72—74, regular in rhythm. Atropine, $\frac{1}{50}$ gr., was given, and in nineteen minutes the rate began to rise and reached 96—100. Increase, 25 per minute. An observation was not obtained under digitalis.

Case XI.—B. L., male, aged 6. Sinus irregularity. This was a boy who had an irregular pulse of the youthful type. He had a rachitic chest and had a slight bronchitis on admission, otherwise there were no symptoms. Heart apparently normal. His pulse when quiet was about 80 per minute and the diastolic periods varied from 0·6 to 1·1 second.

On January 26, 1911, atropine was given and the radial pulse recorded. It rose in thirteen minutes to a rate of 102 and was then regular; diastolic period, 0·6 second, approximately. In seventeen minutes it was 140; diastolic period, 0.42 second. This was the maximum reached, and it slowly fell after this till the effect of the atropine wore off and the irregularity and slow rate returned. The boy was lying quiet or asleep during the observations.

Case XII.—D., male, aged 24. Mitral stenosis and auricular fibrillation. When admitted to hospital (February 2, 1910) he complained of weakness, shortness of breath and palpitation. He had had two attacks of rheumatic fever at 12 and 18 years of age. He has not been able to work for the last year. His breathing was very distressed and there was orthopnœa. Pulse very irregular, rate 88—117 per minute. Heart dullness, $1\frac{1}{2}$ in. to 6 in. Systolic and diastolic murmur at the apex. Pulmonary second sound reduplicated. Liver 3 in. below costal margin and could be felt pulsating.

This patient improved very much under digitalis and was most comfortable with a pulse-rate of between 45 and 50. He was discharged on May 29, and commenced hard work. He regulates his own dosage of digitalis according to his sensations of breathlessness or quickened pulse-rate.

On December 15 he was seen by Dr. Mackenzie. He had been only taking 1 dr. of tincture of digitalis per week, but over a long period. His pulse-rate was 45—50. There was still auricular fibrillation. An injection of $\frac{1}{33}$ gr. of atropine was given and the pulse-rate recorded. First rise in ten minutes. Dryness of the throat in fourteen minutes. The rate reached 74—80 for about twenty minutes and then fell to 60 the remaining twenty minutes he was under observation. Increase 22. There was no control observation when not under digitalis.

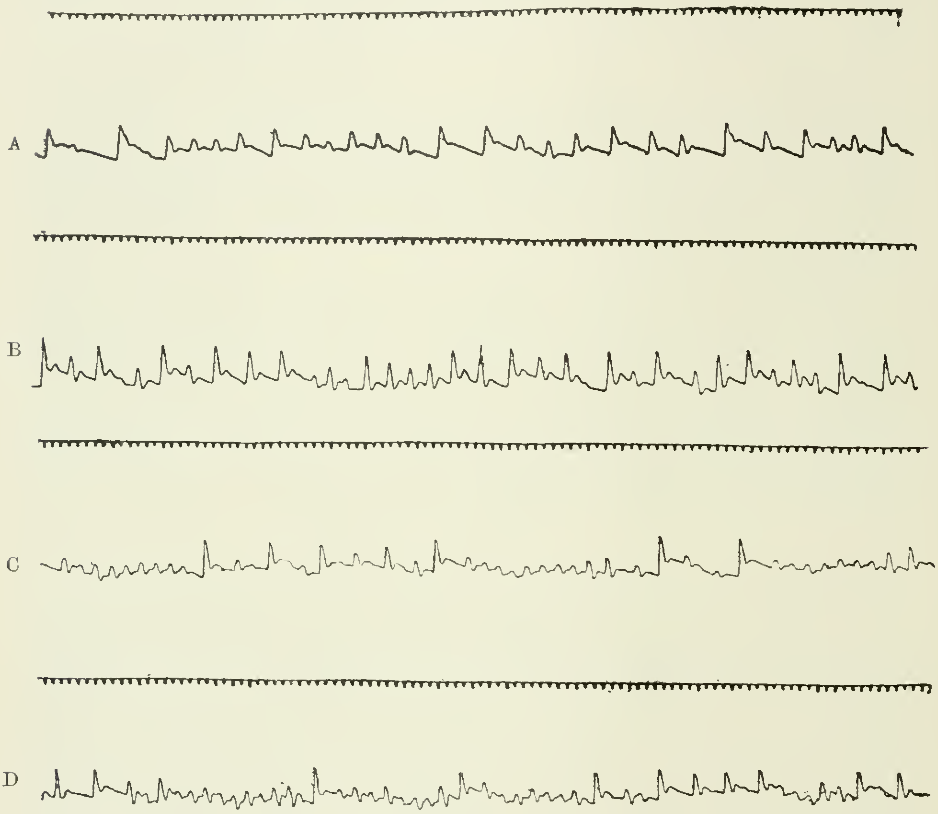


Fig. 1.

Showing the character of the pulse-beats before digitalis is taken.

A, before any drugs ... ... 82 per minute
B, after atropine (about 12 minutes) 100 ,,
C, ,, ,, 15 ,, 123 ,,
D, ,, ,, 30 ,, 140 ,,

Compare with fig. 2.

If we now turn to the Summary (*see* pp. 208 and 209) we see that the fibrillation cases differ widely in respect to the reaction under atropine. Before digitalis treatment Cases I, II, III, and IV show a large increase of pulse-rate—44, 53, 60 and 53, respectively. It will be observed that these cases had an initial rate which is high, the lowest having an average rate per minute of 82, and the highest 125. Compare this increase with Case V, whose initial average rate was 75 and rose to 95, an increase of only 20. The average age of these five cases is 31; youngest 17, oldest 42.

The rate of the released heart thus varies to a remarkable degree in different patients, and those with an initial high rate rose to a much more rapid rhythm under atropine than those which had a slower rhythm, before the drug. The rate before atropine may be considered to depend on two factors: the inherent activity of the rhythm-giving part of the heart, and the degree in which this is held in check by the inhibition. The latter being eliminated under atropine, the heart rate is now determined by the inherent activity alone. In the case of fibrillation this inherent factor may depend, either upon unknown conditions in the auricle, which determine stimulus production, or on the state of the a—v conduction, which determines how many of these stimuli reach the ventricle. Whether the great differences in rhythm observed in our cases under atropine are due to differences in the rate of stimulus production, or of stimulus conduction, cannot be definitely determined, but it seems more plausible to suppose that here the conduction fibres are in an abnormally irritable condition and carry more impulses than the optimal for the ventricle. That is to say, that failure to show the average normal acceleration with atropine is evidence of some structural tissue defect probably in the bundle. The further

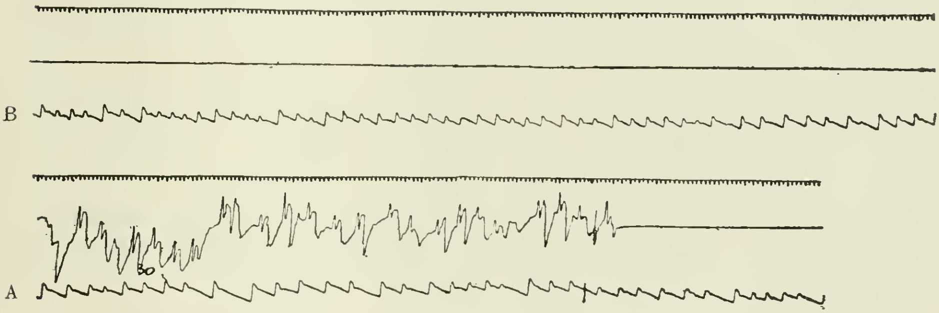


FIG. 2.

Showing the character of the pulse-beats after digitalis (18 dr.) is taken.

A, before atropine ... ... 58 per minute.
B, after ,, ($\frac{1}{25}$ gr.) ... 80 ,,

Compare with fig. 1.

question arises whether such conduction changes would not be a good thing in cases that develop auricular fibrillation, to prevent the ventricle from beating too rapidly in response to the disorderly auricle. Senile cases show practically no acceleration of pulse-rate under atropine (Cases VI and VII).

The fibrillating cases of rheumatic origin, I, II, III, IV, V, and XII, all showed prompt and marked slowing under digitalis. The two senile cases, VI and VII, also reacted well, but this is not the usual experience in the wards. Rheumatic cases of fibrillation with a slow ventricular rate do not as a rule respond to any marked extent to digitalis. Further observations are being made on these two groups of cases. In the rapid hearts the digitalis slowing is due to the smaller beats disappearing, and the pulse-beats are more nearly equal in height and the diastolic periods become more evenly spaced (fig. 2).

If now atropine is given the increase of pulse-rate obtained is due to the small beats reappearing where there were previously the long diastolic pauses, and the more rapid the rate the greater are the number of small beats in proportion to the big beats (fig. 2). The character of the irregularity remains that of auricular fibrillation. It would seem, then, that the digitalis had been acting on the vagus, and the passage of many auricular stimuli had been blocked. When this "damping" effect on the bundle is removed by atropinization more stimuli from the disorderly auricle can cause ventricular response.

But it will be seen that in no fibrillation case except Case V does the rate so produced approach the ordinary rate before digitalis treatment, much less the rate produced by atropine before commencing digitalis. If the action of digitalis were purely vagal, one would expect the heart rate to reach the maximum produced in the first observation. The difference between the maxima is then due to a second factor, probably a nutritional tissue effect, and presumably due to a direct action on the junctional tissues between auricle and ventricle. This latter action is therefore independent of the vagus mechanism and is possibly brought about by the digitalis acting in reducing the irritability of the bundle, and this is probably the result of improved nutrition.

We have, then, two digitalis effects, a vagal component and a cardiac tissue component. The comparative values of these will vary in different groups of cases. This does not mean that the sum of the two components is numerically equal to the figure expressing the digitalis effect. We may diagrammatically represent it (*see* fig. 3).

The patient under conditions of rest alone is atropinized, and we estimate the acceleration of pulse-rate produced by thus cutting out the vagal inhibition. (Senile cases do not give such acceleration.) The case is then brought under the influence of digitalis and a certain slowing produced. This may be called the full digitalis effect and is equal to the difference between this slow rate and the rate before the drug was commenced. Atropine is again given and the increase in rate will be the equivalent of the vagal component in the digitalis action. The maximum produced will fall short of the rate before digitalis. The remainder of this maximum from the maximum produced in the first observation is due to the direct action of the digitalis on the heart, and may be called the digitalis tissue effect.

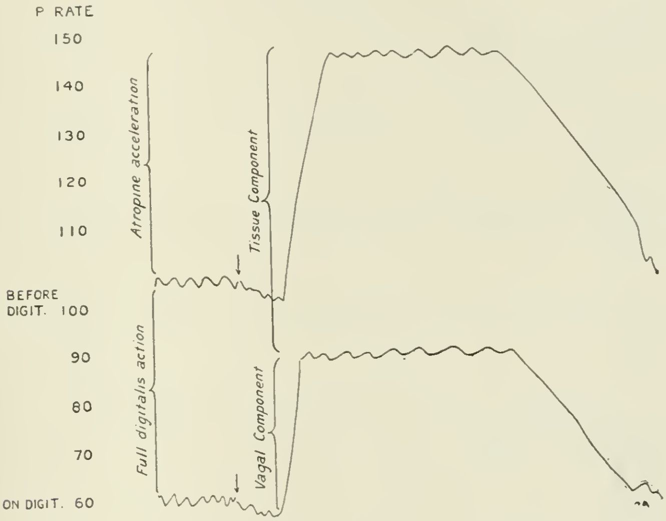


FIG. 3.

In the cases with auricular fibrillation (I, II, III, IV) (*see* Table, p. 210) with a high initial pulse the vagal acceleration is high. These cases have slowed markedly under digitalis. But atropinization has now comparatively little effect—i.e., in these four cases the digitalis effect has been mainly a tissue one and not vagal. In Case V the vagal effect is the greater. In the degenerative cases (VI and VII) the effect is all a tissue one. In the digitalis heart-block (Case VIII) the effect seems to be more vagal than tissue. Observations on the second case of heart-block (Case IX) are not yet complete, and it will be interesting to see if there is any marked difference in these two cases. Case VIII

Summary

No.		Observation	Case	Nature of case	Date	Pulse-rate before injection
1	a	Before digitalis	S. W., female, aged 35	Mitral stenosis, auricular fibrillation	December 9, 1910	90—100
	b	On digitalis	—	—	December 16, 1910	60—64
2	a	Before digitalis	H. G., male, aged 24	Mitral stenosis, auricular fibrillation	January 31, 1911	78—86
	b	On digitalis	—	—	February 21, 1911	66—72
	c	,,		—	February 24, 1911	54—60
3	a	Before digitalis	C. W., female, aged 42	Mitral stenosis, auricular fibrillation	March 15, 1911	120—130
	b	On digitalis *	—	—	March 29, 1911	70
4	a	Before digitalis	D. W., female, aged 17	Mitral stenosis, auricular fibrillation	December 14, 1910	110—130
	b	On digitalis	—	—	December 1, 1910	76—78
5	a	Before digitalis	W. W., male, aged 35	Mitral stenosis, auricular fibrillation	December 9, 1910	70—80
	b	On digitalis	—	—	December 21, 1910	58—60
6	a	Before digitalis	A. M., male, aged 67	Cardio-sclerosis, auricular fibrillation	February 27, 1911	118—122
	b	On digitalis	—	—	March 13, 1911	64—74
7	a	Before digitalis	C. W., male, aged 61	Cardio-sclerosis, auricular fibrillation	March 29, 1911	66—72
	b	On digitalis	—	—	—	56
8	a	Before digitalis	C. O., male, aged 16	Mitral stenosis, aortic regurgitation, regular rhythm	December 7, 1910	120—124
	b	On digitalis	—	—	December 19, 1910	72—84
9	a	Before digitalis	L. W., female, aged 21	Mitral stenosis, aortic regurgitation, regular rhythm	—	90—100
	b	On digitalis	—	—	April 5, 1911	60—64
10	a	Before digitalis	F. P., female, aged 26	Mitral stenosis, regular rhythm	March 3, 1911	72—74
	b	On digitalis	—		—	68
11	a	Before digitalis	B. L., male, aged 6	Sinus irregularity	January 26, 1911	80
	b	On digitalis	—	—	—	—
12	a	Before digitalis	— D., male, aged 24	Mitral stenosis, auricular fibrillation	—	88—117
	b	On digitalis	—	—	December 15, 1910	45—50

OF CASES.

Pulse-rate after injection	Full vagal effect	Full digitalis effect	Vagal component	Tissue component	Dose of atropine	Amount of digitalis taken	Remarks
134—144	44	—	—	—	$\frac{1}{50}$ gr.	—	Throat dry in eighteen minutes
76—84	—	33	18	59	,,	5 dr.	Throat dry in twenty-two minutes
130—140	53	—	—	—	,,	—	—
74—86	—	13	11	55	,,	15 dr.	Throat dry in twenty-three minutes
76—84	—	25	23	55	$\frac{1}{25}$ gr.	18 dr.	Throat dry in twenty minutes
180—190	60	—	—	—	$\frac{1}{50}$ gr.	—	—
90—96	—	55	23	92	,,	11 dr.	* Off digitalis three days; still slight nausea
170—176	53	—	—	—	,,	—	Coupled rhythm broken
86—92	—	43	12	84	,,	?	—
90—100	20	—	—	—	,,	—	Throat dry in twenty minutes
84—92	—	16	29	7	,,	6 dr.	—
120—132	6	—	—	—	,,	—	—
68—74	—	51	2	54	,,	8 dr.	—
66—72	0	—	—	—	,,	—	Not permanent fibrillation
—	—	13	0 (!)	13 (!)	—	15 dr.	Presumed no rise; no tracing obtained
130—133	9	—	—	—	$\frac{1}{50}$ gr.	—	Heart-block produced; abolished by atropine
112—116	—	44	36	17	,,	9 dr.	P—R normal
—	—	—	—	—	—	—	Heart-block produced; abolished by atropine
110—114	—	33	50	—	$\frac{1}{50}$ gr.	12 dr.	P—R wide, 0·28 sec.
96—100	25	—	—	—	,,	—	—
—†	—	5	—	—	—	—	† No observation
140	60	—	—	—	$\frac{1}{50}$ gr.	—	Sinus irregularity abolished
—	—	—	—	—	—	—	—
—	—	—	—	—	—	—	—
74—80	—	55	29	—	$\frac{1}{33}$ gr.	?	1 dr. weekly for a long period

has not a wide P—R interval, Case IX has. Case X was a case of mitral stenosis with a regular rhythm and a normal a—c interval. The result of atropine administration is medium, and that of digitalis slight.

The small boy (Case XI) gives a big acceleration under atropine, and the sinus rhythm, as has been observed by others, was abolished by the drug. Case XII showed marked improvement under digitalis, but the observations are not complete enough to show which factor was the more important.

(The removal of the coupled rhythm in Case IV was probably the result of the increased rate of the heart which usually does away with the occurrence of extra-systoles).

TABLE.

Case	I S. W.	II H. G.	III C. W.	IV D. W.	V W.W.	VI A. M.	VII C. W.	VIII C. O.	IX L. W.	X F. P.	XI B. L.	XII — D.
Full vagal effect ...	44	53	60	53	20	6	0	9	—	25	60	—
Full digitalis effect ..	33	25	55	43	16	51	13	44	33	5	—	55
Vagal component ...	18	23	23	12	29	2	0 (!)	36	50	—	—	29
Tissue component ...	59	55	92	84	7	54	13	17	—	—	—	—

From the analysis of the factors in digitalis slowing it is probable that some guide to the future prospects of the patient may be obtained. Cases of auricular fibrillation with an initial high rate that react well to the drug as a rule do very well, and may even resume work. It would seem that in them the nutrition of the heart muscle is capable of great improvement as a result of using the drug, and that the progress of the case is in this respect a controllable one.

It would seem, then, that by two observations with atropine before and during digitalis administration it is possible to conclude which of two factors, the vagal or the cardiac tissue reaction, is the more important.

REFERENCES.

[1] CUSHNY and EDMUNDS. *Amer. Journ. Med. Sci.*, Philad. and New York, 1907, cxxxiii, p. 66.

[2] FREDERICQ. *Archiv. Internat. d. Physiol.*, Liège et Par., 1904-5, ii, p. 281.

[3] LEWIS. *Brit. Med. Journ.*, 1910, ii, pp. 1670-72.

[4] MATHISON. *Heart*, 1910, ii, p. 54.

INDEX

Proceedings of the Royal Society of Medicine, Vol. IV, 1911

INDEX

(Prepared by Mr. A. L. Clarke)

Note.—The references are to the Sectional *Proceedings*, which are paged independently. In the bound volumes these are arranged in alphabetical order.

Abattoirs (public), hygienic advantages of, Epid. 69
——, see also *Slaughter-houses*
Abbe, recommendation of resection of posterior spinal nerve-roots in treatment of athetosis, Surg. 243
Abbott, subsequent recovery of diphtheria bacillus at site of inoculation in cows, Epid. 85
Abdomen, wall of, actinomycosis of, case of (C. C. Choyce), Clin. 79
Abdominal belt for support of intestines, Electr. 40
—— disease, chronic, causing neurasthenia, Baln. 38
—— diseases, treatment by electricity (H. Manders), Electr. 33
—— operations, hypodermic injection of scopolamine and atropine before chloroform inhalation in, Anæsth. 50
Abduction-frame (Thomas's), Surg. 14, 15
Aberdeen, outbreak of milk-borne gastro-enteritis and diarrhœa at, Epid. 97
—— —— —— sore throat at, Epid. 89
—— —— typhoid fever at, origin discussed, Epid. 111-115
Abortion, dosage of ergot in, Therap. 116
——, question of abortifacient action of ergotin, Therap. 141
——, use of ergot in, Therap. 141-145
Abraham, P. S., case of complete alopecia treated with thyroid gland, quoted, Path. 301, 303
——, discussion on pharmacological effects of the strong sulphur water, Baln. 62
Abrams's method of application of sinusoidal current in treatment of hyperchlorhydria, Electr. 45
Abrikossow, inhalation theory in phthisis, quoted, Child. 228
Abscess (pelvic, tuberculous) diagnosed by rectal examination, Obst. 329, 330
——, post-pharyngeal, anæsthesia in (R. Gill), Anæsth. 34
Absent-mindedness, "hypnoid state" a species of, Neur. 13
Absorption test, Castellani's, Epid. 136, 137
—— —— ——, in differentiation of bacilli, Epid. 52, 67
Accessory sinuses, see *Nose*, accessory sinuses of
Acetanelid in acute frontal sinusitis, Laryng. 127
Acetate and uranium method, estimation of phosphates by, Baln. 47
Acetone in urine during rectal feeding, Therap. 74, 75
Acetonuria, deprivation of carbohydrates factor in production of, Therap. 68
Achondroplasia, case of (F. Langmead), Child. 107
—— —— (E. Pritchard), Child. 1

Achondroplasia, enlargement of epiphyses and costochondral articulations in children, Child. 108
——, human, example of, described, Path. 300
—— and rickets, Child. 106
Achorion Quinckeanum (mouse favus), cultures of (H. G. Adamson), Derm. 47
Achylia associated with secondary anæmia, two cases of, with comments (R. Hutchison), Clin. 141
Acid, percentages produced under mastication of different kinds of bread, Odont. 90, 102, 103
——, production of, in bacilli isolated from urine, in various sugar media, compared with production of gas, Path. 97, 98
—— values, see *Stomach contents*, acid-values of
——, weak, action on teeth prevented by saliva, Odont. 95
Acidogenic and pepsinogenic functions of gastric glands, Med. 27
Acidosis, abolition by assimilation of carbohydrates, Therap. 66
——, reduction of, Therap. 73
Acids, introduction into ear injuring membrana tympani, Otol. 84
Ackroyd, excretion of uric acid in rheumatoid arthritis, Baln. 59
Acne in relation to dental trouble, Odont. 75
—— cachecticorum, Derm. 36
—— rosacea in relation to dental trouble, Odont. 76
—— scrofulosorum in a girl (E. G. Graham Little), Derm. 35
—— —— in patient who had congenital syphilis (H. G. Adamson), Derm. 18
Aconite, action of, degrees of operation, Therap. 153, 154
—— ——, depressant, Therap. 156, 157
—— ——, early experimental tests, Therap. 152, 153
—— ——, on pulse-rate (F. W. Price), Therap. 151-191
—— —— ——, in heart and aortic disease, cases showing, Therap. 158-162, 165-177
—— —— ——, in pulmonary tuberculosis, cases showing, Therap. 163, 178-183
—— —— ——, in scarlet fever, cases showing, Therap. 164, 184-189
—— —— ——, sedative, Therap. 154-157
—— ——, therapeutic, Therap. 190, 191
——, alkaloids of, Therap. 190
——, poisoning by, case of, Therap. 152
——, use of, indications for, Therap. 157
Aconitine, effect of, on bundle of His, Therap. 107
Acromegaly, disease of anterior pituitary body associated with, Odont. 26
——, disturbed functions of thyroid gland associated with, Odont. 26
Actinomycosis, close clinical resemblance to, shown by epithelioma involving whole of lower lip and entire skin of chin (A. W. Williams), Derm. 14
—— in neighbourhood of appendix, Clin. 81, 82
—— —— of spinal cord, Clin. 81
—— of abdominal wall, case of (C. C. Choyce), Clin. 79
Adams, occupation as factor in development of Heberden's nodes, Baln. 32
ADAMSON, H. G.—Acne scrofulosorum in a patient who had congenital syphilis, Derm. 18
——, atrophodermia striata et maculata with phthisis and lichen scrofulosorum, Derm. 1
——, case of angiokeratoma, Derm. 45
—— —— of hyperkeratosis of the palms and soles, Derm. 18
—— —— of sporotrichosis, Derm. 101, 113-121
—— —— of unilateral pigmentary nævus, Derm. 59
——, cheilitis exfoliativa (shown by H. Davis), Derm. 83
——, cultures of *Achorion Quinckeanum* (mouse favus), Derm. 47
——, cultures of violet endothrix trichophyton from a case of "eczematoid" ringworm of the fungus associated with tinea barbæ, Derm. 126
——, discussion on case of acute pityriasis of scalp, Derm. 141
—— —— —— of corymbose syphilide, Derm. 152

Adamson, H. G.—Discussion on case of disseminated tuberculous nodules of the skin, Derm. 49
——, discussion on case of granuloma annulare, Derm. 25
—— —— on a series of ringworm cultures, illustrating the eczematoid ringworm of the hands and feet, Derm. 112
——, erythema ab igne or livedo reticularis with pigmentation, Derm. 46
——, large pigmented vascular sclerosing nævus in a girl, aged 10, Derm. 97
——, macular atrophy in a syphilitic patient, Derm. 122
——, pemphigus foliaceus of sixteen years' duration, Derm. 15
——, photographs of a case of atrophia maculosa cutis, Derm. 2
——, pityriasis rubra pilaris in a girl, aged 5, Derm. 98
——, secondary syphilis in which fading roseola simulated ichthyosis, Derm. 33
——, two cases of adenoma sebaceum in mother and son, Derm. 69
—— (for C. E. **Iredell**).—Epithelioma on lupus vulgaris in a girl, aged 13, Derm. 139
Addison, C., on appendiculo-ovarian ligament, Obst. 345, 346
Addison, O. L.—Discussion of malformation of femur, Child. 165
——, interstitial hernia, Child. 40
——, malformation of femur, Child. 164
——, old healed tuberculous disease of knee-joint, with increase in length of the limb, Child, 39
——, sarcoma of femur, Child. 40
——, tumour on back, probably dermoid, Child, 165
Adeno-carcinoma of uterus and ovaries, case of (M. A. Scharlieb), Obst. 359
Adenoid region, swellings in, causing Eustachian obstruction, Otol. 13, 15
Adenoids and congenital syphilis, Otol. 19
——, influence of, on eye, Laryng. 164
—— (nasopharyngeal), connexion of endometritis in young girls with, Obst. 51
Adenoma, cystic, of facial parotid gland (W. Stuart-Low), Laryng. 59
—— sebaceum (Pringle type), case of (J. H. Sequeira), Derm. 58
—— ——, two cases of, in mother and son (H. G. Adamson), Derm. 69
Adenomatosis vaginæ: condition hitherto undescribed (V. Bonney and B. Glendining), Obst. 18
—— ——, derivation by metamorphosis, inflammatory or reversionary, Obst. 23
—— —— —— from Müllerian ducts, Obst. 23
—— —— —— from Wolffian tubules, Obst. 24
—— ——, historical note, Obst. 21
—— ——, pathology, Obst. 19
Adiposis dolorosa, case of (A. Carless), Clin. 3
—— ——, relation of symmetrical lipomatosis to, Clin. 7
—— ——, sex-incidence, Clin. 7
Adnexa (uterine), appendicitis secondary to, Obst. 354, 355
—— —— diseases of, ætiology, relation of appendix to (H. Macnaughton-Jones), Obst. 341-358
—— —— ——, complicated by appendicitis, Obst. 342, 343
—— —— ——, treatment, surgical, relation of appendix to (H. Macnaughton-Jones), Obst. 341-358
—— —— —— —— —— —— ——, cases illustrating, Obst. 341, 342
—— —— infection from, to appendix, conditions favouring, Obst. 357
—— ——, infection from appendix to, conditions favouring, Obst. 357
—— ——, inflamed, secondary appendicitis arising from, Obst. 355
—— ——, inflammation and appendicitis, differential diagnosis between, Obst. 357
—— —— ——, secondary to appendicitis, Obst. 354, 355
—— ——, operations on, abdominal or vaginal route in, Obst. 348, 349
Adrenalin, action of, in local anæsthesia, Therap. 53, 59-62
—— and cocaine solutions in acute frontal sinusitis, Laryng. 147
——, effect of, on bundle of His, Therap. 107

Adrenalin, injection into gum, effect of, Therap. 60
——, reaction of arteries to, Therap. 80
—— solution, injection of, during anæsthesia, effects, Anæsth. 28, 29, 31, 32, 33
Adults, type of tuberculosis of tonsils in, Laryng. 68
Aeronauts, spontaneous hæmorrhage into tympanic cavity in, Otol. 84
Aertryck, outbreak of food-poisoning at, Epid. 53
"Affect, conversion of," in hysteria, Neur. 11, 12, 15, 16, 18
Africa (North), health resorts of, suitable for neurasthenia, Baln. 42
Agglutination tests, distinction of bacteria by, Epid. 52, 136
—— method, compared with that of complement fixation in differentiation of bacteria, Path. 252
Agglutinins, heterologous, Epid. 136
——, production: mechanism of, in absence of infection, Path. 71
Agitation, motor disturbances characterized by, Neur. 8
Air, constituents of, successive discovery, Baln. 3
——, epidemic constitution of, Epid. 190
Air-hunger, death from, during rectal feeding, Therap. 74
—— —— —— —— ——, presumed course of, Therap. 74, 75
Air of localities, therapeutic uses, antiquity of study, Baln. 1, 2
Air-passages, diseases of, more common in Manchester than St. Marylebone, Epid. 192, 193
—— and œsophagus, removal of foreign bodies from, specimens illustrating (H. Tilley), Clin. 152
AITKEN, D. M.—Discussion on resection of posterior spinal nerve-roots, Surg. 252
Aix-les-Bains, "Douche écossaise" at, Baln. 83
Albinism, complete, in girl, aged 6, with total absence of pigment in skin, hair, choroid and iris (J. M. H. MacLeod), Derm. 7
Albumin in blood and blood serum in splenomegalic polycythæmia, Clin. 92
Albuminuria (adolescent), after-history of cases, Med, 114-120
—— —— —— —— (J. F. Goodhart), Med. 109-120
—— ——, cases should not be excluded from life insurance, Med. 115, 137
—— ——, definition, Med. 121
—— ——, effect on character, Med. 126, 127, 145
—— ——, functional cases, Med. 125, 134, 135, 136-138
—— —— in athletes, Med. 110, 124, 128
—— —— in connexion with late rickets, Med. 132
—— ——, influence of blood-pressure, Med. 124, 144-145
—— —— —— of sexual excitement, Med. 114, 131, 133
—— ——, lordotic cases, Med. 126, 140
—— ——, physique of subjects, Med. 111, 112
—— ——, postural or orthostatic, Med. 124, 125, 138-141, 144
—— (cyclic), Med. 110, 111
—— ——, due to defects in digestion, Med. 111
—— ——, in athletes, Med. 110, 111
—— ——, postural or orthostatic, Med. 121, 122, 138-141, 144
—— (intermitting), Med, 112
—— ——, influence of food-bolting on, Med. 114
—— —— —— of inordinate growth on, Med. 111
—— ——, marked, not a characteristic of nephritis, Med. 112, 113
—— (orthostatic), in congenital heart disease, Clin. 93, 95
——, syphilis, chyliform ascites, and dropsy, case of (H. Batty Shaw), Clin. 112
Alcohol, effect on gout, Med. 78
—— —— on low blood-pressure, Med. 62, 77
——, gas-production of intestinal bacteria from, entirely or mainly undiminished, Path. 102, 103, 105, 109
—— injection of superior maxillary and supra-orbital nerves, anæsthesia produced by, illustrated by case of tic douloureux (W. Harris), Neur. 52

Alcohol, question of permissibility to subjects of high blood-pressure, Med. 78, 79
——, reaction of arteries to, Therap. 79
—— recommended for young children in the eighteenth century, Child. 122, 123
——, sclerosis of kidneys due to, Med. 79
Alcoholics easy to hypnotize, Anæsth. 23
——, need of probation and preparation before electro-therapy, Electr. 103, 104
von Aldor, action of atropine in reducing acid-values of stomach contents, quoted, Med. 24
Aldrich-Blake, Miss, abdomino-anal method of operating for rectal cancer, quoted. Surg. 129
Alexandra Institute of Baths (London), Baln. 98
Algeciras, suitable health resort for neurasthenia, Baln. 42
Algiers, suitable health resort for neurasthenia, Baln. 42
Alimentary tract, introduction of tuberculous infection by way of, Child. 205, 229, 231
—— ——, infection through tuberculous milk by way of, Child. 220, 221
Alkaloids of aconite, Therap. 190
Allbutt, Sir T. Clifford, effects of hard drinking water, quoted, Baln. 121
——, on tuberculous contagion, quoted, Epid. 166
ALLCHIN, Sir W. H.—Discussion on the electrocardiographic method, Med. 99
Allelomorph, definition of, Med. 6
—— for absence of quality, Med. 6, 7
—— for presence of quality, Med. 6, 7
—— —— ——, dominant or recessive, Med. 6, 7
Allingham's dilator (modification) in rectal examination, Obst. 330, 331, 336
Aloes, smearing of nipples with, to overcome child's dislike to strange food, Child. 119
Alopecia areata, Derm. 55
—— —— in relation to dental trouble, Odont. 74
——, cicatricial, Derm. 102
——, complete, treatment with thyroid gland, Path. 301, 303
—— in syphilitic subject (J. H. Sequeira), Derm. 150
—— of acquired syphilis in boy, aged 7 (J. H. Sequeira), Derm. 79
Alps (The), observations on blood-pressure made on, Med. 76, 77
Altitudes, high, danger of hæmoptysis to middle-aged subjects of phthisis at, Med. 60, 76
—— —— —— ——, to subjects of arterio-sclerosis, Med. 59, 60
—— ——, effect on blood-pressure, Med. 76, 77
—— ——, rise in low blood-pressure produced by, Med. 58, 59
Alum, solution of, electrical introduction in treatment of gastric ulcer, Elect. 42
Alveolar process, closure of cleft in, by Bardeleben's operation, Surg. 174, 177
Alypine, Therap. 56
Alzheimer, description of granule cells (Körnchenzellen) present throughout cortex of brain in cases of amaurotic idiocy (Tay-Sachs disease), Path. 177, 193
——, fatty degeneration of cytoplasm of neurones in amaurotic idiocy (Tay-Sachs disease), Path. 178
Amboceptors, free, functions of, Path. 276
America, sporotrichosis in, Derm. 114
Americans, limited knowledge of British health resorts possessed by, Baln. 21, 22
Amino acids in rectal feeding, Therap. 73
—— ——, ultimate products of protein disintegration, Therap. 70
—— ——, utilization in anabolism, Therap. 70
Ammonium, urate of, in uric acid calculus, cancellous variety, Path. 118
Amnesia (hysterical loss of memory), Neur. 4, 5, 6, 7, 17
—— —— —— ——, cases of, Neur. 4
—— —— —— —— in subjects of hypnotism, Neur. 7
Amnio-embryonic cavity, origin of, Obst. 321
Amnion, development in case of very young tubal ovum, Obst. 321
Amniotic cavity, bacteriology of, during labour after rupture of membranes, Obst. 169, 185, 186, 195, 216

Amniotic cavity, prophylactic irrigation of, in doubtful cases of infection where membranes are ruptured, Obst. 173, 185, 186, 198, 199, 213
Amputation through knee-joint for benign cyst of tibia, Clin. 134
Amyl nitrate, reaction of arteries to, Therap. 89
—— ——, therapeutical value tested by accurate clinical records, Therap. 6
"Amyotonia congenita" or "myatonia congenita" (so-called): simple atrophic type of myopathy, case of (F. E. Batten), Child. 100
Anabolism, utilization of amino acids in, Therap. 70
Anæmia (Addisonian, idiopathic pernicious), case of (W. Hunter), Clin. 37
—— —— —— —— ——, glossitis in, Clin. 38
——, von Jaksch's case of (S. Neave) Child. 155
—— ——, relation to syphilis and rickets, Child. 157
—— of labyrinth, Otol. 123
—— (pernicious) associated with melanodermia (J. H. Sequeira), Derm. 26
—— ——, icteric ulcers of legs in (H. Batty Shaw) Clin. 110
—— (secondary) associated with achylia, two cases of, with comments (R. Hutchison), Clin. 141
—— (splenic), diagnosis, Clin. 70
—— ——, pathology in relation to surgical treatment, Clin. 66
—— ——, treatment by splenectomy (G. A. Sutherland and F. F. Burghard), Clin. 58
—— —— —— ——, blood-counts in cases after, Clin. 66, 68
—— —— —— ——, followed by fatal gastro-intestinal hæmorrhage, Clin. 69, 70
—— (splenomegalic), Clin. 130
Anæsthesia, "Cheyne-Stokes" respiration during, case of (C. Hughes), Anæsth. 37
—— (chloroform), bursting of gumma during, causing death by inspiration of gummatous material (W. J. McCardie), Anæsth. 25
——, degree of, under chloroform and ether, Anæsth. 7
—— (general), alleviation under hypnotism, Anæsth. 22
—— ——, examination of cases under, before operation for abdominal hysterectomy, Obst. 87
——, heart-failure during, Anæsth. 27, 28
—— (hysterical), distribution of, Neur. 6
—— ——, restricted field of personal consciousness in, Neur. 6, 17
—— (inhalation) preceded by hypodermic injection of drugs, Anæsth. 43-56
—— —— —— —— —— ——, vomiting under, Anæsth. 50, 51, 52, 53
—— —— —— —— —— —— in women, Anæsth. 50, 51
—— —— —— —— —— of morphine and atropine, Anæsth. 51, 52
—— —— —— —— —— of pantopon, Anæsth. 44, 45, 48
—— in post-pharyngeal abscess (R. Gill), Anæsth. 34
—— (light), prevention of post-anæsthetic vomiting by, Anæsth. 10
——, nasal reflex during (J. Blumfeld), Anæsth. 27
—— produced by alcohol injection of superior maxillary and supra-orbital nerves, illustrated by case of tic douloureux (W. Harris), Neur. 52
——, production in light stage, Anæsth. 21
—— (suggestive) obtained in waking state, Anæsth. 19
—— ——, superimposition in re-inforcement of analgesia produced by hypnotism, Anæsth. 16
—— (surgical) and suggestion, Anæsth. 22
—— ——, production by hypnotism (E. Ash), Anæsth. 13
—— —— —— ——, advantages of, Anæsth. 14
—— —— —— ——, difficulties in, Anæsth. 14, 17, 18
—— —— —— —— in obstetrics, Anæsth. 21
—— —— —— —— not yet suited for urgent operations, Anæsth. 23
—— —— —— ——, testing suitability of subjects for, before operation, Anæsth. 18
—— —— —— ——, theory of, Anæsth. 15
——, uneven, cause of post-anæsthetic vomiting, Anæsth. 9, 10

Anæsthetic, administration during operation for rectal cancers, Surg. 117
——, examination of cases of rectal cancer under, to determine question of operation, Surg. 107
Anæsthetics (general), after-effects of operations under, Anæsth. 2
—— ——, see also *Chloroform*, *Ether*, *Vomiting* (post-anæsthetic)
—— (local) in mouth, drugs used as (F. R. Smyth), Therap. 51-58, 60-62
—— ——, early experiments, Therap. 51, 52
—— ——, see also *Beta-eucaine*, *Cocaine*, *Novocaine*, *Orthoform*, *Stovaine*
Anæsthetization of Welsh miners difficult, Anæsth. 5, 11
Anal canal, Surg. 87, 95
Analgesia produced by hypnotism, Anæsth. 16
—— —— ——, re-inforcement by superimposition of suggested anæsthesia, Anæsth. 16
Anaphylaxis in tuberculosis, question of, Child 223
Andral, on tuberculous disposition, quoted, Epid. 156, 157
Andrewes, F. W., case of muscular hæmorrhage following typhoid fever, quoted, Surg. 50
—— and Horder, T. J., organisms causing sore throat in man, quoted, Epid. 79
Andrews, H. Russell, cases of association of corpus luteum cysts with hydatidiform mole, Obst. 220
——, discussion on case of sarcoma of uterus, Obst. 112
—— —— on operation for cancer of uterus, Obst. 103
Aneurysm, unusual case of (S. Melville), Electr. 76
—— (aortic), supposed, operated on, Clin. 23
—— ——, supposed, operated on, discovered at post mortem ten years later to be congenital abnormality, Clin. 23, 24
—— (aortic, thoracic) associated with œsophageal cancer, Electr. 23, 30
—— —— ——, descending, causing deviation of œsophagus, Electr. 23, 24
—— of aortic arch, occupying position of subclavian artery (C. A. Ballance), Clin. 48
—— (popliteal), communication on (1811), anticipating present knowledge of dynamics of circulation, Med. 151
Aneurysm (thoracic), detection by X-rays, Electr. 22, 23
—— ——, pulsatile swelling in third and fourth left intercostal spaces ? case of (H. French), Clin. 22
Angiokeratoma, Derm. 30
——, case of (H. G. Adamson), Derm. 45
—— —— (A. W. Williams), Derm. 95
——, geographical distribution, Derm. 45, 46
Angiomata of left loin and leg, case of (E. I. Spriggs and J. B. de W. Molony), Child. 85
Angiomatous swellings, symmetrical, in tuberculous subject (J. H. Sequeira), Derm. 28
Anglesey, outbreak of milk-borne sore throat in, Epid. 91
Animals, possible source of infection of poliomyelitis, Epid. 206
Ankle-joint, fracture at, correct alignment necessary, Surg. 4
——, treatment of fractures about, Surg. 18-22
Ankles, peculiar affection of, in lad, aged 18 (G. W. Dawson), Derm. 3
Annulus hæmorrhoidalis, Surg. 94
Anorexia nervosa, case of (J. Walter Carr), Child. 80
—— ——, treatment, Child. 80
Antelopes, cutaneous glands in, function, Path. 286
——, hair-mats in, over carpus, Path. 284
——, specialized glandular areas in, Path. 310
Anthracosis, Child. 205, 211, 229, 230
Anthrax, experimental production, Child. 218
——, outbreaks of, in cows, Epid. 73, 74, 102
—— —— ——, at Chicago, Epid. 74
—— —— ——, possibility of milk infection through, Epid. 74
Antibodies, see *Group* antibodies

Antibody and antigen, excess of, effect, Path. 274, 276
—— ——, quantitative relationship exists between, Path. 258
——, excess of, unfavourable to fixation of complement, Path. 257, 258
Antigen and antibody, excess of, effect, Path. 274, 276
—— ——, quantitative relationship exists between, Path. 258
Antiseptic power of sulphur, Therap. 17
—— property of sulphurous black mustard oil, Therap. 23
Antiseptics, intestinal, action and use of sulphur and sulphur compounds as (R. B. Wild), Therap. 13-24
—— ——, inefficacy of, Therap. 14
Antisera for complement fixation experiments, method of preparation, Path. 256
Antiserum derived from rabbit after intravenous injection of paratyphoid A, Path. 262
——, excess of, effects, Path. 276
——, extreme dilution renders differentiation between *Bacillus typhosus*, *Bacillus paratyphosus* B, *Bacillus paratyphosus* A, possible, Path. 252
——, preliminary titration of, Path. 259, 260
Anti-thyroid preparations, action of, Path. 233-236
Antra (maxillary), operation on, followed by osteomyelitis, Laryng. 143
Antrostomy (nasal) for antrum suppuration, followed by blocking of nasal duct (Dan McKenzie), Laryng. 102
Antrum (maxillary), empyema of, in man, aged 71, becoming malignant (H. J. Davis), Laryng. 30
—— —— ——, in boy (H. J. Davis), Laryng. 31
—— —— (left), large fistulous opening into anterior wall of (J. Donelan), Laryng. 18
—— ——, skiagraphy of, Electr. 90
——, operations on, by nasal route, new form of burr for use in (S. Hastings), Laryng. 105
—— —— (radical) on, by intranasal route, new form of burr for use in (W. H. Kelson), Laryng. 106
—— (right) floor of, tooth removed from, in patient whose corresponding nasal cavity was obstructed by nasal polypus (H. Tilley), Laryng. 98
——, suppuration, nasal antrostomy followed by blocking of nasal duct (Dan McKenzie), Laryng. 102
Anubis baboon, hairless area in, Path. 310
Anuria, complete treatment by nephrostomy, Clin. 162
Anus, correct significance of term, Surg. 87
——, delimitations of, Surg. 95
——, natural, attempt to preserve, after operation for rectal cancer, Surg. 103, 108, 119, 127, 130
—— of fœtus, Surg. 89, 90, 94-98
Anxiety as cause of neurasthenia, Baln. 44
Aorta, aneurysm of arch of, occupying position of subclavian artery (C. A. Ballance), Clin. 48
—— (atheroma of), detection by X-rays, Electr. 26, 29
——, dilatation of, increased obliquity of long axis of heart in, Electr. 25, 27, 28, 29
——, stenosis of, congenital, case of (C. Riviere), Child. 85
—— (thoracic), see *Aneurysm* (aortic, thoracic)
Apenta water, aperient action of, effect on low blood-pressure, Med. 61
Aperients, effect on low blood-pressure, Med. 61
Apert, collection of cases of amaurotic family idiocy, Path. 167
Apes (anthropoid), development of premolars in, Odont. 112
Aphonia, case of, for diagnosis (C. Horsford), Laryng. 16
—— —— (T. J. Faulder), Laryng. 42
——, functional, in boy, aged 15 (L. H. Pegler), Laryng. 115, 116
——, recurring, in a woman, aged 36 (G. C. Cathcart), Laryng. 82
Apparatus, three simple pieces of (G. B. Batten), Electr. 77
Appendices epiploicæ, Surg. 92
Appendicitis, abscess in Douglas's pouch resulting from, Obst. 348

Appendicitis and adnexal inflammation, differential diagnosis between, Obst. 357
—— and dysmenorrhœa, causal relationship between, Obst. 356
—— complicating diseases of adnexa, Obst. 342, 343
——, interstitial hæmorrhage in, Obst. 347
—— in women, Obst. 347
—— ——, adhesions between wall of appendix and cæcum in, Obst. 347
—— involving adnexal inflammation, Obst. 354, 355
—— secondary to diseases of adnexa, Obst. 354, 355
——, sex-incidence discussed, Obst. 349, 350
Appendiculo-ovarian ligament, Obst. 343-347
Appendix, actinomycosis in neighbourhood of, Clin. 81
—— and cæcum, adhesions between wall of, in appendicitis in women, Obst. 347
—— and ovary, paths of infection between, Obst. 343
——, base of, location of, Obst. 354
——, concretion in, skiagram of (A. D. Reid), Electr. 73
——, infection from adnexa to, conditions favouring, Obst. 357
——, long, in women, effects of, Obst. 348
——, obliteration of lumen of, Obst. 347
——, relation to causation and surgical treatment of affection of the adnexa (H. Macnaughton-Jones), Obst. 341-358
—— —— —— —— —— —— —— ——, cases illustrating, Obst. 341, 342
——, removal of, in operations on pelvic organs, question of, Obst. 350-353
——, to adnexa, infection from, special conditions favouring, Obst. 357
Arabinose, fermentation by different strains of *Bacillus typhosus*, Path. 49
—— —— —— —— —— ——, time required, Path. 46
Arcachon, Baln. 18
Archives générales d'Hydrologie, commencement of (1891), Baln. 79
Argelès-Gazost, hypodermic administration of waters at, Baln. 108
Argon, discovery of, in atmosphere, Baln. 3, 4
—— in medicinal waters, Baln. 108
Arm, congenital elephantiasis of (C. T. Dent), Child. 24
——, extension of, during manipulation for dislocation of head of humerus, Surg. 5, 6
Armand-Delille and Boudet, case of progressive spinal muscular atrophy of infants and young children, Neur. 69
Armlet method for recording blood-pressure, Med. 77
ARMSTRONG, H. G.—Discussion on after-history of albuminuria in adolescence, Med. 123
——, prevalence of measles at Wellington College, quoted, Child. 175
ARMSTRONG, W.—Discussion on balneological treatment of neurasthenia, Baln. 44
——, discussion on treatment of cancer by X-rays and radium, Electr. 66
Army (German), cases of traumatic myositis ossificans reported in, Surg. 133
Arnold, dust inhalation and phthisis, quoted, Child. 228
Arnold's alternating stress test for cast and rolled gold plates, Odont. 60
Arsenic, secondary rays derived from, therapeutic effects, Electr. 67
—— (colloidal) in arsenical waters, Baln. 105
—— compounds ("606"), possible action of, on lecithin of protozoa, Path. 2
—— —— in human trypanosomiasis, possible effects, Path. 16, 17
—— in sarcomatosis cutis, Derm. 23
Arteria hæmorrhoidalis superior, Surg. 94
Arteries, action of ergot on arterial pressure, Therap. 146
——, reaction to drugs (D. Cow), Therap. 77-96
——, small, changes of, in human trypanosomiasis, Path. 30, 31
Arterio-sclerosis as cause of osteo-arthritis, Baln. 28
——, causation of, not referred to hard drinking water, Baln. 121
——, causes of, Med. 80
——, excessive consumption of tea, factor in causation of, Med. 63

Arterio-sclerosis, high altitudes dangerous to subjects of, Med. 59, 60
——, tobacco-smoking as cause of, question discussed, Med. 64, 65
Artery (carotid, internal), fatal hæmorrhage from, in case of sloughing tonsil, Child. 204
—— (common iliac), course of, compared to axillary region as regards gland extirpation, Obst. 75
—— (pulmonary), infective polypoid thrombus in, extending into right ventricle, mistaken for patent ductus arteriosus (T. R. Whipham), Child. 199
—— ——, stenosis ? congenital (J. Galloway), Clin. 82
—— —— ——, congenital, case of. estimation of total oxygen capacity and total blood volume, Clin, 88, 89
—— —— —— ——, case of, with special consideration of nature of secondary blood changes (F. Parkes Weber and G. Dorner), Clin. 85-94
—— —— —— —— (congenital heart disease), Child. 191
—— —— —— —— —— —— (J. A. Wright), Child. 190
—— (subclavian), aneurysm of aortic arch occupying position of (C. A. Ballance), Clin. 48
—— (uterine), lymphatic gland on, Obst. 102
Arthritis, chronic, prevalent in damp districts and climates, Baln. 28, 29
—— consecutive to pyorrhœa alveolaris, Clin. 141
—— deformans, low blood-pressure in subjects of, Med. 74
—— (degenerative), treatment by subthermal baths, Baln. 97
——, differential diagnosis of different forms of, Baln. 35
—— due to oral sepsis, Odont. 35
—— (hypertrophic), Baln. 34
—— of left wrist-joint, association of metastatic ophthalmitis with, Path. 242, 243
—— —— —— and metastatic ophthalmitis, simultaneous onset without constitutional symptoms, Path. 242
—— (rheumatoid), association of Graves's disease with, Child. 36
—— ——, case of (F. Langmead), Child. 33
—— ——, connexion with ovary, presumed, Baln. 36
—— ——, distinct from osteo-arthritis, Med. 38
—— ——, excretion of uric acid in, Baln. 59
—— ——, rubbing sound heard in auscultation of joints in, Med. 38
—— ——, success and failure of electro-therapy in, Electr. 109
—— ——, treatment by thyroid extract, Child. 35, 36
—— (villous, chronic), Baln. 34
—— —— ——, bony changes in, Baln. 34
—— —— ——, name of, " chronic articular rheumatism " applied to, Baln. 34
Arthropathies, changes commence in bone in, Baln. 24
——, hyperæmia of synovial membranes with villous hypertrophy in, Baln. 24
——, osteo-arthritis and, essential difference between, Baln. 24
——, relation of osteo-arthritis to, Baln. 24
Asafœtida, Therap. 22
Aschscharumow, physiological effects of aconite, quoted, Therap. 157
Ascites, hæmorrhagic, Surg. 49
—— and hepatic cirrhosis, following splenomegaly with recurrent jaundice (F. P. Weber), Child. 62
Ash, Edwin.—Hypnotism in relation to surgical anæsthesia, Anæsth. 13-24
Ashby, report of outbreak of milk-borne diphtheria by, Epid. 83
Asphyxia, intercurrent, under ether, as standard of comparison for intercurrent asphyxia under chloroform, Path. 206, 217
—— under chloroform (A. G. Levy), Path. 205-222
Association, mental, inappropriate instances in hysteria, Neur. 12
Astasia (functional) suggested by case of congenital brain defect with curious stamping gait and tendency to fall backward (F. E. Batten), Neur. 33
Astasia-abasia, nature of, Neur. 8

Asthma, treatment by diathermic current, Electr. 8
Astigmatism, hypermetropic, with macular change (J. C. W. Graham), Child. 198
Astley Sanatorium, food-poisoning outbreak at, following eating of canned meat, Epid. 66
Astragalus, fracture of, in conjunction with split fracture of lower end of tibia, Surg. 18
—— ——, transverse, treatment, Surg. 19, 20
Asylum statistics of pulmonary tuberculosis, Epid. 180-182
Asylums, typhoid carriers in, Epid. 119, 120, 134
Asymmetry (bodily) as stigma of degeneracy, Odont. 106
—— ——, prehistoric example of, Odont. 109
—— (facial), possible cause of (W. Rushton), Odont. 105-109
Atavism, multiple supernumerary teeth regarded as examples of, Odont. 46
Ataxy (cerebellar), case of (F. E. Batten), Neur. 19
—— —— —— (J. A. Wright), Child. 190
—— (cerebellar congenital), case of (F. E. Batten), Neur. 20
—— (functional)? case of peculiar gait in child (J. A. Wright), Child. 191
—— (spino-cerebellar), probable form of (family nervous disease, case for diagnosis), Child. 198, 199
Ateleiosis, asexual, case of (H. Gilford), Clin. 34
Athens, plague of, recovery of victims after loss of extremities, Child. 13
Athetosis, relief of, by resection of posterior spinal nerve-roots, Surg. 243
Athletes (adolescent), albuminuria in, Med. 110, 124, 128
——, cyclic albuminuria in, Med. 110, 111
Atkins and Macnaughton-Jones, case of atrichosis, quoted, Path. 302
Atlas, skiagraphy of, Electr. 90
Atmosphere, see *Air*
Atmospheric pressure, increase or diminution of, causing rupture of tympanic membrane, Otol. 94, 95
Atoxyl in sarcomatosis cutis, Derm. 23
——, possible action of, on lecithin of protozoa, Path. 2
Atrichosis (congenital), human, treatment with fresh thyroid gland, Path. 301, 302
—— —— in animals, Path. 292-301
—— ——, morphological or zoological variations of, Path. 304
—— ——, pathological, Path. 304
—— ——, question of relationship to thyroid deficiency discussed, Path. 299
—— (human), examples of, Path. 301-304
Atrophy (macular) associated with lupus erythematosus (E. G. Graham Little), Derm. 143
—— —— due to exposure to sun's rays, Derm. 144
—— associated with tuberculosis, Derm. 123
——, case of, photographs of (H. G. Adamson), Derm. 2
—— in syphilitic patient (H. G. Adamson), Derm. 122
—— preceded by local inflammation, Derm. 123
Atrophodermia striata et maculata, with phthisis and lichen scrofulosorum (H. G. Adamson), Derm. 1
Atropine, action on heart, Anæsth. 54, 55
——, administration of, effect on salivary secretion, Med. 31
—— ——, subcutaneously and by mouth, producing diminution in secretion of gastric juice, Med. 23, 24
—— —— —— ——, producing reduction in acid-values of stomach contents, Med. 23, 24
——, combination with scopolamorphine hypodermically injected before ether inhalation, advantage of, Anæsth. 47
—— —— —— hypodermically injected before inhalation anæsthesia, Anæsth. 46, 47
——, effect on pulse-rate in cases under the influence of digitalis (M. D. Silberberg), Therap. 192-210
—— sulphate, hypodermic injection before ether or chloroform inhalation, Anæsth. 53
——, see also *Morphia* and atropine, *Strychnine* and atropine, hypodermic injection

Auché, gangrene of extremities following infectious disease, Child. 11
Auenbrugger, first publication of his work on percussion (1761), Med. 150
Auricle, avulsion of, Otol. 80
——, bites of, Otol. 80
——, incised wounds of, followed by malignant degeneration of resulting scar-tissue, Otol. 80
—— (left), deformity of (G. S. Hett), Otol. 42
—— and meatus, congenital malformation of (A. H. Cheatle), Otol. 25
—— (cardiac), fibrillation of, Med. 92, 96
—— ——, delirium cordis identical with, Med. 92, 93, 96
—— ——, electrocardiograms showing, Med. 92, 93
—— ——, in animals, Med. 100
—— ——, in man, evidence for, Med. 100
—— ——, mechanism of, Med. 93
——, see also *Fibrillation*, electric effects in, auricular
Auricles, both, infiltration of (? chronic lymphangitis), in woman (D. McKenzie), Otol. 37
—— (cardiac) and ventricles, isolated, independent rhythm of heart-beat, Therap. 97
Auricular complex, Med. 85, 86, 88
—— contraction, premature, Med. 87, 88
—— —— ——, electrocardiogram showing, Med. 87
—— ——, sequential, Med. 88
—— representative, electrocardiogram showing, Med. 86
Auriculo-ventricular bundle, Med. 86
—— ——, clamping of, results, Therap. 97, 98
—— ——, congenital syphilitic affection of, Clin. 41, 42
—— ——, effect on, of aconitine, Therap. 107
—— —— ——, of adrenalin, Therap. 107
—— —— ——, of electrical, chemical, and thermal stimulants, Therap. 103
—— ——, excitation and section of (W. E. Dixon), Therap. 97-113
—— ——, see also *Bundle of His*
Auscultation of joints (A. E. Garrod, F.R.S.), Med. 35-39
—— ——, elimination of extraneous sounds, Med. 35, 36, 39
—— ——, how to determine meaning of sounds, Med. 36, 37
—— —— in diagnosis of disease, Med. 35, 39
—— —— —— —— by stethoscope with chest-piece, Med. 35, 36
—— of thorax, sounds heard in, knowledge of meaning, how built up, Med. 36
Australia, epidemics of poliomyelitis in, Epid. 201, 203
——, map showing, Epid. 203
Australian aborigines, hypotrichosis in family of, Path. 303
Austria, establishment of hydrotherapy upon scientific basis in, Baln. 85, 86, 97
Austro-Hungary, teaching of medical hydrology in, Baln. 115
Auto-condensation in electrotherapy, Electr. 104, 106, 107, 108
Auto-inoculation in bronchitis, Therap. 44-47
——, rôle of, in medicine (C. Riviere), Therap. 39-50
Auto-intoxication and neurasthenia, Baln. 44
Auto-reinfection, syphilitic, case of (J. E. R. McDonagh), Derm. 129
Aviemore, districts round, suitable for neurasthenics, Baln. 42
Axenfeld, symptoms of pseudoglioma, quoted, Path. 248
Axilla, region of, course of common iliac artery compared to, as regards gland extirpation, Obst. 75
Axis, skiagraphy of, Electr. 90

"Babees Book (The)," Child. 110
Babinski, hysterical anæsthesia, quoted, Neur. 6
Babinski's sign in amaurotic idiocy (Tay-Sachs disease), Path. 153
Baboon, callosities in, how differing from those in camels, Path. 310
——, see also *Guinea* baboon
Baby-farmers in sixteenth century, Child. 114
—— —— ——, male as well as female, Child. 114
Bacilli, differentiation by agglutination tests, Epid. 52
—— ——, by complement-fixation method, Epid. 71
—— —— by fermentation tests, Epid. 70
—— isolated from urine, comparative gas- and acid-forming powers in sugars, Path. 97, 98, 107
—— of colon group, Epid. 63
Bacillus Aertryck, Path. 253, 254
—— ——, see also *Bacillus suipestifer*
—— *alkaligenes*, Epid. 142
—— *bifidus* and pancreatic infantilism, Child. 61
—— *Bruxelles*, Path. 253
—— *cadaveris* (Klein), presence in canned meat causing blowing of tins, Epid. 67
—— *coli*, chemical activities, Path. 106, 107
—— ——, dextrose-peptone water cultures of, influence of benzoates on, in diminishing gas-production, Path. 98
—— ——, differentiation from *Bacillus typhosus*, Path. 252
—— ——, fermentation reactions of coliform organism freshly isolated from fæces practically identical with those of, Path. 105
—— ——, group of, fermenting lactose, Epid. 125
—— ——, grown on monochloracetic acid agar medium, diversity of size of resulting colonies, Path. 98, 99
—— ——, infection by, accompanying nematode invasion in animals and birds, Child. 218
—— ——, mastitis in cow due to (one case), Epid. 78
—— ——, new variant, fermentation reactions compared with those of normal strain, Path. 101
—— —— —— ——, durability of, Path. 105
—— —— —— ——, fermentation properties, Path. 106, 107
—— —— —— ——, grown under anaerobic conditions in Buchner's tube or anaerobic gas tube, fermentation results, Path. 107
—— —— —— ——, identification of, Path. 102
—— —— —— ——, produced by growth on monochloracetic acid agar medium, Path. 100, 101
—— ——, normal and variant strains, complement-fixation experiment with extracts of, Path. 103, 104
—— —— and *Bacillus typhosus*, intermediate forms in intestine of dog, Epid. 118
—— —— —— ——, strain intermediate between isolated from urine, Path. 97
—— —— and streptococcus causing puerperal infection, Obst. 167, 188
—— —— vaccine, injection before operating for rectal cancer, Surg. 130
—— *diphtheriæ* (Klebs-Löffler), inoculation into cows, Epid. 85
—— —— —— —— ——, subsequent recovery at site of inoculation, Epid. 85
—— —— ——, isolation from ulcerated teats, Epid. 82, 83
—— *Düsseldorff*, Path. 253
—— *enteritidis* (Gaertner), Epid. 137
—— —— —— and food-poisoning, Epid. 64, 65
—— —— ——, bacilli approximating most closely to, connected with causation of food-poisoning, Epid. 63
—— —— ——, differentiation and identification, Epid. 52
—— —— —— —— from *Bacillus suipestifer*, date of, Epid. 58
—— —— —— —— from *Bacillus paratyphosus* B, Epid. 67
—— —— ——, discovery of, Epid. 52
—— —— ——, epizootics associated with, Epid. 61

Bacillus enteritidis (Gaertner), experimental infection of animals with, results, Epid. 68, 69
—— —— —— grown on monochloracetic acid agar medium, diversity in size of resulting colonies, Path. 99, 100
—— —— ——, infection of cows by, Epid. 74
—— —— —— —— ——, decomposition of milk under, Epid. 74
—— —— ——, meat infected with, causing food-poisoning, Epid. 74, 102
—— —— ——, new variant, fermentation reactions compared with those of normal strain, Path. 103
—— —— —— —— ——, identification, Path. 105
—— —— —— —— —— produced by growth on monochloracetic acid medium, Path. 103
—— —— ——, rarity of occurrence, Epid. 61
—— —— ——, rat viruses containing, danger of, Epid. 61, 69
—— —— ——, resistant to salting and pickling, Epid. 69
—— —— ——, strains of, difficult to distinguish from those of *Bacillus suipestifer* (*Bacillus Aertryck*), Epid. 70
—— —— ——, varieties of, Epid. 64
—— —— —— ——, rapid multiplication, Epid. 65
—— *Friedländer*, causative organism of scleroma of nasopharynx, Laryng. 7
—— *fusiformis* present in ulcerative stomatitis in children, Child. 73
—— *Grünthal*, fermentation reactions of coliform organism freshly isolated from fæces practically identical with those of, Path. 105
—— *lactis aerogenes*, variant of, fermentation reaction, Path. 105
—— *Morseele*, Path. 253, 254
—— *nodulifaciens bovis*, Path. 253
—— *paratyphosus* as secondary invader in typhoid fever, Epid. 117, 137
—— ——, differentiation from *Bacillus typhosus*, Path, 252
—— ——, frequency of, Epid. 137
—— —— (A), antiserum derived from rabbit after intravenous injection of, Path. 262
—— —— ——, differentiation and identification, Epid. 52
—— —— ——, discovery of, Epid. 53
—— —— ——, geographical distribution, Epid. 53, 54
—— —— ——, outbreaks associated with, Epid. 117, 118
—— —— ——, rabbit injected with, use of serum from, Path. 263, 264
—— —— (B), association with diseases other than paratyphoid fever, Epid. 54, 55, 70, 71
—— —— ——, differentiation and identification, Epid. 52, Path. 252
—— —— —— —— from *Bacillus Aertryck* by complement-fixation method, Path. 263, 266-270
—— —— —— ——, see also *Bacillus suipestifer*, below
—— —— ——, difficulty of obtaining pure strains, Epid. 70
—— —— ——, discovery of, Epid. 53
—— —— ——, distinction from *Bacillus enteritidis*, Gaertner, Epid. 67
—— —— ——, distribution of, Epid. 56, 57
—— —— —— in gall-bladder, Epid. 56
—— —— —— not associated with summer diarrhœa, Epid. 55
—— —— —— not found in fæces or urine, Epid. 55
—— —— —— —— —— —— of convalescents from typhoid fever, Epid. 55
—— —— —— —— in meat, Epid. 55
—— —— ——, outbreaks associated with, Epid. 117, 118
—— —— —— —— of gastro-enteritis due to, Epid. 56
—— —— ——, presence in healthy individuals, Epid. 54, 55, 70, 71
—— —— ——, specificity of, Epid. 55
—— *pestis*, Epid. 136
—— *Posen*, Path. 253
—— *prodigiosus*, aspiration into lungs during feeding experiments with, Child. 213
—— —— experiments to prove inhalation of, Child. 207

Bacillus pseudo-tuberculosis, Path. 254
—— *psittacosis*, Path. 253, 254
—— *pyocyaneus*, presence in cavernous sinus thrombosis, Odont. 116, 117
—— *Sirault*, Path. 253
—— *suipestifer* (*Aertryck*), Epid. 55, 64, Path. 254
—— ——, carriers of, experimental production, Path. 70, 71
—— ——, differentiation from *Bacillus enteritidis* (Gaertner) Epid. 58
—— —— —— from *Bacillus paratyphosus* B by Castellani's absorption test, Epid. 52, 67
—— —— —— —— —— by complement fixation method, Path. 263, 266-270
—— ——, discovery of, Epid. 53
—— ——, distribution of, limited, Epid. 59
—— ——, habitat of, Epid. 58
—— —— in hog-cholera, Epid. 136
—— ——, presence in food, Epid. 58, 59
—— ——, relation to food-poisoning, Epid. 59, 60, 64
—— ——, secondary invader in swine fever, Epid. 59
—— ——, strains of, difficult to distinguish from those of *Bacillus enteritidis* (Gaertner), Epid. 70
—— *tuberculosis*, Epid. 27
—— —— aspiration of, not a general cause of phthisis, Child. 214
—— —— (bovine), dose needed to produce infection, Child. 220, 221
—— ——, experiments to prove inhalation of, Path. 207
—— —— in sputum as indication of infection, Epid. 46, 47, 48
—— —— injection experiments with, Child. 231
—— ——, mastitis in cow due to (one case), Epid. 78
—— *typhi murium*, Path. 252, 254
—— *typhosus* as secondary invader in typhoid fever, Epid. 117, 137
—— ——, biological characteristics (B. Bradley), Path. 43
—— —— —— ——, modifications, "sugar" media employed in testing, Path. 44
—— —— —— ——, results as to identicity in different strains, Path. 44-47
—— ——, carriers of, see *Typhoid fever*, carriers of
—— ——, different strains, fermentation of arabinose by, Path. 48
—— —— —— —— —— —— ——, time required, Path. 46
—— —— —— —— —— ——, of dulcitol by, Path. 48
—— —— —— —— —— —— ——, increased power, Path. 46
—— —— —— —— —— —— —— ——, not generally sustained, Path. 49, 50
—— —— —— —— —— —— —— ——, varies with length of sojourn in dulcitol-containing medium, Path. 48, 52
—— —— —— —— —— —— —— ——, transference to agar cultures, Path. 47, 48
—— —— —— —— —— ——, time required, Path. 46
—— —— —— —— —— fermentation of lactose or saccharose not effected by, Path. 51, 52
—— ——, differentiation from *Bacillus paratyphosus* by complement fixation, Path. 252
—— —— —— —— ——, presence of group reaction in, Path. 252
—— ——, excretion by carriers, period of, Epid. 3
—— ——, fermentation of lactose by, Epid. 70, 142
—— —— ——, properties, variations, Epid. 142
—— ——, formic acid not gas, produced by, Path. 106
—— ——, gas produced by, when grown with new variant of *Bacillus coli*, Path. 106
—— —— grown on monochloracetic acid agar medium, diversity in size of resulting colonies, Path. 100
—— ——, healthy carriers of, Epid. 126
—— ——, mutation in respect of isodulcite, Epid. 142
—— ——, *paratyphosus* B, and *paratyphosus* A, differentiation between, how rendered possible, Path. 253
—— ——, *paratyphosus*, and *Bacillus Aertryck*, extracts prepared from strains of, and anti-typhoid serum, reaction between, Path. 260-262

Bacillus typhosus, presence in bone abscesses, Epid. 2
—— —— —— in carriers, demonstration by Weber's method, Epid. 120
—— —— —— in fæces, Epid. 4
—— —— —— in London drinking water, Epid. 126
—— —— —— in urine, Epid. 2, 135
—— ——, saprophytic existence, theory of, Epid. 2
—— ——, titration of antityphoid serum with extract made from another strain of, Path. 257
—— —— (type Flexner), occurrence in diarrhœa stools, Epid. 126, 137, 145
—— ——, variability in virulence, Epid. 25
—— —— B, serum from rabbit injected with, titrated with the homologous extract, Path. 263, 265
—— —— and *Bacillus coli*, strain intermediate between, isolated from urine, Path. 97
Back, tumour on, probably dermoid (O. L. Addison), Child. 165
Bacon, W. B.—Discussion on inflammatory eye conditions due to oral sepsis, Odont. 34
——, Presidential Address, Odontological Section, Odont. 1
Bacteria, aspiration into the lungs during feeding experiments, Child. 212, 213
—— ——, risk of, Child. 214
——. classification by differentiation method, Path. 251
——, conceivable portals of entry for, into living body, Child. 206
——, differentiation, by means of complement-fixation and agglutination methods compared, Path. 252
—— ——, by agglutination tests, Epid. 136
—— —— by sugar fermentation tests, Epid. 136
—— —— of, group-reaction in presence of, Path. 252
——, greater number incorporated by bread-crust than bread-crumb in mouth, Odont. 93
—— in canned meat, resistant to heat, Epid. 66
——, increased activity of, in spring, Therap. 14
—— of mouth, special not general forms, factor in production of caries, Odont. 94
—— penetration of normal mucous membranes by, without causing lesion, Child. 217
——, plaques and masses of, on and about teeth, removal to prevent dental caries, Odont. 94, 101
—— presence in bronchi, Child. 206
—— (air-borne), entering lungs, experiments to prove, Child. 207
—— (anaerobic, putrefactive) infective in genital tract, Obst. 170, 188
—— (intestinal), gas-forming powers of, variability in (W. J. Penfold), Path. 97-109
—— ——, gas-production by, from alcohol, entirely or mainly undiminished, Path. 102, 103, 105, 109
—— —— —— from sugar or sugar-yielding bodies, abolished entirely or almost, Path. 102, 103, 105, 109
—— —— ——, testing by use of Durham's tubes, Path. 100, 105
—— —— ——, summary and conclusions, Path. 109
—— ——, mutation of, Epid. 124-127, 136, 137, 141, 142, 146
—— (pathogenic), liquor amnii good culture medium for, Obst. 170
—— ——, temperature requisite to destroy, Epid. 135
Bacterial extracts for complement-fixation experiments, method of preparation, Path. 255
Bacteriology of amniotic cavity during labour after rupture of membranes, Obst. 169
—— of chloroma unworked, Path. 69
—— of food-poisoning, Epid. 51, 52
—— —— (F. A. Bainbridge), Epid. 51, 52, 58
—— of mastitis in cows, Epid. 77-81, 103
—— of paratyphoid fever, Epid. 51, 52
—— of post-nasal catarrh: preliminary study of fifty cases (C. Ernest West), Otol. 43-50
—— of thrombosis of cavernous sinus due to oral sepsis, Odont. 116, 117
—— of vagina during labour, Obst. 169
—— —— during pregnancy, Obst. 169
Badgerow, G. W.—Case of lingual thyroid, Laryng. 81

BADGEROW, G. W.—Small tumour of soft palate, Laryng. 13
——, and POWELL, H. FITZGERALD.—Tumour of post-nasal space, Laryng. 12
—— ——, ulceration of soft palate, Laryng. 11
Baermann, cases of paratyphoid fever (A) in Sumatra, quoted, Epid. 53, 54
Bagellardus, Paulus, mention of infant-feeding in works of, Child, 110
Bagnoles d'Orne, waters of, useless after four days' preservation, Baln. 72
BAGSHAWE, F.—Discussion on calcareous (hard) drinking water, Baln. 127
BAINBRIDGE, F. A.—Discussion on typhoid carriers and contact infection, Epid. 137
——, discussion on value of rectal feeding, Therap. 73
—— —— on rôle of auto-inoculation in medicine, Therap. 49
——, epizootics associated with *Bacillus enteritidis* noted by, Epid. 61
——, habitat of *Bacillus suipestifer*, quoted, Epid. 59
——, the ætiology and epidemiology of paratyphoid fever and food-poisoning, Epid. 51-72
——, and Dudfield, R., outbreak of paratyphoid fever, quoted, Epid. 57
BALDING, E.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 48
BALDWIN, H.—Discussion on comparative elasticity and internal structure of cast and rolled gold plate, Odont. 66
——, discussion on distribution of nerve-fibres of dental pulp, Odont. 55
BALLANCE, C. A., M.V.O.—Aneurysm of aortic arch, occupying position of subclavian artery, Clin. 48
——, case of gumma of brain, Clin. 47
——, discussion on resection of posterior spinal nerve-roots, Surg. 248
——, injuries of middle and internal ear, Otol. 87
Ballner and Reibmayr, differentiation of *Bacillus typhosus* and *Bacillus paratyphosus*, quoted, Path. 252
Balneological and Climatological Section, special department of work, Baln. 4, 9, 10
—— —— ——, suggestion of appointment of Committee to formulate and describe types of joint trouble, Baln. 36
—— —— ——, work of, objects, Baln. 4, 5
—— —— —— ——, summarized, Baln. 4, 5
Balneological treatment, external, of neurasthenia, Baln. 41
—— —— of neurasthenia (W. R. Gore), Baln. 37-44
Balneology and climatology, reasons for association, Baln. 67
—— distinct from hydrotherapy in Germany, Baln. 78
——, earliest references to, in literature, Baln. 1, 2, 3
——, lectures on, at University of Stockholm delivered from 1880, Baln. 116
——, origin and definition of, Baln. 75
—— —— ——, early limitations, Baln. 76
——, rational scientific applications of, advocated, Baln. 5
——, suggestion in, Baln. 5, 6
——, term "hydrology" preferable to, Baln. 99
Baltic Sea, Swinemünde, health resort on, Baln. 19
Baltimore, see *Johns Hopkins University*
BALY, C. F. P.—(1) An erosion cavity on the lingual aspect of a lower incisor; (2) dislocation of the pulp of a temporary incisor, the result of a fall, with subsequent removal of the pulp and apex intact, Odont. 69, 70
——, discussion on comparative elasticity and internal structure of cast and rolled gold plate, Odont. 65
Band form of morphœa in a girl, aged 7 (J. L. Bunch), Child. 27
Banti's disease, symptom-complex of, Clin. 70
—— —— and splenomegaly of inherited syphilis of children, Child. 69
Baratoux and Kirchner, syphilitic endarteritis in blood-vessels of mucous membrane of tympanic cavity, case quoted, Otol. 18
Barbone bufali, Surg. 48

BARCLAY, A. E.—Discussion on lantern slides of three cases, Electr. 75
——, discussion of unusual form of contraction of fingers, Electr. 73
Bardeleben's operation in closure of cleft in processus alveolaris, Surg. 177
Bardenheuer's modification of Kraske's operation for rectal cancer, Surg. 105
BARDSWELL, NOEL.—Case for diagnosis, Laryng. 15
Barger and Carr, ergot researches, quoted, Therap. 114, 115
Barium, presence in strong sulphur water, Baln. 60
—— chloride, reaction of arteries to, Therap. 83
Barkla, Professor, application of bismuth after X-ray treatment, Electr. 57, 62
Barlow, Sir T., Bt., cases of pseudobulbar paralysis, quoted, Neur. 25
Barnardo's (Dr.) Homes, treatment of scarlet fever and measles at, Child. 142-148, 152, 153
—— Village Homes, outbreak of scarlet fever at, cause of, Child. 182
—— —— ——, prevention of spread of measles at, Child. 182, 183
BARNES, STANLEY.—Discussion on case of slowly progressive right hemiplegia suggesting tumour, Neur. 46
——, discussion on case of toxic neuritis of doubtful nature, Neur. 21
Barnick, fatal case of fracture of petrous bone, quoted, Otol. 88
Barracks, supposed typhoid carrier in, Epid. 121, 122
Barraud, gangrene of extremities following infectious disease, quoted, Child. 11, 12
BARRIS, J.—Fibromyoma uteri causing acute intestinal obstruction, Obst. 108
BARROW, A. BOYCE.—Discussion on operative treatment of cancer of rectum, Surg. 115
Barrow-in-Furness, epidemic of poliomyelitis at (1910), Epid. 212-215
BARTON, G. A. H.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 9
——, discussion on death due to inspiration of gummatous material from gumma which burst during chloroform anæsthesia, Anæsth. 26
—— ——, on note on case of "Cheyne-Stokes" respiration during anæsthesia, Anæsth. 39
—— —— on use of drugs before inhalation anæsthesia, Anæsth. 52
Baruch, Professor S., occupant of Chair of Therapy in University of Columbia (New York), Baln. 116
——, on water as a remedial agent, quoted, Baln. 89, 90.
——, "Principles and Practice of Hydrotherapy" (1890), first treatise on subject in English language, Baln. 116
BARWELL, H. S.—Case of subglottic stenosis after tracheotomy, Laryng. 6
——, discussion on case for diagnosis, Laryng. 15
—— —— —— of fungating carcinoma of œsophagus treated by radium, Laryng. 8
—— —— on nasal reflex during anæsthesia, Anæsth. 30
BASKER, C. A.—Case for diagnosis: family nervous disease, Child. 198
Bastian, H. C., F.R.S., view regarding hysteria, quoted, Neur. 2
BATEMAN, M. G. YUNGE.—Discussion on calcareous (hard) drinking water, Baln. 128
BATESON, W., F.R.S.—Discussion on multiple cartilaginous exostoses, Med. 20
Bath, mineral waters at, Baln. 15
Bath-houses, Baln. 76
Bath-keepers, origin of, Baln. 76
Bath treatment, climatic factor in, Baln. 68
Bathing in sewage contaminated water, possible source of infection of poliomyelitis, Epid. 206
Baths, ancient Roman remains of, Baln. 3
—— (cold), avoidance in typhoid fever, Baln. 93, 94
—— ——, in treatment of fevers, first advocacy of, Baln. 83
—— —— —— ——, rules of Giannini for, Baln. 88, 89
——, effect on vasomotor centre, Baln. 93
—— (hot or thermal), best borne in cool places, Baln. 69
—— (hyperthermal), brief *plus* application, Baln. 94
—— (immersion), factor of hydrostatic pressure in, Baln. 73
—— in treatment of typhoid fever, Baln. 93
——, *modus operandi* of, Baln. 93

Baths (subthermal), clinical application, Baln. 96

—— ——, containing carbonic acid gas, sensation produced by, Baln. 109.

—— —— —— —— —— ——, alternative action, Baln. 109

—— —— in treatment of cardio-vascular and nervous diseases, Baln. 68

—— ——, prolonged, Baln. 97

—— —— ——, exchange of variable aërial for watery medium in, Baln. 97

—— (thermal), *plus* application, Baln. 95

—— at Spedale Santa Maria Nuova (Florence), Baln. 89

—— of Mont Dore, Baln. 73

——, see also *Hot air baths ; Hydrotherapy ; Turkish baths ; Vapour baths*

BATTEN, F. E.—Case of congenital cerebellar ataxia, Neur. 20

——, case of progressive cerebellar ataxia, Neur. 19

—— —— of spondylose rhizomélique, Neur. 40

—— —— of toxic neuritis of doubtful nature, Neur. 20

——, congenital brain defect, with a curious stamping gait and tendency to fall backward, suggestive of a functional astasia, Neur. 33

——, discussion on case of gastric crisis in ? tabes, Neur. 50

—— —— on cases of spondylose rhizomélique spondylitis with progressive muscular contracture, Neur. 44

——, progressive spinal muscular atrophy of infants (Werdnig-Hoffmann), Clin. 115

—— —— —— —— —— —— of infants and young children, Neur. 53-84

——, simple atrophic type of myopathy, so-called "myatonia congenita" or "amyotonia congenita," case of, Child. 100

——, slowly progressive right hemiplegia suggesting tumour (operation—no tumour found), Neur. 45

——, the epidemiology of poliomyelitis, Epid. 198-226

——, and Guthrie, L. G., cases of hemiplegia on one side associated with optic nerve atrophy on other, quoted, Child. 196

BATTEN, G. B.—Discussion on diathermic treatment, Electr. 9

——, discussion on Roentgen ray examination of œsophagus, Electr. 28

——, three simple pieces of apparatus, Electr. 77

BATTEN, R. D.—Discussion on inflammatory eye conditions due to oral sepsis, Odont. 35

Battle, W. H., and Shattock, S. G., case of diffuse cancellous osteoma of femur following fracture, quoted, Surg. 138

Bauer and Voit, absorption of proteins by rectum, quoted, Therap. 64

Bauer's qualimeter, for determining the hardness of an X-ray tube by measuring the potential through the tube (W. Deane Butcher), Electr. 71

Bayle, on idea of tuberculous diathesis, quoted, Epid. 155

BAYNES, DONALD.—Discussion on value of electricity in abdominal diseases, Electr. 45

Beadles, C. F., percentage-incidence of gall-stones based upon post-mortem records of Colney Hatch Asylum, quoted, Path. 88

Beale, L. S., kidney diseases and urinary deposits, quoted, Path. 133, 134, 135

Beard, epidemic of poliomyelitis at Carlisle (1910), Epid. 212, 213

Beddard, hair-mat in giraffe, Path. 283

BEDDOES, T. P.—Discussion on case of leucoderma syphiliticum, Derm. 103

——, discussion on case of syphilis, chyliform ascites, dropsy and albuminuria, Clin. 113

—— —— on specimens of hair from an aborignal Indian girl, Derm. 150

Bedford, outbreak of milk-borne sore throat at, Epid. 92

Beef infected with Gaertner bacillus, severe outbreak of food-poisoning following, Epid. 68

Beef-bone impacted in right lung, expelled by coughing, Laryng. 97, 98

Beer, recommended for young children in eighteenth century, Child. 123

Beevor, C. E., case of progressive spinal muscular atrophy of infants and young children, quoted, Neur. 68

Behring, portals of entry of tuberculosis, quoted, Child. 205

Belgium, hydrotherapy in, Baln. 89

Belgium, teaching of medical hydrology in, Baln. 116
Bell, W. Blair.—Nature of hæmatocolpos fluid, with deductions as to chemical composition of menstrual discharge and cause of its normal non-coagulability, also of acidity of vaginal secretion ; and character of obstructing membrane, with its bearing on development of vagina, together with report of three cases of hæmatocolpos, Obst. 234
——, primary abdominal pregnancy in rabbit, Obst. 228
——, transplantation of ovaries, Obst. 116
Belladonna, administration, in dyspepsia (F. C. Moore and L. Kilroe), Med. 23-24
—— —— —— in small doses, in relief of bullous dermatoses, Med. 32
—— —— —— of, inhibiting spasm of colon, Med. 31
—— —— ——, mode of action on gastric secretion, Med. 33, 34
—— —— ——, value of, in gastric carcinoma, Med. 33
—— —— —— (oral), before meals, control of hyperchlorhydria by, Med. 26, 29, 33
—— —— —— ——, before meal, effect on acid-values of stomach contents, Med. 25
—— —— —— —— —— —— —— —— ——, as associated with hyper-acidity, normal acidity, and hypo-acidity, Med. 25, 26
—— —— —— —— —— —— —— —— on peptic values of stomach contents, Med. 26-29
—— —— —— —— —— ——, relaxation of abnormal spasm of gastric musculature by, Med. 29, 30, 33
—— —— —— —— ——, volume of gastric contents not diminished by, Med. 31
Bellot and Sacquepée, epidemic of paratyphoid fever due to carriers, quoted, Epid. 57
Belvidere Hospital, Glasgow, outbreak of milk-borne sore throat at, Epid. 95
Bence, excess of red blood corpuscles in cases of splenomegalic polycythæmia, Clin. 86
Bengal, see *Hazaribagh district*
Benign growths, removal by non-operative measures, Electr. 51
—— ——, tendency to become malignant, Elect. 51
Bennett, F. J.—Discussion on cast and rolled gold plate, Odont. 65
——, discussion on distribution of nerve-fibres of dental pulp, Odont. 54
—— —— on experiments on bread, with reference to dental caries, Odont. 101
—— —— on inflammatory eye conditions due to oral sepsis, Odont. 32
——, the destruction of teeth by milk germs, Odont. 3
Bennett, Sir W. H., posterior spinal nerve-roots first divided by, Surg. 226
Bennett's fracture, Surg. 41, 45
Ben Rhydding, health resort suitable for foreign invalids, Baln. 21
Bentley-Taylor method of mounting mosquitoes (A. Powell), Path. 41
Benzoates, influence on dextrose-peptone water cultures of *Bacillus coli*, in diminishing gas production, Path. 98
Benzo-naphthol in acute lupus erythematosus, Derm. 51
Berdoe, E.—Discussion on action of aconite on pulse-rate, Therap. 190
Berger, treatment of gunshot injuries to ear, quoted, Otol. 102
Berkeley, Comyns, analysis of 216 cases of Wertheim's operation for carcinoma of the cervix, performed by twenty-two British operators, Obst. 94-99
Berlin, hospitals and clinics of, courses of lectures on medical hydrology at, Baln. 114, 115
——, International Congress of Laryngology at, notice respecting exhibitions of instruments, Laryng. 87
——, opening of University Hydrotherapeutic Institution at, Baln. 86
——, University of, courses of lectures on medical hydrology given at, Baln. 114
Bernard, C., injection of narceine before chloroform inhalation, quoted, Anæsth. 44
——, morphine injection before chloroform inhalation, quoted, Anæsth. 43
Berne, University of, lectures on hydrotherapy at, Baln. 116
Bernheim, suggestion combined with chloroform anæsthesia, quoted, Anæsth. 22
Berry, Mrs. Dickinson.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 10
——, discussion on use of drugs before inhalation anæsthesia, Anæsth. 52

Berry, James.—Discussion on the treatment of cleft palate, Surg. 182
——, large congenital diverticulum of the bladder treated by resection, Surg. 158-165
Berry's needle in operation for cleft palate, Surg. 180
Beta-eucaine, Therap. 56
de Beurmann and Gougerot on sporotrichosis, quoted, Derm. 114
Beverages, calcium salts in, Baln. 123
Beveridge, Major W. W. O.—Discussion on paratyphoid fever and food-poisoning, Epid. 67
Biarritz, climate bracing, Baln. 42
Biberon, as feeding bottle, Child. 133, 134
Bibliographies:—
    Amaurotic idiocy (Tay-Sachs disease), Path. 172
    Arteries, reactions of, to certain drugs, Therap. 94
    Asphyxia under chloroform, Path. 222
    Auriculo-ventricular bundle, Therap. 110
    Belladonna, use of, in dyspepsia, Med. 30
    Bile-ducts, congenital obliteration of, Child. 49
    Calcareous (hard) drinking water, influence of, Baln. 126
    Cartilaginous exostoses (multiple), Med. 18, 19
    Chloroma (atypical), Path. 69
    Complement fixation, Path. 277, 278
    Cow, diseases of (excepting tuberculosis), affecting milk, Epid. 98
    Dermoid tumour of ovary, Obstet. 252
    Diet and acute ulcer of stomach, Path. 59
    Gall-stones and carcinoma in insane, Path. 96
    Gangrene following diphtheria, Child. 14
    Gas-forming power of intestinal bacteria, Path. 108
    Hysterectomy (subtotal) for fibromyoma uteri, Obst. 142
    Infant-feeding, history of, Child. 140
    Osteo-arthritis and chronic rheumatism, Baln. 35
    Ovarian cyst (suppurating), Obst. 262, 263
    Ovarian teratoma, Obst. 288
    Patella, transverse fractures of, Surg. 82, 83
    Poliomyelitis, epidemiology of, Epid. 221
    Portals of entry in phthisis, Child. 219
    Posterior spinal nerve-roots, division of, Surg. 225, 245, 246
    Progressive spinal muscular atrophy of children, Neur. 83, 84
    Pulmonary stenosis (congenital), Clin. 94
    Septicæmia hæmorrhagica, Surg. 57
    Syphilis (inherited) and "blue sclerotics," Child. 98
    Teeth and diseases of skin, relation between, Odont. 83
    Thyroid preparations (pro- and anti-), action of, Path. 236
    Trypanosomiasis (human), Path. 39, 40
    Tuberculosis (pulmonary) from standpoint of disposition, Epid. 175-177
Bickel, acidity of native gastric juice, quoted, Med. 26
Bie, W., and Maar, W., dry residue of blood and blood serum in case of cyanosis and polycythæmia, Clin. 91
Biernacki, J.—Discussion on Dr. Milne's paper, "Measles: its Treatment and Prevention," Child. 178
Bier's method, Laryng. 127
—— —— in treatment of case of acute inflammation of the mastoid (G. C. Cathcart), Otol. 58
—— —— —— of aural suppuration, objections to, Otol. 59
Biggs, experimental work on auriculo-ventricular bundle, Therap. 98
Biggs, G. N.—Case of leprosy with involvement of the external ears, palate, pharynx, &c., Otol. 120
——, case of syphilitic disease of the nose treated with "606," Otol. 121

Bile-duct, common, bacterial infection of, group of abdominal symptoms indicating, Electr. 42

—— ——, microscopical appearance at necropsy of case of congenital obliteration of bile-ducts, Child. 46

—— (hepatic), proximal portion, microscopical appearance at necropsy of congenital obliteration of bile-ducts, Child. 46

——, cancer of (primary), gall-stones as factor in causation of, Path. 93, 94

—— —— —— —— ——, question discussed, Path. 94, 95

—— —— ——, incidence at Guy's Hospital, Path. 91

——, catarrh attacking and causing epidemic jaundice, Epid. 192

——, congenital obliteration (or congenital atresia), case of, microscopical examination, Child. 45

—— —— —— —— —— ——, necropsy, Child. 43

—— —— —— —— —— ——, subsequent course, Child. 42

—— —— —— —— —— ——, with hepatic cirrhosis (F. P. Weber and G. Dorner), Child. 41-50

Biliary passages, see *Bile-ducts*

—— salts in treatment of exophthalmic goitre, Clin. 124

Biliousness, seaside, Baln. 126

Birch-Hirschfeld, inhalation theory, in phthisis, quoted, Child. 228

—— —— on use of words "Disposition" and "Anlage," Epid. 150

Birth palsy, congenital elevation of shoulder mistaken for, Electr. 69

Bishop, E. Stanmore.—The rectum in gynæcology, Obst. 327-336

Bismuth, carbonate of, sterilized, dusting over wound surface with, after amputation of breast, Electr. 57, 62

—— ——, or oxychloride of, dusting over ulcerating surfaces with, intensifies actions of X-rays, and shortens exposure, Electr. 57

——, skiagrams showing position of, after bismuth meal in case of idiopathic hypertrophic dilatation of the colon, Child. 20-22

—— lozenges, sucking of, as means of neutralizing gastric secretion during rectal feeding, Therap. 72

—— meals in stomach cases for radioscopy, alleged dangers, Electr. 60

—— ——, skiagraphy of intestines after, Electr. 91

—— ——, X-ray examination of œsophagus by means of, Electr. 14-23, 27

——, and X-rays, gastric and intestinal spasm visible under observation by, Med. 29-32

Black, micro-organisms in mouth, quoted, Odont. 94

Black lotion, syringing with, and dusting in of calomel in treatment of secondary syphilis of external ear, Otol. 10

Blacker, G. F.—Dermoid tumour of the right ovary, removed by abdominal ovariotomy during labour, Obst. 250

——, discussion on hydatid of the uterus, Obst. 247

—— —— on carcinoma of body of uterus, Obst. 312

—— —— on an ovarian tumour obstructing delivery, Obst. 259

—— —— on operation for cancer of uterus, Obst. 104

——, fibromyomatous polypus of uterus in a girl, aged 17, Obst. 63

——, and Galabin, A. L., alternatives for Cæsarean section, quoted, Obst. 166

——, and Lawrence, T. W. P., unilateral hemaphroditism, quoted, Obst. 109

Bladder, calculi in (A. D. Reid), Electr. 74

——, diverticulum of, congenital, treatment by resection (J. Berry), Surg. 158-165

—— ——, treatment by total extirpation, Surg. 163, 164

——, involvement, in cancer of cervix, Obst. 78, 89

——, skiagraphy of, Electr. 90, 91

——, urinary, action of ergot on, in catarrhal inflammation, Therap. 145

——, see also *Ectopia vesicæ*

Bland-Sutton, J., remarks on supposed primary abdominal pregnancy in rabbits, quoted, Obst., 233

Blindness and deafness (temporary), due to intestinal toxæmia, case of (A. L. Whitehead), Otol. 121
Blistering behind ears in treatment of congenital syphilitic deafness, successful, Otol. 9, 19
Blood, calcium-content estimation in congenital heart disease, Clin. 93
——, changes (secondary), in congenital pulmonary stenosis (F. Parkes Weber and G. Dorner), Clin. 85-94
——, circulation of, cutaneous, promotion by means of pediluvia, Baln. 73
—— ——, dynamics of, anticipation of knowledge of (1811), Med. 151
—— ——, poor, low blood-pressure in subjects of, Med. 72
——, examination of, in cases of icteric ulcers of legs in pernicious anæmia, Clin. 111
—— ——, in melanodermia associated with pernicious anæmia, Derm. 27
——, extravasated, in diffuse fibroma of uterus, with central degeneration (R. H. Paramore), Obst. 248
——, fluids entering stomach rendered isotonic with, Baln. 39
—— of typhoid carrier, examination, Epid. 122
——, viscosity of, how estimated, Clin. 88
—— ——, increased, at high altitudes, factor in causation of rise in low blood-pressure, Med. 59
—— and blood-serum, albumin in, in splenomegalic polycythæmia, Clin. 92
—— ——, dry residue in normal persons, Clin. 91
—— —— —— —— in polycythæmia and congenital heart disease, Clin. 91
Blood-corpuscles, red, excess of, in splenomegalic polycythæmia, Clin. 86, 91
—— ——, increased formation in bone-marrow cause of polycythæmia in congenital heart disease, Clin. 90, 91
Blood-count in jaundice with splenomegaly, Clin. 131
—— in cases of splenic anæmia after operation, Clin. 66, 68
Blood-plasma, importance of, Clin. 95
Blood-pressure, arterial methods of reading, auditory (G. Oliver), Clin. 8
—— —— —— ——, visual (G. Oliver), Clin. 8
——, diastolic, Med. 77. 78, 80
—— during taking of sulphur water, Baln. 56
——, effect of high altitudes on, Med, 76, 77
—— (high), Med. 77
—— ——, due to sclerosis of kidneys, Med. 79
—— ——, effect of iodide of potassium on, Med. 77, 78
—— ——, question of permissibility of alcohol to subjects of, Med. 78, 79
——, idiosyncrasy in, Med. 77
——, influence of, in adolescent albuminuria, Med. 124, 144, 145
—— (low) (W. Edgecombe), Med. 51-80
—— ——, accompanied by dilatation of stomach, Med. 79
—— ——, diet in, Med. 74
—— ——, difficult to raise, Med. 74
—— ——, disorders frequently presenting, Med. 72-74
—— ——, effect of alcohol on, Med. 62, 77
—— —— —— of aperients on, Med. 61
—— —— —— of caffeine on, Med. 63, 74
—— —— —— of calcium chloride on, Med. 66, 76
—— —— —— of calcium lactate on, Med. 74
—— —— —— —— on subjects of chilblains, Med. 66
—— —— —— of constipation on, Med. 60
—— —— —— of digitalis on, Med. 67, 74, 75, 78
—— —— —— of drugs on, Anæsth. 66
—— —— —— of ernutin on, Med. 70
—— —— —— of excitement and worry on, Med. 57, 79, 80
—— —— —— of exercise on, Med. 55

Blood-pressure (low), effect of glycogen on, *nil*, Med. 70
—— —— —— of hydrotherapeutic measures on, Med. 71, 72
—— —— —— of Iodal on, Med. 71
—— —— —— of mental work on, Med. 56, 79, 80
—— —— —— of pituitary extract on, Med. 68
—— —— —— of static brush discharge on, Med. 76
—— —— —— of strychnine on, Med. 67, 74
—— —— —— of tea upon, Med. 62, 63, 77, 78
—— —— —— of tobacco-smoking on, Med. 64, 65
—— ——, mental depression accompanying. Med. 79
—— ——, method of taking, Med. 51
—— ——, night and morning readings compared, Med. 52
—— ——, record of, compared with pulse-rate, Med. 53
—— —— recorded by Oliver's compressed air manometer, Med. 51
—— ——, rise in, produced by altitude, Med. 58, 59
—— —— —— —— ——, factors concerned in, Med. 58, 59
—— —— (systolic), selected for record, Med. 51
—— ——, variations in day observations, Med. 54
—— (mean and maximal), comparison between, necessary to render observations practical, Med. 79
—— —— —— —— —— —— —— —— ——, cases illustrating, Med. 78, 79
—— (normal), raising to supernormal unnecessary, Med. 77
——, observation on, made with hæmodynamometer, Med. 76, 77
——, records enumerating maximum figure of sphygmomanometer only partial, Med. 75, 76
Blood-serum, researches on (1811), Med. 153
Blood-vessels of mucous membrane of tympanic cavity, syphilitic endarteritis in, Otol. 18
Blood-volume, total estimation in case of congenital pulmonary stenosis, Clin. 88, 89
"Blue sclerotics" and inherited syphilis, case of (J. D. Rolleston), Child. 96
BLUMFELD, J.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 8
——, discussion on specimens from case of "delayed chloroform poisoning," Child. 83
—— —— on use of drugs before inhalation anæsthesia, Anæsth. 51
——, nasal reflex during anæsthesia, Anæsth. 27
Boardman-Reed, group of abdominal symptoms, quoted, Electr. 42
Body, association of surface areas of, with deeply seated organs, Baln. 92, 93
——, parts of, suppression or coalescence, foundations of Darwinian variation, Path. 306
Body-cells, accessibilty of germ-plasm to, Path. 307
Boeck's miliary sarcoid (non-ulcerating tuberculide), following lupus vulgaris ? case of (J. H. Sequeira), Derm. 67
Boehm and Wartmann, experiments on use of aconite, Therap. 157
Bokenham, T. J., high frequency current in treatment of atonic dyspepsia, quoted, Electr. 41
Boldt, H. J., practice as regards removal of appendix in pelvic operations, Obst. 351
Bologna, lectures on physical therapy given at, Baln. 116
BOLTON, C.—The influence of diet upon the formation and healing of acute ulcer of the stomach, Path. 53-59
Bolton, cause of swine fever, quoted, Epid. 59
Bond, transplantation of ovaries, quoted. Obst. 116
BOND, W. A.—Discussion on diseases of cow affecting milk in relation to human disease, Epid. 102
Bone, formation of, in periosteum separated by injury (Sir A. A. Bowlby, C.M.G.), Surg. 142
——, structure of, adapted to functional needs, Surg. 2, 4
Bone-abscesses, presence of *Bacillus typhosus* in, Epid. 2
Bone-marrow, erythroblastic reaction in, associated with polycythæmia of heart disease, Clin. 93
——, increased formation of red blood-cells in causation of polycythæmia in congenital heart disease, Clin. 90, 91

Bone-overgrowth, excess of, in Charcot's disease, cause of, Baln. 32

Bones and joints, tuberculous disease of, marine climate indicated for, Baln. 17

—— (long), infiltrates in neighbourhood of, in case of chloroma, Path. 67

—— (pelvic), pressure on head of fœtus alleged cause of facial asymmetry, Odont. 107

——, see also *Mollities ossium ; Osteogenesis*, imperfect

Bonn, University of, courses of lectures on medical hydrology given at, Baln. 114

Bonney, V.—Six cases of "utriculoplasty" for uterine hæmorrhage, one of which was followed by pregnancy and labour, Obst. 270-278

—— and Foulerton, A. G. R., micro-organisms causing puerperal infection, quoted, Obst. 167

—— and Glendining, Bryden.—Adenomatosis vaginæ, a hitherto undescribed condition, Obst. 18

Bony changes in chronic villous arthritis, Baln. 34

—— —— in osteo-arthritis, compensatory, Baln. 29

"Booke of Curtasye," Child. 110

Borchardt, osteo-arthritis due to osteo-myelitis, quoted, Baln. 24

Bordet and Gengou, differentiation of *Bacillus typhosus* from *Bacillus coli* through complement fixation by, Path. 251

Borkum, island of, Baln. 19

—— ——, rainfall compared with that of Margate, Baln. 19

Bostock, Dr., "The Serum of the Blood," quoted, Med. 153

Boston, Institute for Hydrotherapy at, Baln. 90, 197

Boudet and Armand-Delille, case of progressive spinal muscular atrophy of infants and young children, Neur. 69

Bouma's red indigo method, colorimetric adaptation in estimation of indican, Baln. 47

Bournemouth, climate compared with that of Swinemünde, Baln. 19

——, climate suitable for neurasthenics, Baln. 44

——, health resort suitable for foreign invalids, Baln. 20

Bovaird, on bovine tuberculosis infection, quoted, Child. 231

Bowels, catarrh attacking, Epid. 192

Bowen, W. H.—Case of congenital nævoid condition of the left thigh, Child. 193

——, case of glandular fever, Child. 195

Bowlby, Sir A. A., C.M.G.—The formation of bone in periosteum separated by injury, Surg. 142

Box, C. R.—Discussion on case of cerebral athetotic diplegia, Child. 79

——, discussion on the oculo-motor type of polio-encephalitis, Child. 93

Boxing ear, danger of, Otol. 84

—— ——, effects of, Otol. 104, 105

—— ——, rupturing tympanic membrane, Otol. 94, 95

Boycott, A. E., epizootics associated with *Bacillus enteritidis*, noted by, Epid. 61

——, uterine tumours in rabbits, quoted, Path. 225-232

Boyd, absorption of fats in rectal feeding, Therap. 66

Boyd, Sidney.—Congenital absence of chondro-sternal portion of right pectoralis major, and of the greater part of pectoralis minor, Clin. 84

——, discussion on case of actinomycosis of abdominal wall, Clin. 81

——, large cavernous nævus of foot, Clin. 83

Boyd and Robertson, percentage of absorption carbohydrates in rectal feeding, Therap. 65

Boyle, H. E. G.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 8

Boyton, I. J. H.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 48 50

Brachial plexus, injury to (D. Drew), Child. 8

Brachialis anticus, ossification in, Surg. 136

—— ——, ossification of, frequency, Surg. 138

Bradburne, abdominal belt, described by, Electr. 40

Bradley, Burton.—Differentiation of bacilli producing food-poisoning, Epid. 70

BRADLEY, BURTON.—The biological characteristics of *Bacillus typhosus*, with special reference to the fermentation of dulcitol and arabinose, Path. 43
——, mutation of micro-organisms, Epid. 124
Braemar, health resort suitable for foreign invalids, Baln. 21
Braid's method of hypnosis, Anæsth. 22
Brain, cells of, changes in amaurotic idiocy (Tay-Sachs disease), Path. 177
——, cortex of, affections of cells in case of amaurotic idiocy (Tay-Sachs disease), Path. 200, 201, 202
—— ——, sections from case of amaurotic idiocy (Tay-Sachs disease), photomicrographs, Path. 182-185, 186
—— defect (congenital) of, with curious stamping gait and tendency to fall backward, suggestive of a functional astasia (F. E. Batten), Neur. 33
——, frontal convolution, stained section of sub-cortical white matter from case of amaurotic idiocy (Tay-Sachs disease), Path. 184, 186
—— in amaurotic idiocy (Tay-Sachs disease), chemical examination of, in two cases, comparison with normal brain, Path. 19
—— —— —— —— ——, pathological anatomy (G. M. Holmes), Path. 199-204
——, influenza attacking, Epid. 192, 223
—— of patient who died from cerebellar abscess (W. H. Kelson). Otol. 40
——, gumma of, case (C. A. Ballance), Clin. 47
Brain-decay and deposition of cholesterol in tissues as reason for greater prevalence of gall-stones among the insane, Path. 91
Brain-tissue, chemical constituents of, Path. 195
——, phosphorus in, Path. 196
——, sulphur in, Path. 196
BRAINE, C. CARTER.—Discussion on death due to inspiration of gummatous material from gumma which burst during chloroform anæsthesia, Anæsth. 26
——, discussion on nasal reflex during anæsthesia, Anæsth. 29
Bramwell, Milne, demonstration of painless extraction of teeth under hypnotism, quoted, Anæsth. 14
Bramwell and Currie, Harvieston epidemic of poliomyelitis, reported by, Epid. 217
Brand, E., baths in treatment of typhoid fever, Baln. 93
——, "Hydrotherapy of typhus" (1861), Baln. 77
Brandt, Cæsarean section and its alternatives, quoted, Obst. 164
Braun, on operation for division of posterior spinal nerve-roots, Surg. 221
Braun, "Curative effects of baths and waters," translated by Sir Hermann Weber (1874), Baln. 65
Braxton Hicks's cephalotribe, Obst. 190
Bread, detergent action on teeth, experiments showing, Odont. 92, 93
——, effect of acid fermentation of, on teeth, Odont. 93, 94
——, experiments and observations on (J. Sim Wallace), Odont. 90-104
—— —— with special reference to causation and prevention of dental caries (J. Sim Wallace), Odont. 90-104
—— in relation to dental caries, Odont. 97
——, made from stone-milled flour, acid produced from, under mastication, Odont. 90, 102, 103
——, white, made from steel-milled flour, acid produced from, under mastication, Odont. 90, 102, 103
Bread and butter, not harmful to teeth, Odont. 97
Bread-crumb, acid produced by, after mastication and incubation, Odont. 92, 93
Bread-crust, acid produced by, after mastication and incubation greater than that produced in bread-crumb, Odont. 92, 93
—— incorporates greater number of bacteria when in mouth than bread-crumb, Odont. 93
—— preferable to bread-crumb for preservation of teeth, Odont. 97
Bread-diet should be supplemented by vegetables, Odont. 98

Bread and jam productive of dental caries, Odont. 98, 99
Bread and marmalade, acid produced by, after mastication and fermentation, Odont. 96
—— —— productive of dental caries, Odont. 96, 98, 99
Breast, amputation of, for cancer, dusting over wound-surface with sterilized carbonate of bismuth after operation, Electr. 57
——, cancer of, treatment by X-rays, encouraging, Electr. 60
Breast-feeding, decline of, Child. 139
—— in Elizabethan period, Child. 112
——, method of choice at beginning of nineteenth century, Child. 127
——, superiority of, when available, Child. 141
Bree, R., "painful affections of the side, from tumid spleen," quoted, Med. 149
Brentry Certified Inebriate Reformatory, typhoid outbreak due to "carrier" at, Epid. 13, 14, 15
Breslau, University of, courses of lectures on medical hydrology given at, Baln. 114
Breton, gangrene of extremities following diphtheria, quoted, Child. 13
Brewer and Freud, views on hysteria, quoted, Neur. 10
"Bridge" operation (complete) for suppuration of labyrinth causing facial paralysis; recovery (W. Milligan), Otol. 63
Briggs, H.—Discussion on indications for, and technique of Cæsarean section and its alternatives, Obst. 184
Brighton, outbreak of milk-borne sore throat at, Epid. 91, 92
Brine baths, British, Baln. 15
Briquet, definition of hysteria, Neur. 2
Bristol, epidemic of poliomyelitis at (1909), Epid. 212
British Balneological and Climatological Society, Baln. 65, 66
—— —— —— ——, foundation of, by Dr. Samuel Hyde (1895), Baln. 65, 66
—— —— —— ——, work of, summarized, Baln. 4, 5
British Isles, localities for climatic treatment of neurasthenia in, Baln. 42, 44
—— ——, milk-borne sore throat outbreaks in, Epid. 89-97
—— ——, teaching of medical hydrology in, absence of, Baln. 113, 117, 118
British Lying-in Hospital, rule as to suckling children established at, Child. 130
British Medical Association, founding of Section of Electro-Therapeutics, significance of, Electr. 50
—— —— ——, work done by, with regard to registration of disease, Epid. 187
—— —— ——, Lancashire and Cheshire Branch, work done by, with regard to registration of disease, Epid. 187
British operators, analysis of cases of Wertheim's operation for carcimona of cervix performed by, Obst. 94-99
Broadbent, Sir John F. H., Bt.—Discussion on absence of abdominal respiratory movement in pericarditis, Med. 49
——, discussion on use of belladonna in dyspepsia, Med. 32
Broad ligaments, lymphatic tracts from uterus to pelvic glands, contained within, Obst. 76
Brodie, Sir B., experiments as to action of aconite, quoted, Therap. 152
Bronchial and mesenteric glands, susceptibility to tuberculous invasion compared, Child. 230
Bronchiectasis, symptoms of, caused by portion of rabbit-bone impacted in right bronchus for over three years, removal of foreign body (H. Tilley), Laryng. 96
Bronchitis complicated by pneumonia, Med. 107
—— complicating pneumonia, sign of pulmonary failure, Med. 107
—— (pneumococcal—acute suffocative catarrh), cases, fatal (S. West), Med. 101, 106, 107
—— —— —— —— —— —— ——, post-mortem examination, Med. 104
—— —— —— —— —— —— ——, treatment, Med. 102
—— —— —— —— —— ——, non-fatal (S. West), Med. 105
——, presence of bacteria in, Child. 206
——, prevalence of, in relation to weather, Epid. 189
——, pulmonary tuberculosis supervening on, in middle and old age, Epid. 35

Bronchitis, treatment of, Therap. 41, 46
—— —— by auto-inoculation, Therap. 44-47
—— —— by depressant expectorants, Therap. 41
—— —— by irrigation, Therap. 43, 46, 47
Broncho-pneumonia complicating radical operation for frontal sinus suppuration, Laryng. 146
Bronchoscopy and œsophagoscopy, demonstrations, Laryng. 151-153
Bronchus (right), portion of rabbit-bone removed from, wherein it had been impacted for over three years, causing symptoms of bronchiectasis (H. Tilley), Laryng. 96
Bronner, A.—Discussion on case of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 103
——, discussion on case of bilateral tuberculous disease of tonsils, Laryng. 68
—— —— —— of papilloma of larynx in child, Laryng. 81
—— —— —— of temporary deafness and blindness due to intestinal toxæmia, Otol, 122
—— —— on eight cases of mastoid operation (Case II), Otol. 127
—— —— on empyema of maxillary antrum in boy, Laryng. 31
—— —— on equine larynx and "ventricle-stripping," Laryng. 91
—— ——, on the treatment of frontal sinus discharge, Laryng. 146
——, recurrent pedunculated fibroma of posterior pillar of fauces 2 in. long, Laryng. 33
——, scarlet fever causing malignant forms of inflammation in sinuses, Laryng. 32
Brophy's method of operating on cleft palate, Surg. 173, 175, 177, 180, 185, 197
Broth as article of diet for young children in eighteenth century, Child. 122
Brown, Arbuckle, food-poisoning following eating of canned meat at Partick, Epid. 66
Brown, D.—Some pharmacological effects of the strong sulphur water (Harrogate). Baln. 45-62
Brown, W. Langdon.—An inquiry into the value of rectal feeding, Therap. 63-76
Browne, Sir T.—"Garden of Cyrus, or the Quincuncial Lozenge," quoted, Path. 142
Bruce, J. Mitchell.—Action of calcium salts, quoted, Baln. 122
——, discussion on absence of abdominal respiratory movement in pericarditis, Med. 49
—— —— on after-history of albuminuria in adolescence, Med. 142
—— —— on auscultation of joints, Med. 39
—— —— on low blood-pressure, Med. 75, 79
—— —— on use of belladonna in dyspepsia, Med. 33
——, Presidential Address [Condition of medicine and surgery one hundred years ago, 1811], Med. 147-156
Bruce, W. Ironside.—Discussion on Bauer's qualimeter, Electr. 72
——, discussion on demonstration of new tube-stand, Electr. 31
—— —— on lantern slides of three cases, Electr. 75
—— —— on Röntgen ray examination of œsophagus, Electr. 27
—— —— on unusual forms of contraction of fingers, Electr. 73
Bruce's nodes, occurrence of, Baln. 25
Brückner, *Bacillus typhosus* in fæces, quoted, Epid. 5
——, paratyphoid carriers, Epid. 56
Brugsch and Schittenhelm, administration of hypoxanthin in gouty patients, quoted, Baln. 59
Brünings's light head bath in treatment of acute frontal sinusitis, Laryng. 127, 128, 143
Bruns, cases of, not regarded as belonging to Werdnig-Hoffmann type of progressive spinal muscular atrophy, Neur. 71
Bruns and Sauerbruch, case of division of posterior spinal nerve-roots for gastric crises of tabes, Surg. 211
Brush discharge (static), effect on low blood-pressure, Med. 76
Brushes fitted to new induction coil, Electr. 80, 81, 83
Brussels, University of, lectures on medical hydrology at, Baln, 116
Buchan, disinfection after measles, quoted, Child. 175
Buchan, G. F.—Discussion on typhoid carriers and contact infection, Epid. 140
Buchanan, G. S.—Discussion on diseases of cow affecting milk in relationship to human disease. Epid. 99

BUCHANAN, G. S.—Discussion on paratyphoid fever and food-poisoning, Epid. 63
——, discussion on the problem of disposition in pulmonary tuberculosis, Epid. 183
Buchner's tube, anaerobic growth of variant *Bacillus coli* in, Path. 107
BUCKLEY, C. W.—Ætiology of gout, quoted, Baln. 120
——, discussion on balneological treatment of neurasthenia, Baln. 43
——, osteo-arthritis and its relation to chronic rheumatism, Baln. 23-36
Budapest, University of, lectures on hydrotherapy and climatology at, Baln. 115
Budd, W., on tuberculous contagion, quoted, Epid. 164
Building Fund, appeal for donations and subscriptions to, Med. 155
Buist, R. C., pathology of ureters in cancer of cervix, quoted, Obst. 78
BULLOCH, W.—Analysis of publications on portals of entry of tuberculosis, quoted, Child. 206
——, and GREENWOOD, MAJOR.—The problem of pulmonary tuberculosis considered from the standpoint of disposition, Epid. 147-177, 184
Bullous dermatoses, production by ptomaine poisoning, Med. 32
—— —— relieved by small doses of belladonna, Med. 32
Bumm, E., Cæsarean section and its alternatives, quoted, Obst. 164
——, indications for craniotomy, given by, Obst. 214
——, practice as regards removal of appendix in pelvic operations, Obst. 352
——, primary mortality from Wertheim's operation, Obst. 95
BUNCH, J. L.—Case of lichen spinulosus, Child. 86
——, case of morphœa in band form in a girl, aged 7, Child. 27
——, congenital syphilis treated by "606," Child. 158
Bundle of His, Therap. 87, 113
—— ——, see also *Auriculo-ventricular bundle*
Buphthalmos and hydrocephalus, case of (J. C. W. Graham), Child. 197
BURGESS, W. MILNER.—Discussion on case of rheumatoid arthritis, Child. 37
——, discussion on case of splenic enlargement, Child. 29
—— —— on specimen from case of necrosis of lower jaw, Child. 53
BURGHARD, F. F.—Discussion on the treatment of cleft palate, Surg. 188
—— and SUTHERLAND, G. A.—Treatment of splenic anæmia by splenectomy, Clin. 58
Burness, A. G., and Mavor, F. F., experiments as to action of aconite, Therap. 157
Burns of middle and internal ear, Otol. 95, 96
Burr, new form of, for use in radical antral operation by intranasal route (W. H. Kelson), Laryng. 106
—— —— —— —— in antral operation by nasal route (S. Hastings), Laryng. 105
BURT, J. B.—Discussion on osteo-arthritis and chronic rheumatism, Baln. 35
BUTCHER, W. DEANE.—Bauer's qualimeter; an instrument for determining the hardness of an X-ray tube by measuring the potential through the tube, Electr. 71
——, discussion on diathermic treatment, Electr. 9
——, osmotic growths, Electr. 93-96
BUTLER, W.—Discussion on diseases of cow affecting milk in relationship to human disease, Epid. 100
——, discussion on tuberculosis and its prevention, Epid. 47
Butterflies, climatic variation in, Path. 307
BUXTON, DUDLEY W.—The use of scopolamine, morphine, atropine, and similar drugs by hypodermic injection before inhalation anæsthesia, Anæsth. 43-56
Buxton, elevation in relation to treatment of circulatory disorders at, Baln. 69
——, mineral waters at, Baln. 15
——, neurasthenia benefited at, Baln. 42
BUZZARD, E. F.—Case of spondylose rhizomélique, Neur. 42
——, case of tabes with lumbo-sacral disease, Clin. 13
——, discussion on case of syringomyelia, with symptoms precipitated by trauma, Neur. 39
—— —— on spondylitis, with progressive muscular contraction, Neur. 44
Buzzard, T., "Cases illustrating the infective origin of infantile paralysis," quoted, Epid. 210

Cæcum and appendix, adhesions between wall of, in appendicitis in women, Obst. 347
——, tuberculosis of, hyperplastic, Path. 240
—— and ascending colon, resection of, case of, showing chronic hypertrophy of mucous membrane, with stenosis simulating cancer (C. Gordon Watson), Path. 237
Cæsarean section (extra-peritoneal), Obst. 162, 190, 203, 205, 206, 215
—— —— ——, advocacy of, Obst. 165, 166
—— —— ——, disadvantages in septic cases, Obst. 163, 164, 173
—— —— ——, disapproval of, Obst. 164
—— —— in contracted pelvis and protracted labour complicated by fibroids, Obst. 204
—— —— —— —— —— ——, contra-indicated by presence of gonorrhœa, Obst. 169
—— —— —— —— —— —— —— in gravely infected cases, Obst. 190
—— —— —— —— —— —— in septic cases followed by peritonitis, Obst. 186, 191, 209
—— —— —— —— —— —— increasing safety, dispenses with craniotomy, Obst. 216
—— —— —— —— —— ——, indications for, Obst. 172, 192
—— —— —— —— —— ——, injection of polyvalent antistreptococcic serum in, Obst. 191
—— —— —— —— —— ——, inoculation of lactic bacillus in, Obst. 203, 211
—— —— —— —— —— —— in suspected septic cases, Obst. 162, 185, 188, 189, 192, 204, 205, 213
—— —— —— —— —— —— limitation to aseptic cases, advocated, Obst. 184
—— —— —— —— —— ——, mortality in aseptic cases, Obst. 162
—— —— —— —— —— —— —— in suspected septic cases, Obst. 162
—— —— —— —— —— —— preliminary use of utero-parietal suture, Obst. 202, 203
—— —— —— —— —— ——, risks of, Obst. 196, 209
—— —— —— —— —— ——, technique, Obst. 188, 205, 209, 211
—— —— in eclampsia gravidarum, communications on summarized, Obst. 8
—— —— in placenta prævia, communications on (1909-10), summarized, Obst. 7
—— —— in tumours obstructing labour, Obst. 252, 255
—— ——, irrigation of amniotic cavity as prophylactic measure before, in doubtfully septic cases, Obst. 173, 185, 186, 198, 199, 213
—— —— (Porro's), for septic cases of contracted pelvis and protracted labour, Obst. 162, 208
—— —— —— in advanced cancer complicating labour, Obst. 191
—— ——, rupture of gravid uterus through scar of (S. J. Cameron), Obst. 147
—— —— (supra-symphyseal), Obst. 188
—— —— (transperitoneal), Obst. 163
—— —— (transverse), diffuse cellulitis following, Obst. 188
—— —— and total abdominal hysterectomy in cancer, complicating labour, Obst. 191
Caffeine, effect of, on low blood-pressure, Med. 63, 74
——, reaction of arteries to, Therap. 90
Cagliari, lectures on hydrology at, Baln. 116
Cairo, stations round, suitable for climatic treatment of neurasthenia, Baln. 42
Caisson workers, spontaneous hæmorrhage into tympanic cavity in, Otol. 84
Calamine lotion in lupus erythematosus, Derm. 92
Calcareous substances contained in Vittel waters, Baln. 124
Calcium chloride, effect on low blood-pressure, Med. 66, 76
—— ——, reaction of arteries to, Therap. 90
—— content of blood, estimation in congenital heart disease, Clin. 93
—— lactate, effect on low blood-pressure, Med. 74
—— ——, effect on low blood-pressure in subject of chilblains, Med. 66
—— oxalate, intercalated zones of, in uric acid calculus of cancellous variety, Path. 115
—— ——, see also *Calculi* (urinary, calcium oxalate)
—— salts, addition to distilled water, Baln. 122
—— ——, effects of, discussed, Baln. 125
—— —— —— on skin, Baln. 122
—— —— in drinking water, Baln. 128
—— —— in foods and beverages, Baln. 123

Calcium salts, therapeutic applications, Baln. 121
—— ——, withdrawal from body alleged cause of oxalic acid poisoning, Baln. 124
—— sulphate, therapeutic value of, Therap. 22
Calculi in bladder (A. D. Reid), Electr. 74
——, oxalate of lime, prevention of formation, Baln. 124
—— (urinary), crystalline rosettes and spheres of, Path. 142
—— ——, production and growth as influenced by movement of urine, Path. 141
—— (urinary, calcium oxalate), microscopical structure of, Path. 111
—— (urinary, uric acid) (cancellous), "crystalline" rubble in, Path. 118, 121, 130, 137
—— —— —— —— ——, difference in structure from that of compact variety explained, Path. 139, 140
—— —— —— —— ——, fracture of, Path. 136
—— —— —— —— ——, microscopical structure, Path. 113, 130
—— —— —— —— ——, nucleus of, Path. 113, 114, 115, 116, 118, 120, 121, 130
—— —— —— —— —— ——, nature, Path. 136-138
—— —— —— —— ——, with intercalated zones of calcium oxalate, microscopical structure, Path. 115
—— —— —— —— ——, with urate of ammonium and trace of urate of sodium, microscopical structure, Path. 118
—— —— —— —— (compact), Path. 123
—— —— —— —— ——, cross-cleavage in, Path. 123, 132, 133
—— —— —— —— ——, cross-striation of masses in, how produced, Path. 141
—— —— —— —— ——, crystals elongated, Path. 138
—— —— —— —— ——, difference in structure from that of cancellous variety explained, Path. 139, 140
—— —— —— —— ——, microscopical structure, Path. 123, 132
—— —— —— —— ——, miliary, Path. 130
—— —— —— —— ——, nucleus of, Path. 123
—— —— —— —— —— ——, nature, Path. 136-138
—— —— —— —— —— ——, sometimes absent, Path. 123
—— —— —— —— —— ——, stellate, Path. 129
—— —— —— ——, crystals forming compared with uric acid sediments, Path. 133-136
—— —— —— ——, formation of, secretion of urine supersaturated in uric acid necessary before crystallization can take place in, Path. 138
—— —— —— ——, microscopic structure (S. G. Shattock), Path. 110-146
—— —— —— —— —— ——, technique of examination, Path. 111, 112
—— —— —— ——, nucleus of, microscopical structure, Path. 113
Calculus, production of, through hard water, questioned, Baln. 124
—— (ureteral) discovered by examination *per rectum*, Obst. 329
—— (vesical), alleged prevalence in Norfolk, Baln. 125
Calf, experimental infection with Gaertner bacillus, result fatal, Epid. 69
Calliper splint, use after fractures about hip-joint, Surg. 14
Callosities in camel, Path. 280
—— ——, capacity to acquire alone transmitted, Path. 309
—— in giraffe, Path. 283
—— ——, capacity to acquire alone transmitted, Path. 309
—— in monkey, how differing from those in camel, Path. 309
——, Lamarckism in relation to (S. G. Shattock), Path. 279-312
——, see also *Ischial callosities*
Callus, assuming form of cancellous osteoma, Surg. 138
—— formation in fractures, Surg. 7, 8, 9
Calmette, injection experiments with tubercle bacilli, Child. 230
—— portals of entry of tuberculosis, quoted, Child, 205, 206
Calomel, administration of, increases excretion of bodies of indoxyl group, Baln. 56
——, dose of, following slight chloroform anæsthesia in successful treatment of epilepsy complicated by acute mania, Baln. 7

Calomel, dusting in with, after syringing with black lotion, in treatment of secondary syphilis of external ear, Otol. 10

Caloric tests applied to exposed inferior crus of posterior semicircular canal, in case where disease in large sinus tympani caused persistence of discharge after radical operation (A. H. Cheatle and G. J. Jenkins), Otol. 54

Calx sulphurata, precautions in use of, Therap. 18, 19

Camel, callosities in, Path. 280

—— ——, capacity to acquire alone transmitted, Path. 309

Cameron, H. C.—Discussion on therapeutic use of digitalis group, Therap. 36

Cameron, Murdoch, unjustifiability of craniotomy, quoted, Obst. 165

Cameron, S. J.—Discussion on indications for and technique of Cæsarean section and its alternatives, Obst. 210

——, gravid uterus ruptured through the scar of a Cæsarean section, Obst. 147

——, osteomalacic pelvis, Obst. 151

——, twin tubal pregnancy, Obst. 149

Campbell, Harry.—Discussion on case of congenital cerebellar ataxia, Neur. 20

Campbell, J., practice as regards removal of appendix in pelvic operations, Obst. 35

Campkin, P.—Discussion on comparative elasticity and internal structure of cast and rolled gold plate, Odont. 66

Canadians, limited knowledge of British health resorts possessed by, Baln. 22

Cancer of bile-ducts, incidence at Guy's hospital, Path. 91

—— —— (primary), gall-stones as factor in causation of, Path. 93, 94

—— of body of uterus in patient, aged 29 (H. R. Spencer), Obst. 308-312

—— of breast, dusting over wound-surface with sterilized carbonate of bismuth after operation, Electr. 57

—— ——, treatment by X-rays encouraging, Electr. 60

—— of cervix uteri, treatment, Obst. 101, 103

—— complicating labour, procedure in, Obst. 190, 191

—— —— ——, advanced cases, procedure in, Obst. 191

—— of deep pharynx with extension to gullet and posterior margin of larynx (W. Hill), Laryng. 44

——, diagnosis, completeness and accuracy in, impossible, under early and thorough treatment, Electr. 48, 50

——, dust, dirt, and uncleanly habits factors in causation of, Electr. 51

—— of gall-bladder (primary), gall-stones as factor in causation of, Path. 93, 94

—— —— —— —— gall-stones possibly formed concomitantly with and dependent upon malignant disease, Path. 95

—— —— ——, incidence at Guy's Hospital, Path. 91

—— —— ——, incidence in insane, Path. 91, 92

——, growth of, rapidity cannot be determined by histological examination, Obst. 70

—— ——, rate varies according to different kinds, Obst. 70

——, immunity to, experiments to investigate, Path. 80-86

——, inoperable, treatment by radium emanations dissolved in water, Electr. 66

——, irritation one of commonest causes of, Electr. 51

—— of larynx, case of (P. Watson-Williams), Laryng. 93

—— ——, origin of (R. H. Scanes Spicer), Laryng. 19

—— of lung (primary), local prevalence in cobalt mines at Schneeberg, Path. 78

—— —— ——, pneumokoniosis predisposes to, Path. 78

—— —— ——, rarity, Path. 78

—— of œsophagus (W. Hill), Laryng. 113

—— ——, associated with aneurysm, Electr. 23, 30

—— ——, causing obstruction, Electr. 17-20

—— ——, diathermic current indicated in, Electr. 9, 11

—— ——, dysphagia treated by new form of intubation apparatus (W. Hill), Laryng. 9

—— ——, treatment by radium, cases reported, Electr. 64-66

Cancer of œsophagus, treatment by radium, disappearance of fungation and ulceration under (W. Hill), Laryng. 8

—— —— —— —— ? temporary cure (W. Hill), Laryng. 43

—— of ovary, columnar-celled (H. P. Dimmock), Obst. 62

—— (party-wall) of larynx and œsophagus (L. H. Pegler), Laryng. 101

—— of pharynx (party-wall) (C. Graham), Laryng. 55

—— ——, treatment by radium, cases reported, Electr. 64-66

—— of rectum in patient from whom malignant papillomatous cysts of both ovaries had been removed six years previously (M. A. Scharlieb), Obst. 358

—— ——, relation of multiple polypi to, Surg. 105, 106

—— ——, treatment, operative, Surg. 99-132

—— simulated by hyperplastic tuberculosis of colon, Surg. 239, 240

—— —— by stenosis in case of resection of cæcum and ascending colon, showing chronic hypertrophy of mucous membrane (C. Gordon Watson), Path. 237

——, "spontaneous cure of," discussed, Electr. 48, 49

—— (squamous-celled); excision of larynx, lower part of pharynx, and upper end of œsophagus for, subsequent report on case of (A. Evans), Clin. 142

—— of stomach, administration of belladonna valuable in certain cases of, Med. 33

—— ——, tuberculous invasion, Child. 226

—— of thyroid, with extensive involvement of lymphatic glands; operation three and a half years ago; no recurrence (W. Trotter), Clin. 118

—— of tonsil (primary), case of (E. A. Peters), Laryng. 79

——, transmission from one individual to another, question discussed, Path. 78, 79

——, treatment by diathermic current, Electr. 5

—— —— —— ——, avoidance of use of knife except for surface incisions, Electr. 11

—— —— by radium (J. Hall-Edwards) Electr. 47-69

—— —— ——, dosage, Electr. 62

—— —— ——, large quantities essential, Electr. 62-63

—— —— by X-rays (J. Hall-Edwards), Electr. 47-69

—— —— ——, atrophy of growth following, Electr. 60

—— —— ——, cases showing good results, Electr. 64

—— —— ——, difficulty in following up cases after treatment, Electr. 57

—— —— ——, dosage, Electr. 63

—— —— ——, two chief factors in, Electr. 60

—— —— —— or radium to be determined by accessibility of region, Electr. 62

—— ——, non-operative, surgical scepticism as to, Electr. 50

—— ——, operative, Electr. 47, 48

—— of uterus, treatment by total abdominal hysterectomy (Wertheim's method), cases recorded (C. P. Childe), Obst. 79-100

—— —— ——, operative, pathological bases for (A. Leitch), Obst. 69-78

Cancer-cells, lymphatic glands permeated by, do not coincide with channels marked out by artificial injection, Obst. 76

Cancer-spray, transmission of mouse cancer to mice attempted by means of, Path. 77

CANDLER, J. P.—The incidence of gall-stones and of primary carcinoma of the gall-bladder and biliary passages in the insane, Path. 87-96

Canis egyptius, Path. 298

—— *familiaris*, see *Dogs*, hairless

Canton, A., case of hypertrophy of gums and alveolar processes, quoted, Odont. 22

CAPES, R.—Discussion on action of aconite on pulse-rate, Therap. 190

Capillaries, changes in, in human trypanosomiasis, Path. 30, 31

Carbohydrate starvation theory of delayed chloroform poisoning, Child. 84

Carbohydrates, absorption in rectal feeding, Therap. 65

——, assimilation of, causing abolition of acidosis, Therap. 66

——, deprivation of, factor in production of acetonuria, Therap. 68

——, fermentable, non-detergent, lodgment in contact with teeth, undisturbed by saliva, main source of caries, Odont. 97

Carbon dioxide, reaction of arteries to, Therap. 79
Carbon dioxide snow, Electr. 51, 59
——— ——— ——— in acute lupus erythematosus not indicated, Derm. 51
——— ——— ———, treatment by, of rodent ulcer, effect of, Electr. 61
Carbon monoxide method (Haldane and Lorrain Smith's) for estimating total oxygen capacity and total blood volume, Clin. 89
Carbonic acid gas in medicinal waters, Baln. 108, 109
——— ——— ———, subthermal baths containing, sensation produced by, Baln. 109
Carcinoma, see *Cancer*
Carcinomatous degeneration in chronic dermatosis (C. Rowntree), Clin. 159
Cardiac inhibition, reflex, influence of chloroform and asphyxia upon, Path. 206-214
Cardiolysis for adherent pericardium, case of (G. A. Sutherland), Child. 41
Cardiovascular changes, degenerative, association of delirium cordis with, Med. 92, 96
——— system, diseases of, climatic factor in bath treatment of, Baln. 68, 69
——— ——— ———, treatment by subthermal baths, Baln. 68
CARGILL, L. VERNON.—Double proptosis due to a growth, lymphoma (hæmangio-lymphadenoma) invading each orbit, Clin. 53
Caries, dental, ætiological factors, bread and marmalade and bread and jam, Odont. 96, 98, 99
——— ——— ——— ———, main factor formulated, Odont. 97
——— ——— ——— ———, mouth-breathing, Odont. 102
——— ——— ——— ———, sugar, Odont. 96, 100, 102
——— ———, age incidence, greatest, coincides with period of greatest consumption of milk, Odont. 3
——— ———, artificial, production by milk-germs (F. J. Bennett), Odont. 3
——— ——— ——— ——— ———, specimens compared with those of natural caries, Odont. 4
——— ———, bread in relation to, Odont. 97
——— ———, immunity to, Odont. 2
——— ———, natural, liquefaction of dentine in, Odont. 6
——— ———, possible production through agency of lactic acid bacillus, Odont. 8, 9
——— ———, prevention of, in children, Odont. 102
——— ———, production of, fermentability of food-stuffs in relation to, Odont. 91
——— ———, removal of plaques and masses of bacteria on and about, to prevent, Odont. 94, 101
——— ———, site of origin in teeth, Odont. 91, 92
——— ———, special, not general, forms of oral bacteria, factor in production of, Odont. 94
——— ———, susceptibility to, Odont. 2
——— and necrosis of temporal, parietal, and occipital bones following mastoiditis due to scarlet fever in girl, aged 5; operation; recovery (H. J. Davis), Otol. 70
——— (spinal), diagnosis of progressive spinal muscular atrophy of infants and young children from, Neur. 67
——— ———, specimens showing, Child. 202
CARLESS, ALBERT.—Adiposis dolorosa, Clin. 3
———, discussion on cases of familial pigmentary dermo-fibromatosis, Clin. 76
——— ——— on specimens illustrating the removal of foreign bodies from the air-passages and the œsophagus, Clin. 154
Carlier, nephrostomy with drainage of kidneys for complete anuria, quoted, Clin. 162
CARLING, E. ROCK.—Discussion on myositis ossificans traumatica, Surg. 157
———, and GOSSAGE, A. M.—Multiple cartilaginous exostoses, Med. 1-22
Carlisle, epidemic of poliomyelitis at (1910), Epid. 212, 213
CARLYLL, H. B., and MOTT, F. W., F.R.S.—Seven cases of amaurotic idiocy (Tay-Sachs disease): Part I, by Dr. Carlyll; Part II, by Dr. Mott; Histological and chemical examination of the nervous structure, Path. 147-198
Carmichael, Scott, transplantation of ovaries, quoted, Obst. 116
Carpus, hair-mats over, in antelopes, Path. 284
Carr and Barger, ergot researches, quoted, Therap. 114, 115

CARR, J. WALTER.—Case of anorexia nervosa, Child. 80

——, discussion on case of enlargement of liver, Child. 55

—— —— —— of splenic enlargement, Child. 29

—— —— —— of von Jaksch's anæmia, Child. 157

—— —— —— on two cases of infantilism, Child, 60

Carrier state, experimental production of, by feeding (G. F. Petrie and R. A. O'Brien), Path. 70-72

Carriers of diphtheria, see *Diphtheria*, carriers of

—— of human trypanosomiasis, see *Glossina palpalis*

—— of tuberculosis; carriers of tuberculosis (pulmonary), see *Tuberculosis*, carriers of; *Tuberculosis* (*pulmonary*), carriers of

—— of typhoid fever, see *Typhoid fever*, carriers of

Carter, limitation of amaurotic idiocy (Tay-Sachs disease) to Hebrew race, quoted, Path. 167

Carter, H. Vandyke, microscopical structure of urinary calculi, quoted, Path. 111

Cartilage, abnormal constitution in multiple cartilaginous exostoses, Med. 5

——, "islets" of, Med. 3

Caruso's sagittal method of fundal incision into uterus, Obst. 178

Casciani, Professor, lectures on medical hydrology in University of Rome, Baln. 115

Casein, powdered, and milk, absorption in rectal feeding, Therap. 64

CASSIDY, M. A.—Discussion on absence of abdominal respiratory movement in pericarditis, Med. 46

—— and PAGE, C. M.—Method for determining the absolute pressure of the cerebro-spinal fluid, Clin. 56

Castellani's absorption test in differentiation of bacilli, Epid. 52, 67, 136, 137

Castex and Parrel, syphilitic showing rhythmical elevations of soft palate accompanied by rhythmical clickings in Eustachian tube and tympanum on left side, Otol. 13

Cataract, chronic, case of (J. C. W. Graham), Child. 197

Catarrh (acute suffocative) of pneumococcal origin, see *Bronchitis* (pneumococcal)

—— attacking bile-ducts and causing epidemic jaundice, Epid. 192

—— —— stomach and bowels, Epid. 192

—— (gastro-intestinal) in neurasthenia, Baln. 40

——, post-nasal, chronic, bacteriology (C. Ernest West), Otol. 43-50

——, prevalence of, in relation to weather, Epid. 189

——, respiratory, dry form, British seaside climate suitable for, Baln. 17

CATHCART, G. C.—Case of acute inflammation of the mastoid treated by Bier's method, Otol. 58

——, discussion on origin of cancer in larynx, Laryng. 23

—— —— on Dr. Watson-William's osteo-plastic frontal sinus operations, Laryng. 157

——, papilloma of pharynx in male, aged 42, Laryng. 62

——, recurring aphonia in a woman, aged 36, Laryng. 82

——, ulceration of the left vocal cord in a male, aged 67, case for diagnosis, Laryng. 63

Cats, experimental formation of acute gastric ulcer in, by injection of gastro-toxic serum, Path. 53, 54, 55

Cattle, lead-poisoning in, symptoms of, Laryng. 91

CAUSH, D. E.—Discussion on experiments and observations on bread, with special reference to the causation and prevention of dental caries, Odont. 101

Caustics, introduction into ear injuring membrana tympani, Otol. 84

Cautery knife, value of, in operations for cancer of cervix, Obst. 101, 103

CAUTLEY, EDMUND.—Case of cerebral athetotic diplegia, Child. 77

——, case of splenic enlargement, Child. 28

——, cerebral sclerosis and idiocy, Child. 184

——, discussion on a preliminary communication on syphilis and mental deficiency, Child. 170

—— —— on case of achondroplasia, Child. 106

—— —— —— of anorexia nervosa, Child, 80

—— —— —— of congenital aortic stenosis, Child. 86

CAUTLEY, EDMUND.—Discussion on case of congenital dilatation of colon, Child. 77
——, discussion on case of congenital obliteration of bile-ducts, Child. 49
—— —— —— of enlargement of liver, Child. 55
—— —— —— of extensive necrosis of lower jaw, Child. 31
—— —— —— of infantilism with thyroid inadequacy, Child. 96
—— —— —— of injury to brachial plexus, Child. 9
—— —— —— of malformation of the heart, Child. 39
—— —— —— of morphœa in band form in a girl, aged 7, Child. 28
—— —— —— of nævus verrucosus linearis, Child. 33
—— —— —— of sclerema neonatorum, Child. 7
—— —— —— of severe tuberculous peritonitis cured by laparotomy, Child. 187
—— —— —— of simple atrophic type of myopathy, Child, 102
—— —— —— and specimen of congenital malformation of heart, Child. 4
—— —— —— of syphilitic osteitis of femur, Child. 105
—— —— —— of von Jaksch's anæmia, Child. 158
—— —— on chronic (recurrent) tetany, Child. 18
—— —— on the history of infant-feeding from Elizabethan times, Child. 141
—— —— on idiopathic hypertrophic dilatation of colon, Child. 22
—— —— on malformation of femur, Child. 164
—— —— on oculo-motor type of polio-encephalitis, Child. 93
—— —— on portals of entry of phthisis, Child. 220
—— —— on specimens from case of "delayed chloroform poisoning," Child. 84
—— —— on tumour of rectus abdominis in child, Child. 8
—— —— on two cases of infantilism, Child. 61
—— —— on word-blindness and idioglossia, Child. 52
——, pyloric hypertrophy and spasm, Child. 185
Caverly and McPhail, epidemic of poliomyelitis in Rutland (Massachusetts), quoted, Epid. 200
Cavernous sinus, thrombosis of, case of, specimen of temporal bone from (H. J. Davis), Otol. 27
—— —— ——, due to oral sepsis, Odont. 116, 117
—— —— —— —— —— ——, case of (G. Taylor), Odont. 113-118
—— —— —— —— —— ——, rarity, Odont. 113
—— —— —— —— suppuration of nose or ear, Laryng. 162
Cell (living), both producer and distributor of electricity, Electr. 93
——, nuclei, pyknosis of, in case of atypical chloroma, Path. 64
—— (somatic), parental constituents, Med. 6
—— ——, see also *Allelomorphs*
Cellular contents, dentigerous cyst with (W. W. James), Odont. 85
Cellulitis (diffuse) following transverse Cæsarean section, Obst. 188
—— (external or orbital), indication for radical operation in chronic frontal sinus suppuration Laryng. 132
—— (hæmorrhagic), cases of, Surg. 50-56
—— of soft tissues, externally, complicating acute frontal sinusitis, Laryng. 128
—— (orbital), results of, following ethmoidal operation (A. J. M. Wright), Laryng. 159
—— ——, usually subsequent to septic processes in nasal cavities, Laryng. 162
Celsus, method of removing foreign bodies from auditory meatus, quoted, Otol. 96
—— on use of the bath, quoted, Baln. 3
Cephalotribe (Braxton Hicks's), use of, in neglected labour, Obst. 190
Cerebellar fossa abscess, sloughing through jugular foramen into neck; sloughing of middle third of internal jugular vein; operation; recovery (H. J. Davis), Otol. 28
Cerebellum, abscess of, brain from fatal case of (W. H. Kelson), Otol. 40
—— —— (extradural), tracking through jugular foramen into neck (H. J. Davis), Otol. 71
—— ——, following otitis media suppurativa chronica (W. Milligan), Otol. 61
—— ——, cases of; operation; recovery (W. Milligan) Otol. 61
——, atrophy of, in case of amaurotic idiocy (Tay-Sachs disease), Path. 201

Cerebellum, loss of function compensated for by cerebrum, Neur. 20
——, section of, from case of amaurotic idiocy (Tay-Sachs disease (photomicrograph), Path. 186, 187, 188 ; (plate), Path. 191
——, tumour of, causing secondary optic atrophy (?) case of (O. K. Williamson), Child. 109
—— ——, dysdiadokokinesia in, Otol. 36, 37
——, see also *Ataxy* (cerebellar)
——, see also *Retro-cerebellar abscess*
Cerebral athetotic diplegia, case of (E. Cautley), Child. 77
—— sclerosis and idiocy (E. Cautley) Child. 184
Cerebro spinal fluid, absolute pressure of, method for determining, Clin. 56
—— ——, as a secretion, assignment of credit for priority of idea, Med. 153
—— ——, escape after injury to head, Otol. 91
—— ——, examination, in amaurotic idiocy (Tay-Sachs disease) Path. 154, 155, 157, 161, 165, 178
Cerebrum, loss of functions of cerebellum compensated for by, Neur. 20
Cerne Abbas, epidemic of poliomyelitis at, Epid. 216, 226
Cervix uteri, cancer of, involvement of vagina in, Obst. 78
—— ——, treatment by high amputation, Obst. 101, 103
Cetecea (fossil), periotic bones of (W. Jobson Horne), Otol. 114
Chair for screening chest in skiagraphy, Electr. 88
Chalmers, A. K., outbreak of milk-borne sore throat at Belvidere Hospital, Glasgow, Epid. 95
Chalybeate waters, tonic effect on neurasthenia, Baln. 40
Champneys, Sir F. H., Bt.—Discussion on indications for and technique of Caesarean section and its alternatives, Obst. 201
Changing box, adjunct of plate-holder, Electr. 86
Chapman, C. W.—Discussion on case of anorexia nervosa, Child. 80
——, two cases of deformity of the hands, Clin. 159
Characters, acquired by increased exercise, transmission, Path. 280
—— ——, inheritance of, Path. 304
—— ——, transmissibility, indirect, Path. 307
—— —— —— ——, climatic agency, Path. 307
—— ——, transmission, Path. 280
—— —— —— denied, Path. 280
Charcot, use of douche bath by, Baln. 88
Charcot's disease, excess of bony overgrowth in, cause, Baln. 32
—— ——, identity with osteo-arthritis insisted on by Virchow, Baln. 24
Charity Organization Society and home visitation of tuberculosis contacts, Epid. 38
Charles II, reign of, infant mortality during, Child. 117, 118
Charwomen, cause of swollen and distorted knuckle-joints among, Baln. 28
Chaulmoogra oil in leprosy, Laryng. 27
Chavannaz, division of posterior spinal nerve-roots for pain, Surg. 204
Cheatle, A. H.—Case of congenital malformation of the auricle and meatus, Otol. 25
——, case of erysipelas of the head : invasion of the right auditory meatus, where an abscess formed with extension backwards to the sterno-mastoid muscle, and invasion of the whole sheath of that muscle, Otol. 111
—— ——, of exostoses in the middle ear, Otol. 56
—— —— of healed fracture of the right temporal bone in which the line of fracture in the meatus can be seen, Otol. 26
——, discussion on bacteriology of chronic post-nasal catarrh, Otol. 51
—— —— on brain of patient who died from cerebellar abscess, Otol. 41
—— —— on case of acute inflammation of mastoid treated by Bier's method, Otol. 59
—— —— —— of acute vertigo with repeated falls always to left, Otol. 73
—— —— —— of caries and necrosis following mastoiditis due to scarlet fever, Otol. 70
—— —— —— of chronic dermatitis of pinna, Otol. 39
—— —— —— of chronic otitis media suppurativa, Otol. 66

CHEATLE, A. H.—Discussion on case of extradural cerebellar abscess, Otol. 30
——, discussion on case of infiltration of both auricles, Otol. 38
—— —— —— of temporary deafness and blindness due to intestinal toxæmia, Otol. 122
—— —— —— of unilateral deafness associated with vertigo, Otol. 61
—— —— on deafness due to occlusion of Eustachian tubes by scar tissue, Otol. 109
—— —— on eight cases of mastoid operation (Case IV), Otol. 130
—— —— on labyrinthine vertigo (Menière's symptoms, non-infective), treated by operation, Otol. 119
—— —— on large tuberculous polyp removed from middle ear of infant, Otol. 42
—— —— on new growth of unknown nature in tympanum, Otol. 78
—— —— on specimen of temporal bone from case of thrombosis of cavernous sinus, Otol. 27
—— —— on two cases of cerebellar abscess, Otol. 63
——, fifteen specimens of temporal bone, showing types of fractures, Otol. 107
——, note on case of nerve deafness in a syphilitic of thirty years' standing, Otol. 26
——, syphilis in relation to otology: an address introductory to a discussion on the subject, Otol. 1-24
——, three specimens of chronic middle-ear suppuration in each of which the opposite side was normal, the six bones being all of the diploëtic infantile type, Otol. 112
——, and JENKINS, G. J.—Case in which disease in a large sinus tympani caused persistence of discharge after the radical operation; caloric tests applied to the exposed inferior crus of the posterior semicircular canal, Otol. 54
Cheek, tumour of (C. F. Walters and N. S. Finzi), Laryng. 160
Cheeks, mucous membrane of (?) lupus erythematosus of, Laryng. 104
"Chelsea pensioner" remedy, Therap. 16
Chest, chair for screening, in skiagraphy, Electr. 88
——, see also *Respiratory tract*
Cheyne, cold and tepid applications employed by, Baln. 83
Cheyne-Stokes respiration during anæsthesia, case of (C. Hughes), Anæsth. 37
Chiari, multiple cartilaginous exostoses, quoted, Med. 1
Chicago, outbreak of anthrax among cows at, Epid. 74
Chick, sexual precocity in, Path. 305
Chicken-pox and scarlet fever, concurrent (J. D. Rolleston), Child. 14
Chilblains, effect of calcium lactate on low blood-pressure in subject of, Med. 66
Child, peculiar gait in (? functional ataxia) (J. A. Wright), Child, 191
——, tumour of rectus abdominis (J. P. Lockhart Mummery), Child. 7
CHILDE, C. P.—Twenty-three cases of Wertheim's abdominal panhysterectomy, Obst. 79-100, 106
Childhood, delayed development in, marine climate indicated for, Baln. 17
Children easiest subjects to hypnotize, Surg. 18
——, fractures about elbow-joint in, Surg. 6
——, hygienic measures to counteract predisposition to pulmonary tuberculosis in, Epid. 41, 42
——, infection with tuberculosis through alimentary tract, Child. 205, 221, 229, 231
——, injuries to facial nerve during aural operations on, Otol. 85
—— (Jewish), amaurotic idiocy (Tay-Sachs disease) occurring among, Path. 147, 148, 167, 174
——, kinds of solid food first allowed to, after weaning, Child. 119, 120
——, method of accustoming to artificial food after weaning, Child. 119
——, mortality from typhoid fever slight in, Epid. 10
——, patent foods for, origin and history of, Child. 135, 136
——, percentage infected with bovine tuberculosis, Child. 233, 234
—— —— —— with milk from tuberculous cows, small, Child. 222
——, prevention of dental caries in, Odont. 102
——, type of tuberculosis of tonsils in, Laryng. 68
——, ulcerative stomatitis in (S. F. Rose), Child. 70
—— under 1 year, frequency of oculo-motor type of polio-encephalitis in, Child. 89, 92

Children under 2 years of age, grouping of, in bills of mortality, possible explanation, Child. 120
—— (young), alcohol recommended for, in eighteenth century, Child. 123
—— ——, kinds of food recommended for, after weaning, in eighteenth century, Child. 122, 123
—— ——, and infants, progressive spinal muscular atrophy of (F. E. Batten), Neur. 53-84
—— ——, of crawling age, infection from subjects of tuberculosis, Child. 222, 223
——, see also *Illegitimate children*
Chimpanzee, sitting attitude of, Path. 312
Chin, entire skin of, and whole of lower lip, epithelioma involving, showing close clinical resemblance to actinomycosis (A. W. Williams), Derm. 14
Chinese dog, hairless, Path. 298
Chitral valley, endemic goitre in, Child, 78, 79
Chloral, enema of, following hypodermic injection of morphia in treatment of status epilepticus, Baln. 8
——, hypodermic injection before chloroform inhalation, Anæsth. 44
—— hydrate, oral administration before ether inhalation, Anæsth. 44
Chloroform, administration of, heart failure after change from recumbent to sitting posture during, Anæsth. 29
—— anæsthesia, bursting of gumma during, causing death by inspiration of gummatous material (W. J. McCardie), Anæsth. 25
—— ——, by, combined with suggestion, Anæsth. 22, 23
—— ——, one degree of, Anæsth. 7
—— ——, reactionary shock more frequent after ether than after, Anæsth. 2
——, asphyxia under (A. G. Levy), Path. 205-222
—— and asphyxia, influence of, upon cardiac inhibition, Path. 206
——, effect of medium anæsthesia of, on action of ergot, Therap. 132, 133
——, inhalation of, preceded by hypodermic injection of atropine sulphate, Anæsth. 53
—— —— —— —— —— of chloral, Anæsth. 44
—— —— —— —— —— of morphine, Anæsth. 43
—— —— —— —— —— —— and atropine, Anæsth. 44
—— —— —— —— —— of narceine, Anæsth. 44
—— —— —— —— —— of scopolamorphine and atropine, Anæsth. 50
—— —— —— —— —— —— ——, use of Vernon-Harcourt regulator in, Anæsth. 50
—— —— —— —— —— of strychnine and atropine, Anæsth. 54
—— —— —— —— —— of veronal, Anæsth. 44
—— poisoning, delayed, specimens from case of (A. W. G. Woodforde), Child. 81
—— —— ——, ætiology discussed, Child. 83
——, slight anæsthesia under, followed by dose of calomel in successful treatment of epilepsy complicated by acute mania, Baln. 7
Chloroleukæmia, myeloblastic, Path. 68
Chloroma, ætiology unsolved, Path. 69
——, atypical, case of (A. C. D. and J. C. G. Ledingham), Path. 60-69
—— —— ——, clinical history and post-mortem examination, Path. 60-63
—— —— ——, histological examination, Path. 63-67
——, bacteriology unworked, Path. 69
——, classification, Path. 69
——, connexion with leukæmia and leuko-sarcomatosis, Path. 68, 69
——, green tint in, partial distribution, Path. 67, 68
—— —— ——, striking feature, but not of first importance, Path. 68
——, infiltrates in, distribution, Path. 67, 68
Cholæmia ? congenital family (splenomegaly), case of (R. Hutchison), Clin. 129
Cholecystitis, bacterial infection causing, indicated by groups of abdominal symptoms, Electr. 42
Cholelysin, internal administration of, with farado-massage in extrusion of gall-stones, Electr. 44

Cholera (epidemic), more severe in London than in Manchester, Epid. 192
Cholesteatoma filling cavity in left mastoid process of woman, aged 75, cast showing smooth polished surfaces (F. H. Westmacott), Otol. 124
Cholesterin, probable finding of (1811), Med. 153
Cholesterol, absence from cerebro-spinal fluid in amaurotic idiocy (Tay-Sachs disease), Path. 157, 161
——, deposition in tissues, resulting from brain decay, as reason for greater prevalence of gall-stones among the insane, Path. 91
Choline, absence from cerebro-spinal fluid in amaurotic idiocy, Path. 155, 157, 161
Chondro-sarcoma, association with multiple exostoses, Med. 21
Chorda tympani, injury to, causing loss of taste, Otol. 93
—— —— nerve, injury to, resulting in disturbance of taste, Otol. 84
Chorionic cells, action of (James Young), Obst. 291
—— epithelium, mode of action of (James Young), Obst. 294
Chorionepithelioma, necrotic changes in, Obst. 298
Choroiditis due to oral sepsis, Odont. 31
—— —— —— syphilis, Odont. 31
——, metastatic, Path. 245, 246
CHOYCE, C. C.—Actinomycosis of the abdominal wall, Clin. 79
——, symmetrical gangrene of both legs, fingers of both hands, and nose, Clin. 77
Christian Science and voluntary loss of consciousness to pain, Anæsth. 19
—— ——, dangerous views urged by advocates of, Baln. 6, 7
Christiania, outbreak of milk-borne sore throat at, Epid. 97
Church, Sir Wm. S., Bt., K.C.B., on tuberculous contagion, quoted, Epid. 166
Church Stretton, health resort suitable for foreign invalids, Baln. 21
Chyliform ascites, syphilis, dropsy and albuminuria, case of (H. Batty Shaw), Clin. 112
Chyluria, absorption of fats in rectal feeding in case of, absent, Therap. 66, 67
Cicatrices resulting from ulcerating syphilitic lesions, keloids forming on (J. M. H. MacLeod), Derm. 74
Cicatricial obliteration of nasopharynx (hereditary syphilis) (Dan McKenzie), Laryng. 104
Cirrhosis, hepatic, and ascites, following splenomegaly with recurrent jaundice (F. P. Weber), Child. 62
—— ——, with congenital obliteration (or congenital atresia) of bile-ducts (F. P. Weber and G. Dorner), Child. 41-50
Clado, on appendiculo-ovarian ligament, quoted, Obst. 343
CLARKE, W. BRUCE.—Discussion on case of actinomycosis of abdominal wall, Clin. 82
——, discussion on transverse fractures of the patella, Surg. 84
"Claw" hand in amaurotic family idiocy (Tay-Sachs disease), Path. 150, 151, 155, 157, 159, 160
Claybury Asylum, incidence of primary carcinoma of gall-bladder and biliary passages as ascertained at, Path. 91, 92
—— ——, percentage incidence of gall-stones based upon post-mortem records of, Path. 89
CLAYTON-GREENE, W. H.—Discussion on fractures in the neighbourhood of joints, Surg. 36
——, method of removal of case of cyst of jaw, quoted, Odont. 17
——, and HARRIS, W.—The treatment of acute suppression of urine by incision and drainage of the kidneys, Clin. 161
Clift, first record of hairless mouse by, Path. 292, 293
Climate, damp, predisposing to diseases of joints, Baln. 28, 29
——, influence on habit of plants, Path. 308
—— in relation to gout, Baln. 119, 120
—— —— to hydrotherapy, Baln. 70
—— in treatment of neurasthenia (W. R. Gore), Baln. 37-44
—— of British health resorts compared with climate of Continental, Baln. 15
—— (over-stimulating, or "bracing"), when injurious, Baln. 69
——, possible influence on germ-plasm, Path. 307

Climates (cool), hot or thermal baths best borne in, Baln. 69
Climatic factor in bath treatment, Baln. 68
Climatology and balneology, reasons for association, Baln. 67
——, earliest references to, in literature, Baln. 1, 2
—— (medical), foundation of, assured, Baln. 68
CLIPPINGDALE, S.—Discussion on calcareous (hard) drinking water, Baln. 127
CLOGG, H. S.—Extensive necrosis of the lower jaw, Child. 30
——, specimen from case of necrosis of lower jaw, Child. 53
Cloquet, delimitation of rectum, quoted, Surg. 86
Club-hand, double congenital, of the radio-palmar variety, with absence of radius on both sides (A. H. Tubby), Child. 164
Coal-miners, comparative absence of phthisis among, how explained, Child. 219
COATS, GEORGE, and FORBES, J. GRAHAM.—Relation of *Meningococcus intracellularis* to pseudoglioma, Path. 242
Cobalt mines, Schneeberg, local prevalence of primary cancer of lung in, Path. 78
COBBETT, LOUIS, experimental results as to infectivity of bovine tuberculosis, quoted, Epid. 49
——, portals of entry in phthisis; an address introductory to a discussion on the subject, Child. 205-234
Cocaine, action of, Therap. 53-55
——, administration, mixed with food, in anorexia nervosa, Child. 80
—— and adrenalin solutions, plugging with, in acute frontal sinusitis, Laryng. 147
—— and hemisine solution, plugging middle meatus with, in acute frontal sinusitis, Laryng. 142, 146
——, application in acute frontal sinusitis, Laryng. 127
——, history of use of, Therap. 52
——, importance of local ischæmia, Therap. 53
——, strength of solution for dental purposes, Therap. 54
——, substitutes for, Therap. 55-58
——, symptoms of poisoning by, Therap. 54
Cocci, Gram-negative, Epid. 70
Cochlea, aqueduct of, in man and other animals, Otol. 115
—— of ornithorhynchus and that of other mammals, link between, method of finding suggested, Otol. 115
Codrenine, Parke Davis's, Therap. 60
Cohn, experimental work on auriculo-ventricular bundle, Therap. 98
Cohnheim, on tuberculous contagion, quoted, Epid. 164
——, "rest" theory of neoplasms, Med. 3
Coitus, as means of trypanosome infection, Path. 11, 12
Colchester, outbreak of milk-borne sore throat at, Epid. 95, 96
Colchicum, selective action, Electr. 45
Cold, sensitiveness to, marine climate indicated for, Baln. 17
Cold water dressings in surgery, early advocacy of, Baln. 87
COLE, PERCIVAL P.—Case of pseudo-hermaphroditism, Obst. 53-61
Coleman, outbreak of milk-borne sore throat at Surbiton reported by, Epid. 91
COLEMAN, FRANK.—A new form of stopcock for gas and air administration, Anæsth. 40
——, discussion on case of pneumococcal stomatitis, Odont. 12
—— —— on cyst of jaw, Odont. 16
Colitis (membranous), complicated with pyorrhœa alveolaris, treatment by vaccine, Clin. 140
——, treated by sulphur water lavage of large intestine, Therap. 19
Collapse (post-operative) following ether anæsthesia, Anæsth. 2, 3
Collar-bone, fractures of, advantage of slinging wrist to neck with elbow acutely flexed, Surg. 23, 24, 29, 45, 46
Colles's fracture, Surg. 3
—— ——, case of (H. P. Tayler), Electr. 72
—— ——, importance of early massage in, Surg. 29, 30

Colles's fracture, importance of slack bandaging in, Surg. 26
—— ——, reasons for operative treatment, Surg. 35
—— ——, reduction, method described, Surg. 11
—— —— —— of so-called impacted condition, Surg. 25
Collier, James, and Mummery, J. P. Lockhart.—Case of tabes dorsalis, with early rectal crises, Clin. 117
Collier, W., cyclic albuminuria in athletes, quoted, Baln. 110
——, discussion on after-history of albuminuria in adolescence, Med. 127
Collins, Sir W. J., connexion between diseases of eye and diseases of teeth, quoted, Odont. 33
Colloidal state, metals present in thermal waters in, Baln. 104, 105
Colloids, influence on crystalline form, Path. 136
Collum, R. W.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 9
Colney Hatch Asylum, percentage incidence of gall-stones based upon post-mortem records of, Path. 88, 89
Colon (ascending) and cæcum, resection of, case of, showing chronic hypertrophy of mucous membrane, with stenosis simulating cancer (C. Gordon Watson), Path. 237
——, dilatation (congenital) of, case of (J. P. Parkinson), Child. 75
—— —— ——, treatment, operative, methods, Child. 76
—— ——, idiopathic, hypertrophic, case of (J. B. Molony and H. D. Rolleston), Child. 18
—— —— —— —— ——, skiagram showing position of bismuth, after bismuth meal, Child. 20-22
—— ——, with putrefying fæces, associated with chronic recurrent tetany, Child. 17
——, flushing of (Plombières system), in neurasthenia, Baln. 41, 43
—— (pelvic), delimitation of pelvic rectum from, Surg. 92
—— ——, of fœtus, Surg. 89, 90, 92, 96, 97
——, spasm of, inhibited by belladonna, Med. 31
——, tuberculosis of, hyperplastic, Path. 239, 240
—— —— ——, simularity of fibromatosis of stomach to, Path. 240
—— —— ——, simulating cancer, Path. 239, 240
Colon-group, bacilli of, Epid. 63
Colorimeter, Meisling's, Baln. 47, 48
Colostomy, formation in operation for rectal cancer, Surg. 116
—— —— —— —— ——, advantages, Surg. 116
——, permanent formation, in abdomino-perineal operation for rectal cancer, Surg. 111, 118, 121, 131
—— ——, in palliative treatment of inoperable rectal cancer, Surg. 125
Colotomy, avoidance in congenital dilatation of colon, Child. 76
——, preliminary to operative treatment of cancer of rectum, Surg. 103
Colour-blindness, sex-limited disease, Med. 20
Colts, poliomyelitis in, Epid. 206, 207
Columbia, University of (New York), teaching of hydrotherapy at, Baln. 116
Colyer, J. F.—Discussion on experiments and observations on bread, with special reference to the causation and prevention of dental caries, Odont. 102
——, discussion on inflammatory eye conditions due to oral sepsis, Odont. 36
—— —— on malocclusion of permanent molars, Odont. 112
—— —— on model of maxilla with exostoses, Odont. 41
Comby, J., disinfection after measles, quoted, Child. 175
Complement-fixation after preliminary absorption of group antibodies, Path. 272
——, differentiation between *Bacillus paratyphosus* B and *Bacillus Aertryck* by, Path. 263, 266-270
—— —— between *Bacillus typhosus* and *Bacillus paratyphosus* by, Path. 252
—— —— of *Bacillus typhosus* from *Bacillus coli* by, Path. 251, 252
——, experiments as to, preparation of antisera, Path. 256
—— —— ——, preparation of bacterial extracts for, Path. 255

Complement-fixation, experiments, hæmolytic system, Path. 256
——— ——, results of, analysis, Path. 257-268
——— ——— ——, general discussion of, Path. 268-272
——— ——, strains of bacilli used in, Path. 256
——— ——, technique, Path. 256
——— ——, results as to hæmolysis, Path. 258-262, 264-271, 273-275
——— —— with extracts of normal and variant strains of *Bacillus coli*, Path. 103, 104
—— method compared with that of agglutination in differentiation of bacteria, Path. 252
—— —— in differentiation of bacilli, Epid. 71
—— —— of, classification of strains isolated from cases of food-poisoning by, Path. 253
——, practical applications, Path. 251
——, studies in, summary and conclusion, Path. 276
—— ——, with strains of typhoid, paratyphoid, and allied organisms (H. R. Dean), Path. 251-278
Condenser of new induction coil apparatus, Electr. 80, 81
Condylomata of external ear, Otol, 10
—— (syphilitic) of external ear, Otol. 10, 11
—— —— —— ——, painful, Otol. 20, 21, 23
—— —— —— —— followed by acute inflammation of middle ear, Otol. 11
Congenital anomalies and diseases, see under names of Organs and Regions
Congestions, absorption by subthermal bath treatment, Baln. 96
Conjoint tendon and Cooper's ligament, approximation in inguinal method of radical cure of femoral hernia, Surg. 166, 168
Conjunctiva and pharynx, pemphigus of (P. Watson-Williams), Laryng. 158
——, œdema of, from acute frontal sinusitis (A. J. M. Wright), Laryng. 160
Conjunctivitis accompanying pneumococcal stomatitis, Odont. 12
—— due to intranasal disease, Laryng. 103
Connective tissue, stimulation of, following application of diathermic current, Electr. 5
Conradi, frequency of *Bacillus paratyphosus*, Epid. 137
——, non-effective typhoid carriers, Epid. 124
—— and Hübener, association of *Bacillus typhosus* B with diseases other than paratyphoid fever and presence in healthy individuals, Epid. 54, 55, 70, 71
—— ——, ingestion of *Bacillus suipestifer* with impunity, Epid, 58, 59
Conrath, hyperplastic tuberculosis of cæcum, quoted, Path. 240
Consciousness, field of, Neur. 3
——, voluntary loss of ("Statuvolism"), Anæsth. 19
Constipation, cause of, attributed to hard water, Baln. 125
——, effect on low blood pressure, Med. 60
——, habitual, in women, Obst. 328
Constitution, phthisical, see *Disposition*
Consumptives (advanced), homes of refuge for, advocated, Epid. 31
—— ("cured"), difficulty in obtaining work, Epid. 31, 32
——, delay in entering sanatoria, Epid. 31
——, homes of, visitation to examine "contacts," Epid. 31, 38, 39
Contagion in tuberculosis, growth of belief in, Epid. 161-165
—— —— ——, nineteenth century investigations, Epid. 165, 166
—— —— ——, value of segregation, Epid. 171, 172
Continental health resorts, Baln. 12
—— —— ——, see also *Health resorts* (Continental)
Contrexéville, waters of, beneficial to gout, yet containing carbonate of lime, Baln. 123
Convalescence from acute disease, seaside climate indicated for, Baln. 17
——, subthermal baths during, Baln. 97
Converters, rotary, for wireless telegraphy, Electr. 83
Cooper, Sir Astley, "Dissection of a limb on which the operation for popliteal aneurysm had been performed," quoted, Med. 150, 151

Cooper's ligament, approximation of conjoint tendon to, in radical cure of femoral hernia by inguinal method, Surg. 166, 168
—— ——, definition of, Surg. 166
Corner, E. M.—Osteitis deformans, Clin. 160
——, trophic ulceration over a large formation of scar-tissue, Clin. 160
Cornu cutaneum with atrophic lichen planus (A. Whitfield), Derm. 11
Coronoid, fracture of, Surg. 10
—— ——, followed by traumatic myositis ossificans, Surg. 10
Corpus luteum cysts, association with hydatidiform mole, Obst. 220
Cossor radiator tubes, Electr. 63
Costo-chondral articulations, enlargement in children, Child. 108
Côte-d'Azur, Baln. 18
Cotte, case of division of posterior spinal nerve-roots for gastric crises of tabes, Surg. 213
Cotton, impacted fracture of femur, quoted, Surg. 14
Couch for use of patients under skiagraphy, Electr. 88
Cova, recommendation of hebosteotomy, Obst. 165
Cow, Douglas.—Reaction of arteries to certain drugs, Therap. 77-96
Cowen, R. J., first treatment of epithelioma with X-rays, advocated by, Electr. 58
Cow-horn, earliest form of sucking bottle, Child. 131
Cowhouses in London (1850), insanitary condition, Child, 134, 135
Cow-pox, see *Vaccinia*
Cows affected by mastitis, ingestion of milk from, causing septic sore throat and gastro-enteritis, Epid. 76
——, diseases of (excluding tuberculosis), affecting milk in relationship to human disease, (W. G. Savage), Epid. 73-104
——, gastro-enteritis in, Epid. 74
—— ——, decomposition of milk under, Epid. 74
——, infection by Gaertner bacillus, decomposition of milk under, Epid. 74
——, inoculation of diphtheria bacilli into, Epid. 85
——, outbreak of anthrax in, Epid. 73, 74, 102
——, scarlet fever in man reproduced in modified form in, Epid. 102
——, septic conditions in, Epid. 75
—— —— ——, effects on milk, Epid. 75, 102
—— (tuberculous), percentage of, Child. 221, 222
—— —— —— of children infected by milk from, small, Child, 222
——, ulcerated teats in, Epid. 81
——, see also *Mastitis* in cows
Coxa vara, Surg. 43, 45
—— ——, traumatic diagnosis, Surg. 13
—— —— ——, production of, Surg. 12
—— —— ——, treatment, Surg. 13
Coxon, S. A. T.—Discussion on experiments and observations on bread, with special reference to the causation and prevention of dental caries, Odont. 102
Craigmore, outbreak of milk-borne sore throat at, Epid. 90
Craniotomy, cause of danger to mother's life in, Obst. 204
——, increasing safety of Cæsarean section dispenses with, Obst. 216
——, indications for, and justifiability of, Obst. 165, 166, 168, 173, 174, 175, 195, 201, 202, 209, 210, 213, 214, 216
——, two classes of cases of, Obst. 202
——, unjustifiability of, Obst. 165, 182, 205, 211, 214, 216
Creed, Dr. (of Sydney), use of hypnotism in surgery, quoted, Anæsth. 19
Crenotherapy, see *Spa treatment*
Cretinism, cases of (E. J. Cross) (J. A. Wright), Child. 189
——, heredity in, question of, Med. 8
——, nervous form of, Child. 79

Cretinism, nervous, resemblance to cerebral diplegia, Child. 79
Crile, G., on brain of animals under ether and nitrous oxide respectively, alluded to. Anæsth. 2
Crile's method of suggestion before operations for exophthalmic goitre, Anæsth. 22, 23
Cripps, W. Harrison.—Criticism of extensive operation for rectal cancer, alluded to, Surg. 102
——, discussion on operative treatment of cancer of rectum, Surg. 105
Crocker, H. Radcliffe, case of congenital atrophy of skin, depicted by, Path. 302
Cromer, health resort suitable for foreign invalids, Baln. 20
Croom, Sir Halliday, practice as regards removal of appendix in pelvic operations, Obst. 353
Cross, E. J.—Case of cretinism, Child. 189
Cross, F. R.—Discussion on diseases of nose and accessory sinuses in relation to affections of eye and orbit, Laryng. 163
Crossen, H., adhesions between wall of appendix and cæcum in appendicitis in women, observed by, Obst. 347
Croup (inflammatory) complicating measles, Epid. 192
Crowborough, health resort suitable for foreign invalids, Baln. 21
Crowder, T. R., hyperplastic tuberculosis of cæcum, quoted, Path. 240
Crystalline "rubble" in cancellous uric acid calculi, Path. 118, 121, 130, 137
Crystallization, thermal increase arising as result of, effect on striation in calculi, Path. 141
Crystals, cuboidal, forming nucleus of cancellous uric acid calculi, Path. 113, 115, 117, 122, 141
——, elongation of, how produced, Path. 138-142
——, form of, influence of colloids on, Path. 136
——, forming calculi, comparison with uric acid sediments, Path. 133
—— —— nucleus of cancellous uric acid calculi, Path. 113, 114, 115, 116, 130
——, genesis of, must take place in supersaturated or labile solution, Path. 137, 138
——, minute, filling interspaces in cancellous uric acid calculi, Path. 116, 122
——, wedges, columns, and zones of, in compact variety of uric acid calculi, Path. 123, 124, 125, 126, 127, 128, 138
Cullen and Kelly, preservation of ovaries in operations for myomata of uterus, Obst. 117-118
Cullis, Miss, experimental work on excitation and section of the auriculo-ventricular bundle, Therap. 98, 103
Curetting, results of, in treatment of chronic endometritis (A. Donald and W. Fletcher Shaw), Obst. 37-49
Curgenven, J. B. and J. S., inunction treatment of measles, Child. 176, 177
Currie and Bramwell, Harvieston epidemic of poliomyelitis, reported by, Epid. 217
Currie, James, "Medical reports on the effect of water, cold and warm, as a remedy in fever and other diseases" (1797), Baln. 83
Cushny, A. R., F.R.S.—A plea for the study of therapeutics (presidential address), Therap. 1-12
——, discussion on drugs used as local anæsthetics in the mouth, Therap. 60
—— —— on ergot; a clinical experimental study, Therap. 150
—— —— on therapeutic use of digitalis group, Therap. 37
Cyanosis accompanied by polycythæmia, Child. 4
—— accompanying pericarditis, explanation, Med. 45
—— (chronic), and adherent pericardium, case of, blood-changes in, Clin. 92
—— (congenital), family history of, in case of congenital heart disease without murmur F. P Weber), Child. 159
Cyclic nature of natural functions, Med. 111
Cyllin, valuable bactericide, Obst. 213
Cynocephalus mormon (mandril), Path. 290
—— *sphinx* (Guinea baboon), Path. 288
Cyst (benign) of tibia (H. A. Lediard), Clin. 132
—— —— ——, description, Clin. 135

Cyst (benign), of tibia, description, microscopic, Clin. 138
—— (dentigerous) with cellular contents (W. W. James), Odont. 85
—— of jaw, cases of, method of removal, Odont. 17
—— —— resulting from traumatism (H. A. T. Fairbank), Odont. 14
—— —— —— ——, origin of, Odont. 14
—— (ovarian, suppurating), in girl aged 10, probably from hairpins impacted in vagina (H. J. Paterson) Obst. 259
—— (thyrolingual), removal from girl; operation followed by symptoms of acute thyroidism (H. J. Davis), Laryng. 27
Cystic disease of kidneys (J. Fawcett), Clin. 151
Cysts (dental) following septic irritation, Odont. 15-17
—— ——, origin of, Odont. 15-17
—— —— ——, epithelial, Odont. 15, 16
—— (malignant papillomatous), of both ovaries, removal followed six years later by carcinoma of rectum (M. A. Scharlieb), Obst. 358
—— (ovarian), in children (not including dermoids), (H. J. Paterson) Obst. 262
—— —— and peritoneal), with hydrosalpinx (H. Macnaughton-Jones), Laryng. 221

Dachshund, administration of thyroid extract to, Path. 299
——, thyroid gland of, Path. 299, 300
Dacryocystitis connected with nasal disease, Laryng. 168
Daiber, "Mikroskopie der Harnsedimente," Path. 135
DALE, H. H.—Discussion on drugs used as local anæsthetics in mouth, Therap. 59
——, discussion on reaction of arteries to certain drugs, Therap. 94
—— —— on use and action of ergot, Therap. 149
—— —— on value of rectal feeding, Therap. 74
—— —— on ergot: a clinical experimental study, Therap. 149
——, ergot researches, quoted, Therap. 115
——, on use of ergot in pulmonary hæmorrhage, quoted, Therap. 147
Dalton, epidemic of poliomyelitis at Cerne Abbas (1910), reported by, Epid. 216, 226
DALTON, N.—Discussion on Addisonian anæmia, Clin. 38
——, discussion on treatment of splenic anæmia by splenectomy, Clin. 70
—— —— on varix of right superficial epigastric vein, Clin. 51
Daltonism, Med. 7
Damp, predisposing to diseases of joints, Baln. 28, 29
Daniels, case of ulcerating granuloma inguinale, quoted, Derm. 135
Danlos, case of macular atrophy in syphilis, quoted, Derm. 124
D'Arsonval's currents, distinguished from diathermic currents, Electr. 1, 2
Darwin, theory of pangenesis, Path. 307
Darwinian theory, attitude of Virchow towards, Path. 306
—— varieties, grosser departures constituting foundation of, Path. 306
Dastre, danger of morphine injection before chloroform inhalation, Anæsth. 43
—— and Morat, injection of morphine and atropine before chloroform inhalation, Anæsth. 44, 55
DAVIS, HALDIN.—Case of lichen scrofulosorum, Derm. 19
—— (for H. G. ADAMSON), case of cheilitis exfoliativa, Derm. 83
Davis, Hall, cyst of vagina, quoted, Obst. 21, 22
DAVIS, H. J.—Acute vertigo, with repeated falls always to the left, during two months, Otol. 71
——, caries and necrosis of temporal, parietal, and occipital bones following mastoiditis due to scarlet fever in girl, aged 5; operation; recovery, Otol. 70
——, case of leprosy in a man, Laryng. 25
——, discussion on case of antrum suppuration: nasal antrostomy followed by blocking of nasal ducts, Laryng. 102

Davis, H. J.—Discussion on cast of cavity in the left mastoid process filled with cholesteatoma, Otol. 134
——, discussion on case of fungating carcinoma of œsophagus treated by radium, Laryng. 8
—— —— —— of large growth in larynx, Laryng. 85
—— —— —— of lateral sinus thrombosis, Otol. 33
—— —— —— of nasopharyngeal fibrous polypus, Laryng. 61
—— —— —— of removal of rabbit-bone from right bronchus, Laryng. 97
—— —— on ectopia of pituitary, Laryng. 54
—— —— equine larynx and "ventricle-stripping," Laryng. 91
—— —— on injuries of ear, Otol. 104
—— —— on syphilis in relation to otology, Otol. 23
—— —— on treatment of frontal sinus discharge, Laryng. 148
—— —— on two cases of cerebellar abscess, Otol. 63
——. empyema of antrum in man, aged 71, now malignant (sarcoma), Laryng. 30
—— —— of maxillary antrum in boy, Laryng. 31
——. extradural cerebellar abscess tracking through the jugular foramen into neck, Otol. 71
—— —— —— —— —— —— —— —— ——, sloughing of middle third of internal jugular vein; operation; recovery, Otol. 28
——. large nasopharyngeal polypus removed from boy, Laryng. 32
——. removal of the upper jaw in a man, aged 71, Laryng. 86
——, specimen of temporal bone from case of thrombosis of cavernous sinus, Otol. 27
——, thyro-lingual cyst removed from girl; operation followed by symptoms of acute thyroidism, Laryng. 27
——, unilateral optic neuritis and complete ophthalmoplegia externa resulting from acute sphenoidal sinusitis, Laryng. 28
Dawson, G. W.—Case for diagnosis, Derm. 102
——, case of fibromatous tumours in a man, aged 55, Derm. 101
—— ——, multiple facial tumours, Derm. 3
——, discussion on case for diagnosis, Derm. 63
—— —— on epithelioma of back of hand, Derm. 142
—— —— on macular atrophy associated with lupus erythematosus, Derm. 144
——, extensive lupus erythematosus associated with symmetrical gangrene in a man, aged 49, Derm. 101
——, lichen annularis in a man, aged 40, Derm. 3
——, peculiar affection of the ankles in a lad, aged 18, Derm. 3
——, syphilitic keloid in a man, aged 47, Derm. 3
—— (for L. F. Knuthsen), case of purpura, Derm. 60
Deaf-mutism, congenital syphilis and, Otol. 3, 21
—— due to congenital syphilis, rarity, Otol. 3
Deafness and blindness (temporary) due to intestinal toxæmia, case of (A. L. Whitehead), Otol. 121
—— and discomfort in right ear as early symptoms in case of epithelioma originating near right Eustachian tube, fatal termination (E. Law), Otol. 67
—— due to occlusion of Eustachian tubes by scar-tissue (D. R. Paterson), Otol. 109
—— —— —— —— —— —— ——, treatment discussed, Otol. 110
—— in hemiplegia, Otol. 13
—— (internal ear), perversion of taste and facial paralysis associated with herpes of neck, Otol. 17
—— —— ——, and tinnitus in tabes, Otol. 17
—— (labyrinthine) due to injury, irremediable, Otol. 103
—— (syphilitic, acquired, tertiary), Otol. 14-18
—— ——, congenital, Otol. 4
—— —— ——, age-incidence, Otol, 4, 19
—— —— ——, diagnosis, Otol. 8
—— —— ——, lesion accompanying, Otol. 4, 6, 8

Deafness (syphilitic), congenital, lesions producing, nature of, Otol. 7
—— —— —— —— ——, site of, Otol. 7
—— —— ——, mode of onset. Otol. 4
—— —— —— —— —— with giddiness, Otol. 4, 5, 7
—— —— —— —— —— —— and vomiting, Otol. 4, 5
—— —— —— —— —— without giddiness, Otol. 4, 7
—— —— ——, sex-incidence, Otol. 4
—— —— ——, treatment, Otol. 8, 9, 23
—— —— —— —— by blistering, Otol. 9, 19
—— —— —— —— by mercury and iodide of potassium, useless, Otol. 8, 22
—— —— —— —— by pilocarpine injections, Otol, 9
—— —— —— ——, preventive, Otol. 9, 19
—— and tinnitus in early case of general paralysis, Otol. 18
—— (unilateral) occurring in tabes, Otol. 61
—— ——, probably complete, associated with vertigo (Dan McKenzie), Otol. 60
——, see also *Nerve-deafness*
Dean, G., and Todd, G., isolation of diphtheria bacillus from ulcerated teats, Epid. 82, 185
DEAN, H. R.—Experiments as to differentiation of *Bacillus typhosus*, *paratyphosus* B, and *paratyphosus* A, quoted, Path. 253
——, non-effective typhoid carriers, quoted, Epid. 124
——, studies in complement fixation with strains of typhoid, paratyphoid, and allied organisms, Path. 251-278
Death, causes of, discovery frequently impossible from Registrar-General's mortality returns, Epid. 188
—— ——, verification, Epid. 197
Debility (thermal), dangers of, Baln. 69, 95
Decidua, function of (James Young), Obst. 302
—— ——, and action of chorionic cells (James Young), Obst. 291
Defence neurosis, hysteria originating as, Neur. 12, 13
—— hysteria, phobias and obsessions accounted as defence neuroses, Neur. 12
Degeneracy, asymmetry as stigma of, Odont. 106
Degenerates, bad subjects for hypnotism, Anæsth. 20
Delbet, P., case of gunshot injury to ear, quoted, Otol. 99, 100
DÉLÉPINE, Professor A. SHERIDAN.—Discussion on paratyphoid fever and "food-poisoning" (communication by letter), Epid. 63
——, investigation of Derby pork-pie poisoning case, Epid. 65
——, and Rieder, "Atlas of urinary sediment," quoted, Path. 132, 133, 184, 135
Delirium cordis, Med. 92, 96
—— ——, association with progressive sclerotic myocarditis, Med. 96
—— —— —— with rheumatic heart disease and degenerative cardio-vascular changes, Med. 92
—— ——, identical with auricular fibrillation, Med. 92, 93, 96
Delivery obstructed by ovarian tumour fixed by suction in pelvis (J. D. Malcolm), Obst. 253
—— (spontaneous), Obst. 216
Delsaux's operation in chronic frontal sinus suppuration, Laryng. 134, 143
Demarquay, morphine injection before chloroform inhalation, quoted, Anæsth. 43
DEMPSEY, M.—Discussion on case of endothelioma of palate, with metastatic growth in neck, treated by radium, Laryng. 95
Denmark, State support for treatment of cleft palate in, Surg. 174
DENSHAM, ASHLEY.—Discussion on inflammatory eye conditions due to oral sepsis, Odont. 35
DENT, C. T.—"Congenital elephantiasis" of the arm, Child. 24
Dental pulp, nerve-fibres of, distribution (J. Howard Mummery), Odont. 51
Denticles, report on, in case of multiple supernumerary teeth, Odont. 44, 45,
Dentigerous cyst with cellular contents (W. W. James), Odont. 85
Dentine, decalcification by fermented milk, Odont. 4, 5, 6, 9

Dentine, liquefaction in dental caries, Odont. 6
"Dents de scie" of vocal cords, case illustrating (W. J. Horne), Laryng. 10
Denture removed from œsophagus (N. Paterson), Laryng. 54
Derby, food-poisoning at, following eating of pork pie, Epid. 65
Dermatitis, chronic, of pinna, due to infection by *Staphylococcus aureus*, under treatment by vaccine (C. E. West), Otol. 38
—— herpetiformis mistaken for pemphigus foliaceus, Derm. 17
Dermatosis (chronic), carcinomatous degeneration in (Cecil Rowntree), Clin. 159
Dermo-fibromatosis, familial pigmentary (J. D. Rolleston and N. S. Macnaughtan), Clin. 71
—— —— ——, growth on upper lip in one case of, Clin. 75, 77
—— —— —— relation to v. Recklinghausen's disease, Clin, 74, 75
Dermoid tumour on back, probable case of (O. L. Addison), Child. 165
De Santi, P., see under *S*
Desault, Pierre (1675-1737), on the phthisical disposition, quoted, Epid. 154
Déspine, J., introduction of use of shower-bath from Scotland into Savoy, Baln. 83
Deszo, Dr. K., Lecturer on Hydrotherapy and Climatology at University of Budapest, Baln. 115
Determann's clinical viscosimeter, Clin. 88
Deucher, absorption of fats in rectal feeding, quoted, Therap. 66
——, percentage of absorption of carbohydrates in rectal feeding, quoted, Therap. 65
Development, defect of, in causation of malocclusion of permanent molars, Odont. 112
——, excess of, one of the foundations of Darwinian variation, Path. 306
Dextrose, absorbability in rectal feeding, Therap. 65
——, addition to rectal salines, effect of, Therap. 69, 70
—— in rectal feeding, Therap. 73
—— —— ——, value of, Therap. 73
——, subcutaneous administration, Therap. 74
Diabetes causing iritis, Odont. 28, 29
——, chemical pathology of inquiries into (1810-1850), Med. 152
—— mellitus, urine of, inquiry as to urea in (1811), Med. 152
Diacetic acid in urine during rectal feeding, Therap. 74, 75
Diagnosis, cases for (Noel Bardswell), Laryng. 15
—— —— (G. W. Dawson), Derm. 102
—— —— (T. Jefferson Faulder), Laryng. 14
—— —— (T. Colcott Fox), Derm. 21, 34
—— —— (E. G. Graham Little), Derm. 73
—— —— (J. M. H. MacLeod), Derm. 90
—— —— (Sir M. Morris), Derm. 25
—— —— (Sir M. Morris and S. E. Dore), Derm. 107
—— —— (F. A. Warner and W. Fox), Derm. 40
—— —— (A. W. Williams), Derm. 95
—— —— (A. J. Wright), Laryng. 56
—— ——, family nervous disease (C. A. Basker), Child. 198
—— ——, imperfect osteogenesis? (Frank Kidd), Clin. 106
—— ——, leprosy, commencing (E. G. Graham Little), Derm. 60-63
—— ——, syphilide (E. G. Graham Little), Derm. 52, 53
—— ——, relapse syphilide (S. E. Dore), Derm. 70
—— ——, ulceration of left vocal cord in male, aged 67 (G. C. Cathcart), Laryng. 63
——, clinical methods of, present knowledge contrasted with that of 100 years ago, Med. 149
——, physical methods of, advances in, Med. 150
——, value of X-rays in, testimony to, Med. 150
Diaphragm, high inspiratory position of, in acute pericarditis, Med. 49
——, inhibition of action of, as sign of pericarditis, Med. 40
—— —— ——, observed by X-rays, Med. 44
—— —— ——, unilateral, due to diseases of lungs and pleura, Med. 40

Diarrhœa, milk-borne, outbreaks of, Epid. 97, 98
——, prevalence of, relation to weather, Epid. 190
——, sulphur treatment, Therap. 15, 16
——, see also *Fæces* (diarrhœal)
Diathermic currents, action on heart and circulation, Electr. 6, 7
—— ——, distinguished from d'Arsonval's currents, Electr. 1, 2
—— ——, entrance resistance absent in, Electr. 3
—— ——, localization of effects in tissues and fluids, Electr. 2, 3
—— ——, treatment by (F. Nagelschmidt), Electr. 1-8
—— —— ——, advantages, Electr. 5
—— —— ——, distinct from fulguration and Forrest's needle, Electr. 4
—— —— ——, effects on tissue destructive or restorative of function, Electr. 4
—— —— ——, followed by formation of keloid, Electr. 9, 11
—— —— ——, hyperæmia following, Electr. 5, 6
—— —— ——, indications for, Electr. 7, 10-12
—— —— ——, limitations, Electr. 4
—— —— ——, maximum strength of current permissible, Electr. 9, 12
—— —— ——, of asthma, Electr. 8
—— —— ——, of cancer, Electr. 4, 5, 11
—— —— —— ——, avoidance of knife except for surface incisions, Electr. 11
—— —— ——, of chronic nephritis, Electr. 8
—— —— ——, of gout, Electr. 7
—— —— ——, of laryngeal tuberculosis, Electr. 6
—— —— ——, of œdema, Electr. 7
—— —— ——, of tabes, Electr. 7
—— —— ——, operative, Electr. 8
—— —— ——, stimulation of connective tissue following, Electr. 5
—— —— ——, technique, Electr. 9-12
—— —— ——, two classes of case submitted to, Electr. 4
Diathermy, Electr. 51, 59
Diathesis ("Disease-Anlage"), distinguished from "disposition," Epid. 151
—— (tuberculous), influence of Bayle on idea of, Epid. 155; see also *Disposition*, phthisical
Dichloracetic acid, growth of bacteria on, Path. 108
Diet in low blood-pressure, Med. 74
——, influence upon formation and healing of acute ulcer of stomach (C. Bolton), Path. 53-59
—— of young children during eighteenth century, Child. 122, 123
Dietl, sceptical ideas as to therapeutics, Baln. 85
Digalen, Therap. 35
Digby, K.—Case of cicatrizing sarcoma (shown for C. J. Symonds), Derm. 126
Digestion, defective cyclic albuminuria due to, Med. 111
——, impairment of, creating vicious circle, Baln. 39
——, slight power of, possessed by large intestine, Therap. 63
Digitalis, and vomiting, Therap. 30, 35-38
——, danger of use owing to blood-pressure, Therap. 36, 37
——, difficulties in use of, Therap. 29, 30
——, dosage of, Therap. 26, 36
——, effect of atropine on pulse-rate in cases of heart disease under influence of, Therap. 192-210
—— —— on low blood-pressure, Med. 67, 74, 75, 78
——, reaction of arteries to, Therap. 84
——, therapeutical value tested by accurate clinical records, Therap. 7-12
Digitalis-group of drugs, therapeutic use of (H. H. Turnbull), Therap. 25-38
—— —— ——, value in rheumatic heart disease, Therap. 26
Dilution, principle of spa treatment, Baln. 110

Dimmock, Lieut.-Col. H. P.—Columnar-celled carcinoma of the ovary, Obst. 62
—— —— —— ——, report of pathology committee on, Obst. 63
Dioxydiamidoarsenobenzol, see *Ehrlich-Hata's* remedy, *Salvarsan*
Diphtheria (bovine), Epid. 83, 85, 101
——, carriers of, Epid. 33, 144
——, gangrene of leg following (J. D. Rolleston), Child. 10
—— of fauces, complicated by hæmorrhages, Surg. 49
—— outbreak, concurrent with scarlet fever and derived from same milk-borne source, Epid. 101
—— ——, milk-borne presumably, from infection from ulcerated teats of cows, Epid. 82, 83, 85, 101
—— and septic sore throat, milk-borne, outbreak of, at Leith, Epid. 96
——, sequelæ of, varying, Epid. 194
—— ——, see also *Hemiplegia* and paralysis (post-diphtheritic)
Diplegia (cerebral athetotic), case of (E. Cautley), Child. 77
—— (cerebral), resemblance of nervous cretinism to, Child. 79
Diploëtic infantile type of bone, in specimens of chronic middle-ear suppuration in which opposite side was normal (A. H. Cheatle), Otol. 112
Dirt, factor in causation of cancer, Electr. 51
"Disease-Anlage" (diathesis), definition and use of word, Epid. 150, 151
Disease, chronic, advances in knowledge of, Baln. 111, 112
—— ——, alterative treatment, Baln. 110
—— ——, causes of, Baln. 112
—— ——, spa treatment alone concerned with, Baln. 112
—— ——, treatment by water of medicinal springs, Baln. 118
Diseases, relation of, to state of weather, Epid. 189
Disinfection after measles, Child. 175
—— in prevention of spread of pulmonary tuberculosis, Epid. 42
Dislocation of head of humerus complicating fractures of upper end, treatment, Surg. 5, 6
—— —— —— —— —— —— —— ——, indications for removal of fragments, Surg. 6
—— —— —— —— —— —— —— ——, by manipulation, Surg. 5-6
—— —— —— —— —— —— —— ——, by reduction (McBurney's method), Surg. 6
—— of pulp of temporary incisor, with subsequent removal of pulp and apex intact (C. F. P. Baly), Odont. 69
Dispensaries (anti-tuberculosis), local, Epid. 39, 40, 44
—— —— ——, agency in home visitation to examine tuberculosis "contacts," Epid. 39, 40
Disposition in disease, definition of, Epid. 149-151
——, phthisical, evidence of, bacteriological and pathologico-anatomical, Epid. 167, 168
—— —— —— of belief in, from medical classics, Epid. 151-153
—— —— —— —— —— writings of seventeenth and eighteenth centuries, Epid. 153-155
—— —— —— —— —— writings of nineteenth century, Epid. 156-160
—— —— ——, clinical, Epid. 172-174
—— —— ——, epidemiological and statistical, Epid. 168-172
—— ——, theory of transmitted phthisical habitus, Epid. 172
——, problem of pulmonary tuberculosis considered from standpoint of (W. Bulloch and Major Greenwood, jun.), Epid. 147-184
Diuretic action of sulphur water, Baln. 50
Divers, spontaneous hæmorrhage into tympanic cavity in, Otol. 84
Diverticulum, congenital (large), of bladder, treated by resection (J. Berry), Surg. 158
Diving, injuring membrana tympani, Otol. 84
Dixon, W. E.—Discussion on rôle of auto-inoculation in medicine, Therap. 49
——, excitation and section of the auriculo-ventricular bundle, Therap. 97-110
Djelaleddin-Moukhtar on *Trichophyton roseum* and *ectothrix*, quoted, Derm. 94

Döderlein, primary mortality from Wertheim's operation, Obst. 95
Dog, auricular fibrillation in, Med. 100
——, intestine of, intermediate forms of *Bacillus coli* and *Bacillus typhosus* in, Epid. 118
Dogs (hairless), specimens of, Path. 297, 298
——, immune to infection with Gaertner's bacillus, Epid. 69
Don, Dr., thesis on treatment of skin diseases by thyroid extract, Derm. 72
Donald, Archibald, and Shaw, W. Fletcher.—The symptoms and treatment of chronic endometritis, with special reference to the results of curetting, Obst. 37-49
Donelan, J. Discussion on case of leprosy, Laryng. 27
——, discussion on plaster casts of unusual malformation of upper jaw, Laryng. 76
—— —— on the treatment of frontal sinus discharge, Laryng. 141
——, large fistulous opening into anterior wall of left maxillary antrum, Laryng. 18
——, swelling at junction of right vocal cord and apex of vocal process, Laryng. 80
Doran, Alban.—Discussion on case of adenomatosis vaginæ, Obst. 25
——, discussion on hydrosalpinx with ovarian and peritoneal cysts, Obst. 222
—— —— on hysterectomy, followed by double phlebitis, Obst. 224
——, subtotal hysterectomy for fibromyoma uteri; forty additional histories, Obst. 115-142, 145
Dore, S. E.—Case for diagnosis, Derm. 70
——, discussion on case for diagnosis, Derm. 91
—— —— on case of lupus erythematosus associated with ulceration, Derm. 54
——, monilithrix in a male child, aged 16 months, Derm. 71
——, and Morris, Sir Malcolm.—Acute lupus erythematosus in girl, aged 21, Derm. 106
—— ——, case for diagnosis, Derm. 107
—— ——, extensive scarring and pustular eruption following Scinde ulcers, Derm. 107
—— ——, tylosis in a woman, aged 49, Derm. 108
—— (for Sir Malcolm Morris).—Case of summer eruption, Derm. 4
Dorner, G., and Weber, F. Parkes.—Case of congenital pulmonary stenosis with special consideration of the nature of the secondary blood-changes, Clin. 85-94
—— ——, congenital obliteration (or congenital atresia) of bile-ducts, with hepatic cirrhosis, Child. 41-50
Dorset, cause of swine fever, quoted, Epid. 59
Douche, cool or cold, Baln. 95
Douche-bath, subthermal, without pressure, Baln. 95
——, use of, by Charcot, Baln. 88
"Douche écossaise," origin of, Baln. 83
Douches, hot, value of, Baln. 99
——, hot and cold, application to spine in neurasthenia, Baln. 41
Douglas, C. G., and Haldane, J. S., estimation of total oxygen capacity and total blood volume in case of congenital pulmonary stenosis, Clin. 88, 89
Douglas's pouch, abscess in, resulting from appendicitis, Obst. 348
Dourine, or *mal de coit*, transmission of, Path. 2, 11
Dover, outbreak of milk-borne sore throat at, Epid. 89
Downie, J. Walker, post-mortem examination of case of congenital syphilitic deafness, quoted, Otol. 7, 8
Dowsett, E. B.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 50
——, discussion on cyst of jaw, Odont. 16
Drainage-tube, insertion into sinus, through fronto-nasal canal, to avoid infection after operation, Laryng. 144
Drescher, multiple cartilaginous exostoses, quoted, Med. 2
Dressmakers, Heberden's nodes among, Baln. 32
Drew, Douglas.—Case of supposed hermaphroditism, Obst. 109
——, chronic pyæmia of five years' duration following suppurative epiphysitis of the upper end of the humerus, Child. 162

Drew, Douglas.—Discussion on malformation of femur, Child. 165
——, discussion on treatment of cleft palate, Surg. 180
—— —— on tumour of rectus abdominis in child, Child. 8
——, injury to the brachial plexus, Child. 8
——, sarcoma of the uterus in a patient, aged 25, Obst. 111
——, separation of the lower epiphysis of the humerus, with displacement, Child. 9
Droitwich, Baln. 15
Dropsy, syphilis, chyliform ascites, and albuminuria, case of (H. Batty Shaw), Clin. 112
Drugging of infants by monthly nurses, Child. 141
Drugs, as local anæsthetics in mouth (F. R. Smyth), Therap. 51-58, 60-62
—— —— ——, substitutes for, Therap. 55-58
——, hypodermic injection before inhalation anæsthesia (D. W. Buxton), Anæsth. 43-56
—— —— —— —— —— ——, in women, Anæsth. 51
—— —— —— —— —— ——, length of time beforehand of making injection when one injection used, Anæsth. 51
——, unscientific use of, Therap. 2, 3, 4
Dublin Foundling Hospital, excessive infant mortality at (1775-96), Child, 130
Dubois, injection of chloral before chloroform, Anæsth. 44
Duckworth, Sir Dyce.—Discussion on outlines of medical hydrology (Hyde Memorial Lectures: Lecture II, Modern hydrotherapeutics), Baln. 99
Ductus arteriosus, patent, Clin. 83
—— —— ——, case of (T. R. Whipham), Child. 31
—— —— —— (?) case of (congenital heart disease) (G. S. Haynes, for E. Lloyd Jones), Child. 191
—— —— ——, heart showing infective polypoid thrombus in pulmonary artery extending into right ventricle mistaken for (T. R. Whipham), Child. 199
Dudfield, R.—Discussion on problem of pulmonary tuberculosis, Epid. 183
——, discussion on tuberculosis and its prevention, Epid. 46
——, and Bainbridge, F. A., outbreak of paratyphoid fever, quoted, Epid. 57
Dudgeon, L. S., and Shattock, S. G.—Experiments with air-dried mouse cancer: I, The lethal effect of drying upon mouse-cancer; and II, the inability of air-dried mouse cancer, or of a centrifugalized extract, to produce prophylactic or curative immunization, Path. 73-86
Dukes, C.—Discussion on after-history of albuminuria in adolescence, Med. 129
Dulcitol fermentation by different strains of *Bacillus typhosus*, Path. 48
—— —— —— —— —— ——, increased powers of, Path. 47
—— —— —— —— —— —— —— ——, not generally sustained, Path, 49, 50
—— —— —— —— —— —— —— ——, transference to agar cultures, Path. 47, 48
—— —— —— —— —— —— —— ——, vary with length of sojourn in dulcitol-containing medium, Path. 48, 52
—— —— —— —— —— ——, time required for, Path. 46
Dunbar, epizootics associated with *Bacillus enteritidis* noted by, Epid. 61
Duncan, W. H.—Post-mortem report on case of infective polypoid thrombus in pulmonary artery extending into right ventricle, mistaken for patent ductus arteriosus, Child. 201
Duncanson, J. Gray.—Discussion on reaction of arteries to certain drugs, Therap. 94
——, discussion on therapeutic use of the digitalis group, Therap. 36
—— —— on value of rectal feeding, Therap. 73
Dunn, H. Percy.—Remarks on case of unilateral optic neuritis and complete ophthalmoplegia externa resulting from acute sphenoidal sinusitis, Laryng. 29
Duodenum, ulcer of, effect of oral administration of belladonna before meal on acid and peptic values of stomach contents in, Med. 26
Durham, H. E., discovery of *Bacillus suipestifer* (*Aertryck*) by, Epid. 53
Durham's tubes, testing gas production of intestinal bacteria under use of, Path. 100, 105
Dust, factor in causation of cancer, Electr. 51
——, inhalation in pulmonary tuberculosis, Child. 128

Dust, inhalation, with sharp spicules, leading to infection by pulmonary tuberculosis, Child. 217

Dwight and Germain, cases of oral sepsis causing thrombosis of cavernous sinus, Odont. 113, 116

Dynamic test employed to prove comparative strength of cast and rolled gold plate, Odont. 60

Dyschondroplasia, multiple cartilaginous exostoses associated with, Med. 3

—— —— enchondromata associated with, Med. 3

Dysdiadokokinesia in cerebellar tumours, Otol. 36, 37

Dysentery, prevalence of, relation to weather, Epid. 190

——, sulphur treatment, Therap. 15, 16, 21

——, treatment by mercury, recommended (1811), Med. 148

Dysmenorrhœa and appendicitis, causal relationship between, Obst. 356

—— in chronic endometritis, results of treatment by curetting, Obst. 43

——, membranous, Obst. 44

Dyspepsia (atonic) with gastric dilatation, due to disturbances of nervous system, treatment by high-frequency current, Electr. 40

—— (functional), classification of forms, Electr. 34

—— ——, first stage (hyperchlorhydria), Electr. 34

—— ——, second stage (hyperchlorhydria and motor insufficiency), Electr. 35

—— —— —— —— —— —— ——, treatment by electricity, Electr. 35

—— ——, third stage (motor insufficiency and hypochlorhydria, treatment by electricity, Electr. 36

—— —— —— —— —— —— ——, with dilatation, treatment by farado-massage, Electr. 38

—— —— —— —— —— —— —— —— —— by lavage and electricity (intragastrically), Electr. 36

—— (gouty), treatment by high-frequency current administered by auto-condensation, Electr. 41

——, use of belladonna in (F. C. Moore and L. Kilroe), Med. 23-34

Dysphagia (in carcinoma of œsophagus) treated by new form of intubation apparatus (W. Hill), Laryng. 9

Dyspnœa accompanying pericarditis, explanation, Med. 45

——, relief of, by venesection in case of pneumococcal bronchitis, Med. 102

—— (urgent) and œdema of glottis in woman, aged 50, due to hæmorrhage into thyroid (A. Evans), Clin. 25

Ear (external), gummatous ulceration mistaken for epithelioma, Otol. 12, 19

—— ——, injuries to, resulting in malignant disease, Otol. 83

—— ——, malformation of, in case of congenital heart disease (F. P. Weber), Child. 161

—— —— and middle, injuries of (W. Milligan), Otol. 79

—— ——, syphilis of, acquired, primary, Otol. 10

—— —— —— ——, secondary, Otol. 10

—— —— —— ——, tertiary, Otol. 12

—— —— ——, congenital, Otol. 2

—— ——, syphilitic condylomata of, Otol. 10, 11

——, foreign bodies in, attempts to extract, proving fatal, Otol. 97

——, gunshot injuries to, Otol. 98, 99, 104

—— —— ——, followed by facial paralysis, Otol. 99, 100

—— —— ——, treatment, Otol. 102

——, hæmorrhage from, following fracture of petrous bone, Otol. 90

——, lesions, questions whether traumatic, medico-legal bearing, Otol. 104, 105

—— (middle), exostoses in, case of (A. H. Cheatle), Otol. 56

—— ——, large tuberculous polyp removed from, in infant (H. J. Marriage), Otol. 41

—— ——, suppuration (chronic), three specimens of, in each of which opposite side was normal, the six bones being all of diploëtic infantile type (A. H. Cheatle), Otol. 112

Ear (middle), syphilis of, acquired, primary, Otol. 10
—— —— —— ——, secondary, Otol. 11, 12
—— —— ——, congenital, Otol. 2
—— —— —— —— with nerve-deafness, Otol. 21
—— —— and internal, complicated by fractures of base of skull, Otol. 87
—— —— ——, burns and scalds of, Otol. 95, 96
—— —— ——, injuries of (C. A. Ballance), Otol. 87
—— —— —— —— to, resulting from "freak" treatment, Otol. 95, 104
—— —— —— ——, symptoms, Otol. 90
—— —— —— ——, treatment, principles of, Otol. 100-103
——, mutilating operations on, among savage tribes, Otol. 103
——, neuritis of, Otol. 15
——, operations on, injury to facial nerve during, Otol. 85
——, perceptive portion, syphilis of (acquired, secondary), Otol. 12
—— —— —— ——, congenital, Otol. 3
—— —— —— —— (tertiary), Otol. 14
——, suppuration in, treatment by Bier's method, objections to, Otol. 59
—— (syphilis of), treatment by Ehrlich-Hata's remedy, "606," Otol. 9, 12, 22, 2
Ears, pain in, caused by adhesion of soft palate to pharyngeal wall, Otol. 13
—— (external), palate, pharynx, &c., involved in case of leprosy (G. N. Biggs), Otol. 120
Earth (The), construction of centre, theories regarding, Path. 143
—— —— ——, probably radially crystalline, Path. 143
——, internal heat of, Path. 143
Eastbourne, health resort suitable for foreign invalids, Baln. 20
Eck, "Hydrotherapy in croup" (1834), Baln. 77
Eckersdorff, cases of paratyphoid fever (A) in Sumatra, reported by, Epid. 53, 54
Eclampsia gravidarum, Cæsarean section in, communications on, summarized (1909-1910), Obst. 8
Ectopia of pituitary, with other congenital anomalies of nose, palate, and upper lip (A. R. Tweedie and A. Keith), Laryng. 47-52
—— vesicæ in female child, case of (E. M. Woodman), Clin. 157
Eczema, in relation to dental trouble, Odont. 76
Eczematoid ringworm of hands and feet, illustrated by series of ringworm cultures (A. Whitfield), Derm. 111
Eden, T. W.—Discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 209
——, removal of uterus in cases of contracted pelvis and protracted labour with infection of uterine cavity, Obst. 166
Edge, Frederick.—Discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 185
Edgecombe, W.—Discussion on balneological treatment of neurasthenia, Baln. 43
——, some observations on low blood-pressure, Med. 51-80, 151
Edmunds, W.—Action of pro- and anti-thyroid preparations, Path. 233-236
Edsall and Miller, absorptions of proteins in rectal feeding, quoted, Therap. 64
—— ——, percentage of absorption of fats in rectal feeding, quoted, Therap. 66
Education, medical, in relation to laryngology, Laryng. 1-5
Edwards, F. Swinford.—The operative treatment of cancer of the rectum; an address introductory to a discussion on the subject, Surg. 99
—— —— —— —— —— —— —— —— —— (reply), Surg. 131
——, statistics of permanent cure of rectal cancer after operation, quoted, Surg. 110
Effusion, diseased joints silent under auscultation in, Med. 37
Egg albumen unsatisfactory in rectal feeding, Therap. 65
Egg-yolks and beer recommended as diet for young children after weaning, Child. 123
Eggs, milk and pancreatic extract, absorption in rectal feeding, Therap. 64
——, peptonized, absorption in rectal feeding, Therap. 64

Egypt, health resorts of, suitable for neurasthenia, Baln. 42
Egyptian dog, hairless, Path. 298
Ehrlich-Hata's remedy, "606," convenient method of giving (intravenous route) (J. E. R. McDonagh), Derm. 36
—— —— —— —— —— —— —— ——, advantages, Derm. 38
—— —— —— in syphilitic aural diseases, Otol. 9, 12, 22, 23
—— —— —— in treatment of case of congenital syphilis (J. L. Bunch), Child. 158
—— —— —— —— of case of inherited syphilis (J. H. Sequeira), Derm. 10
—— —— ——, three cases illustrating benefit from (J. E. R. McDonagh), Derm. 5
von Eicken, treatment of acute frontal sinusitis with Brünings's light head bath, Laryng. 127, 128
Einhorn's intragastric electrode, Electr. 36
Elasticity (comparative) of cast and rolled gold plate (H. J. Morris and A. McWilliam), Odont. 57
—— —— —— —— ——, test for, Odont. 58
Elbow-joint, escape of synovial fluid from, in relation to ossific process, Surg. 140
——, fractures about, cases for operative treatment, Surg. 37
—— ——, in children, Surg. 6
—— ——, treatment, advantage of slinging wrist to neck with elbow acutely flexed, Surg. 23, 24, 29, 45, 46
—— —— —— by acute flexion and active movements, Surg. 29
—— —— —— by supination followed by flexion of joint, Surg. 7, 8
——, fracture-dislocation, old-standing, treatment by forcible flexion, Surg. 10
——, traumatic myositis ossificans in neighbourhood of, Surg. 153, 154
Electric changes accompanying contraction of muscle-strips, Med. 82, 83, 84
—— current, "make" and "break" of, with new induction coil apparatus, Electr. 80, 81, 82
—— effects in fibrillation, auricular and ventricular, Med. 93
Electricity and lavage in treatment of ulcer of stomach, Electr. 41, 42
—— (intragastric application of), preceded by massage in treatment of functional dyspepsia (obstinate third stage, with dilatation), method, Electr. 36-38
—— in treatment of abdominal diseases (H. Manders), Electr. 30-33
—— —— of functional dyspepsia (second and third stages), methods, Electr. 35, 36
—— produced, as well as distributed by the living cell, Electr. 93
—— (static), value of, Electr. 45
—— ——, with lavage in treatment of dilatation of stomach following operations or shock, Electr. 41
——, see also *Brush discharge* (static)
Electro- and actino-therapy, department of, at Guy's Hospital, Baln. 98
Electrocardiogram, definition of, Med. 84
—— of heart-beat, Med. 85
——, physiological, Med. 84
—— ——, method of obtaining, Med. 84, 85
——, showing auricular and ventricular representatives, Med. 86
—— —— fibrillation of auricle, Med. 92, 93
—— —— last stage of heart-block, Med. 91, 95
—— —— paroxysmal tachycardia, Med. 91
—— —— premature auricular contraction, Med. 87
Electrocardiograms showing premature ventricular contraction, Med. 89, 90
Electrocardiographic method (T. Lewis), Med. 81-94
—— ——, relationship to clinical medicine (T. Lewis), Med. 81, 95-99
Electro-Therapeutics, Section of (British Medical Association), founding of, Electr. 50
Electro-therapy, auto-condensation in, Electr. 104
—— ——, varied results of, Electr. 106, 107, 108
——, brain faradization, for insomnia, Electr. 102, 109
——, failure in, apparent, from persistence of symptoms, Electr. 103

Electro-therapy, failure in alcoholic cases due to want of probation and preparation, Electr. 103, 104
—— ——, due to ignorance of cause of disease treated, Electr. 101, 102
—— ——, due to need of longer treatment in disease of long-standing, Electr. 109
—— ——, due to non-treatment of spinal root of nerve in rheumatic neuritis, Electr. 104
—— ——, due to patient's unreadiness for treatment, Electr. 102-104
—— ——, due to want of repose in patient before and after application, Electr. 107
—— ——, due to wrong coil, Electr. 109, 110
—— ——, from certain gynæcological conditions, Electr. 111, 112
—— ——. in certain cases of melancholia, Electr. 110
—— ——, through failure to influence specific condition in certain diseases, Electr. 108, 109
——, regulation of dose, Electr. 107, 108
——, success in, from local applications in gastro-intestinal trouble, Electr. 108
—— —— case of bad spinal disease, Electr. 106
—— —— —— of severe gastric dilatation, Electr. 110
—— —— —— of spastic paralysis, Electr. 106
—— —— cases of melancholia, Electr. 110
—— —— —— of simple glycosuria, Electr. 108
—— ——, partial in cases of incurable disease, Electr. 104
—— —— or failure in, consideration of some of the causes (S. Sloan). Electr. 99-113
—— in gynæcology, communications on, summarized, Obst. 3
Electrode (intragastric, Einhorn's), Electr. 36
Elephant, example of hairless mammal, Path. 304
"Elephantiasis, congenital," of arm (C. T. Dent), Child. 24
—— of lip, case of (J. H. Sequeira), Derm. 56
Elham Rural District, case-rate of typhoid fever low in, Epid. 117, 123
Elimination, principle of spa treatment, Baln. 110
Elizabeth, Queen, prohibition of new buildings within walls of City of London by, Child. 115
Elizabethan period, feeding of infants in, Child. 111-116
Elliott-Smith, Professor, on appendiculo-ovarian ligament, Obst. 341
Elmslie, R. C.—Discussion on case of mollities ossium, Clin. 109
Embolism of central artery of retina in girl (S. Stephenson), Child. 56
Embryo, head of, position *in utero*, Odont. 107
Embryoma (ovarian), thyroid tissue in (R. Drummond Maxwell), Obst. 217
Embryotomy, indications for. Obst. 214
——, substitute for Cæsarean section, Obst. 165, 198, 199, 214
Emery, W. d'Este, blood-counts in cases of splenic anæmia after operation, Clin. 68
Emotions, complicated mental states, Neur. 5
——, hysterical, relate to past circumstances, Neur. 5
Emphysema, moderate, marine climate indicated for, Baln. 17
Empirical treatment, advances on, suggested by consideration of principles of vaccine therapy (C. Riviere), Therap. 43
—— —— of infective diseases, example of vaccine therapy (C. Riviere), Therap. 39
Empyema, hæmorrhagic, Surg. 49
—— of antrum in man, aged 71, becoming malignant (H. J. Davis), Laryng. 30
—— of maxillary antrum in boy (H. J. Davis), Laryng. 31
Enamel, structure of, Odont. 101
Encephalitis (chronic) ? case of (A. H. Miller), Child. 197
Enchondromata (multiple) and multiple cartilaginous exostoses, Med. 3
Endarteritis (syphilitic) in blood-vessels of mucous membrane of tympanic cavity, Otol. 18
Endemic conditions, influence upon epidemics, Epid. 192
Endocarditis (infective), *Staphylococcus albus* associated with, Obst. 194
——, malignant or infective, case of, treated by autogenous vaccine administered by the mouth (A. Latham and E. L. Hunt), Clin. 14-21

Endocarditis, malignant or infective, case of, treated by autogenous vaccine administered by mouth, bacteriological report, Clin. 17
Endognathion, Odont. 47
Endometritis, chronic, pathology discussed, Obst. 50, 51
—— ——, symptoms (A. Donald and W. F. Shaw), Obst. 37, 39
—— ——, treatment by curetting, results (A. Donald and W. F. Shaw), Obst. 41-46
—— —— —— —— —— in references to individual symptoms, Obst. 43-49
—— in young girls, connexion with adenoid vegetations in nasopharynx, Obst. 51
Endometrium, preservation in operations for uterine tumours, Obst. 118, 139, 143, 145
Endothelioma (congenital xanthoma), in infant, case of (J. E. R. McDonagh), Derm. 76
—— of gum (H. A. T. Fairbank), Odont. 12
—— of palate, with metastatic growth in neck, treated by radium (J. G. French), Laryng. 94
Endothrix trichophyton, violet, cultures of, from case of "eczematoid" ringworm of fungus associated with tineæ barbæ (H. G. Adamson), Derm. 126
Endotoxins, food-poisoning after eating canned meat due to, Epid. 66
Enema, nutrient, employed to test nitrogenous metabolism during rectal feeding, Therap. 67
Enghien, Baln. 18
England, East coast of, climatic advantages, Baln. 20
——, epidemics of poliomyelitis in 1909-10, Epid. 212-216
—— —— —— ——, map, Epid. 211
——, hydrotherapy in, date of first serious advocacy, Baln. 82
—— ——, empiric basis of, Baln. 99
—— ——, needs to be placed on scientific basis, Baln. 97
——, introduction of vapour baths into, Baln. 85
——, scopolamorphine prepared in, advantages of German production over, Anæsth. 54
—— (South coast of), climatic advantages, Baln. 20
—— (West of), first record of rickets as appearing in (seventeenth century), Child. 119
—— and Wales, typhoid fever in, decline in mortality, Epid. 25
Enteric fever, see *Typhoid fever*
Enteritis I, strains of bacteria composing, Path. 253, 254
—— II, strains of bacteria composing, Path. 253, 254
Epidemic constitution of air, Epid. 190
—— diseases, form of wave of, Epid. 195
—— ——, sequelæ of, varying nature of, Epid. 194
—— ——, virus of, lowered intensity again regaining virulent powers, Epid. 195
Epidemics, influence of endemic conditions upon, Epid. 192
—— of poliomyelitis, Epid. 200
——, relation of, to state of weather, Epid. 189
——, succession of, Epid. 194
——, varying fatality of, Epid. 192, 223
Epidermolysis bullosa, Path. 302
—— ——, case of (Willmott Evans), Derm. 34
Epigastric vein, right superficial varix of (L. McGavin), Clin. 51
Epiglottis, epithelioma of, case of (N. Patterson), Laryng. 83
Epilepsy complicated by acute mania, case of, successful method of treatment, Baln. 7
—— (minor), Stokes-Adams symptoms diagnosed from, Clin. 41
——, see also *Status epilepticus*
Epiphora, connexion of, with nasal disease or operation, Laryng. 168
Epiphyses, enlargement in achondroplasia, Child. 106
——, separation of, in fractures of hip-joint, Surg. 12
—— —— —— ——, diagnosis, Surg. 13
Epiphysitis, suppurative, of upper end of humerus, followed by chronic pyæmia of five years' duration (D. Drew), Child. 162
Episcleritis due to oral sepsis, Odont. 34
—— due to oral sepsis, treatment with mixed vaccines, Odont. 34

Epithelial origin of dental cysts, Odont. 15, 16
Epithelioma, apparent, in case of probable laryngeal tuberculosis (W. J. Horne), Laryng. 117
—— causing complete stenosis of larynx (StC. Thomson), Laryng. 119
—— gummatous ulceration of external ear mistaken for, Otol. 12, 19
—— involving whole of lower lip and entire skin of chin, showing close clinical resemblance to actinomycosis (A. W. Williams), Derm. 14
——, liability of warts and moles upon face to develop, Electr. 51
——, of back of left hand (T. Colcott Fox), Derm. 142
—— —— —— —— ——, treatment, Derm. 142
—— of epiglottis, case of (N. Patterson), Laryng. 83
—— of larynx, further notes on case of (P. Watson-Williams), Laryng. 153
—— of oral mucous membrane in relation to dental troubles, Odont. 78
—— on lupus vulgaris, cases of (H. G. Adamson, for C. E. Iredell), Derm. 139
—— —— —— —— (G. N. Meachen), Derm. 132
—— on right palato-glossal fold in man, aged 48 (W. Stuart-Low), Laryng. 60
—— originating near right Eustachian tube, with deafness and discomfort in right ear as early symptoms; fatal termination of (E. Law), Otol. 67
——, treatment by radium, Electr. 59
—— —— by X-rays, Electr. 58
—— —— ——, preliminary to operation, Electr. 58
Epithelium, chorionic, mode of action of (James Young), Obst. 294
Epizootics associated with *Bacillus enteritidis*, Epid. 61
Erasmus, on insanitary condition of London in sixteenth century, quoted, Child. 115, 116
Erepsin, digestive power of, Therap. 63
Ergot, action of, abnormal, Therap. 137-140
—— —— abortifacient, question of, Therap. 141
—— ——, extreme, Therap. 135-137
—— ——, method of investigating, Therap. 117, 118
—— ——, on arterial pressure, Therap. 146
—— ——, on urinary bladder, Therap. 145
—— ——, rapidity in, Therap. 124, 125
—— ——, when given at third or fourth month of pregnancy, Therap. 143-145
—— —— on uterus, Therap. 117-145
——, active principles of, in ernutin, Med. 70
——, chemistry and physiology of, Therap. 114-150
——, clinical experimental study, Therap. 114-150
——, comparison of action of infusion and extract of, Therap. 124, 126
——, effect on, of medium chloroform anæsthesia, Therap. 132, 133
—— —— of opium on ecbolic action of, Therap. 130-132
——, erysipelas following use of, Therap. 140
——, general effect of, on labour, Therap. 124
—— in child-birth, need of accurate clinical records, Therap. 4, 5
—— in pulmonary hæmorrhage, Therap. 146-148
——, indications for use of, Therap. 123
——, preparations and dosage, Therap. 116, 140
——, reaction of arteries to, Therap. 89
——, selection of patient for exhibition of, Therap. 117
——, so-called disadvantages of, Therap. 133-137
——, use in abortions and bleeding generally, Therap. 116, 141-145
—— —— in *post-partum* hæmorrhage, Therap. 141
—— useful in case of toneless uterus, Therap. 123, 137
Ergotin, reaction of arteries to, Therap. 89
Ergotoxin, alkaloid, in ernutin, Med. 70
——, differences in action of, from liquid extract or infusion, Therap. 125, 127, 149
——, rapidity of action of hypodermic injection of, Therap. 124, 125

Erichsen, Sir J. E., Bt., case of hypertrophy of gums, quoted, Odont. 22
——, hypertrophy of gums and alveolar processes, quoted, Odont. 23
Erlanger, experimental work on auricular ventricular bundle, Therap. 97, 98.
Ernutin, composition of, Med. 70
——, effect on low blood-pressure, Med. 70
Erosion cavity on lingual aspect of a lower incisor (C. F. B. Baly), Odont. 69
Eruption, generalized, probably mycosis fungoides (T. Colcott Fox), Derm. 20
—— (pustular) and extensive scarring following Scinde ulcers (Sir Malcolm Morris and S. E. Dore), Derm. 107
Erysipelas (facial, recurrent), indications for radical operation in chronic frontal sinus suppuration, Laryng. 132
—— following use of ergot, Therap. 140
—— of head: invasion of right auditory meatus, where abscess formed with extension backwards to sterno-mastoid muscle, and invasion of whole sheath of that muscle (A. H. Cheatle), Otol. 111
Erythema ab igne or livedo reticularis with pigmentation, case of (H. G. Adamson), Derm. 46
—— induratum, experimental production of tuberculosis by inoculation of material from, Derm. 49
—— in relation to dental trouble, Odont. 77
Erythism, nervous, treatment by subthermal baths, Baln. 96
Erythræmia, see *Polycythæmia* (splenomegalic)
Escherich, gangrene of extremities following infectious disease, quoted, Child. 11
Esdaile, production of surgical anæsthesia under hypnotism by, Anæsth. 13, 14
Ether, anæsthesia by, degree of, Anæsth. 7
—— ——, post-operative collapse following, Anæsth. 2, 3
—— —— (prolonged) alternating with nitrous oxide gas, after-effects, Anæsth. 8
—— —— —— alternating with nitrous oxide gas, effect of undue prolongation of gas, Anæsth. 5
—— —— —— —— —— ——, quantity of each anæsthetic employed, Anæsth. 5, 7
—— —— —— —— —— ——, reflex effects, Anæsth. 8
—— —— —— —— —— ——, time record for distribution of the two anæsthetics, Anæsth. 4
—— —— —— —— —— ——, variation in depth, Anæsth. 8, 9, 10, 11
—— —— —— —— —— —— and oxygen (A. L. Flemming), Anæsth. 1
—— —— —— —— —— —— ——, indications for changing from gas to ether, Anæsth. 4
—— —— —— —— —— —— ——, preliminary use of morphia and atropine, Anæsth. 3, 4
—— —— —— —— —— —— ——, re-breathing with gas and oxygen, Anæsth. 7
—— —— —— —— —— —— ——, suitable subjects for, Anæsth. 5
—— —— —— —— —— —— ——, technique, Anæsth. 3, 4
—— —— —— —— —— —— —— ——, variations in, Anæsth. 3
—— ——, reactionary shock more frequent after, than after chloroform, Anæsth. 2
—— ——, use of ethyl chloride during, Anæsth. 9
——, inhalation of, after hypodermic injection of scopolamorphine and atropine, after-effects, Anæsth. 50
—— —— —— —— —— —— ——, condition of patients during operation, Anæsth. 49
—— —— —— —— —— —— ——, method, Anæsth. 49
—— —— —— —— —— ——, morphine and atropine, narcotic effect before operation, Anæsth. 49
—— ——, preceded by hypodermic injection of atropine sulphate, Anæsth. 53
—— —— —— —— —— of morphine, Anæsth. 44
—— —— —— —— —— of scopolamorphine and atropine, Anæsth. 47
—— —— —— by oral administration of chloral hydrate, Anæsth. 44
——, intercurrent asphyxia under, as standard for asphyxia under chloroform, Path. 206, 217
——, open, administration to woman, results satisfactory, Anæsth. 51
—— toxæmia, method of avoidance under ether anæsthesia followed by nitrous oxide and oxygen, Anæsth. 3

Ether, see also *Nitrous oxide gas* and ether
Ethmoidal cells, disease of, connexion of, with retrobulbar neuritis, Laryng. 167, 168
—— disease, effect on vision, Laryng. 163, 164-166
—— operation, followed by paralysis of right third nerve (A. J. M. Wright), Laryng. 159
—— ——, results of orbital cellulitis following (A. J. M. Wright), Laryng. 159
Ethmoiditis (acute), complicating acute frontal sinusitis, Laryng. 128
—— (septic) treatment by Ogston-Luc operation, cases illustrating (W. Stuart-Low), Laryng. 126
Ethyl chloride, use during ether anæsthesia, Anæsth. 9
Eucalyptol, see *Menthol*, eucalyptol and terebene
Eucalyptus oil, inunction with, in treatment of measles, Child. 177, 183, 184
—— —— —— —— —— (R. Milne), Child. 142-154
—— —— —— —— of scarlet fever, Child. 142-148, 152, 153, 177
Europe (Continental), health resorts suitable for neurasthenia, Baln. 42
——, epidemics of poliomyelitis in, Epid. 200, 201
—— —— —— (map showing), Epid. 201
——, first lectures on balneology and climatology in, Baln. 115
——, use of vapour baths in, Baln. 85
Eustachian tube, foreign bodies in nasopharynx injuring mouth of, Otol. 86
—— ——, obstruction of, due to syphilitic nasal ulceration, Otol. 12, 13
—— —— —— in syphilitic pharyngitis, Otol. 19
—— —— and tympanum on left side, rhythmical clickings in, accompanying rhythmical elevations of soft palate, in syphilitic, Otol. 13
—— tubes, occlusion of, by scar-tissue, causing deafness (D. R. Paterson), Otol. 109
Evans, Arthur.—Subsequent report on a case of excision of the larynx, lower part of the pharynx, and upper end of the œsophagus for malignant disease (squamous-celled carcinoma) of those structures, Clin. 142
——, urgent dyspnœa and œdema of the glottis in a woman, aged 50, due to hæmorrhage into the thyroid, Clin. 25
——, and Pring, C. H.—Case of Graves's disease; its treatment, first by medical methods, and later by excision of one of the lobes and the isthmus of the thyroid gland, Clin. 122
Evans, Willmott.—Case of epidermolysis bullosa, Derm. 34
Eve, Sir F.—Case of traumatic myositis ossificans in neighbourhood of femur, quoted, Surg. 154
——, discussion on operative treatment of cancer of rectum, Surg. 110, 131
Ewald, experiments as to absorption in rectal feeding, Therap. 64
Ewald, P., cases of ossification of muscles, quoted, Surg. 133
——, escape of synovial fluid from knee- or elbow-joint as stimulus to ossific process in traumatic myositis ossificans, Surg. 140
Ewart, William.—Discussion on case of simple atrophic type of myopathy, Child. 102
——, "Dorsal percussion of the thorax and of the stomach," quoted, Med. 149
Ewers, C., action of aconite, quoted, Therap. 157
Exanthemata, sequelæ of, producing liability to pulmonary tuberculosis, Epid. 48
Excitement, effect on low blood-pressure, Med. 57
Exercise, effect on low blood-pressure, Med. 55
——, increased transmission of characters acquired by, Path. 280
Exhaustion as cause of neurasthenia, Baln. 44
Exognathion, Odont. 47
Exophthalmos in thyroid feeding of rats, Path. 235, 236
Exostoses, cartilaginous, multiple (A. M. Gossage and E. Rock Carling) Med. 1-22
—— —— ——, abnormal constitution of cartilage in, Med. 4
—— —— ——, ætiology discussed, Med. 3, 4
—— —— ——, characters, Med. 1
—— —— ——, complications arising from, Med. 2
—— —— ——, conditions associated with, Med. 3, 21

Exostoses, cartilaginous, multiple, congenital syphilis and, Med. 3
—— —— ——, details of cases, Med. 8-16
—— —— ——, families affected with, Med. 4, 5
—— —— ——, heredity in, Med. 4
—— —— —— ——, approximation to Mendel's law, Med. 6
—— —— ——, initiation in intra-uterine life, Med. 3
—— —— ——, method of removal, Med. 2
—— —— ——, multiple enchondromata and, Med. 3
—— —— ——, question of complete dominance in male, incomplete in female, Med. 7, 8
—— —— ——, rickets and, Med. 3
—— —— ——, sex-incidence, greater in males, Med. 3, 4
—— —— —— —— —— ——, explanation, Med. 7
—— —— ——, spontaneous necrosis of, Med. 2
—— —— ——, tendency to run upwards in limbs, Med. 21
—— in middle ear, case of (A. H. Cheatle), Otol. 56
—— (mandibular), somewhat symmetrical, shown by skull from barrow (W. de C. Prideaux), Odont. 39
—— (multiple), case of (C. Maynard Heath), Child. 106
—— or osteomata of os calcis as cause of painful heel, Med. 3
Expectorants (depressant) in bronchitis, Therap. 41
Explosions, damaging organ of hearing, Otol. 93
—— injuring membrana tympani, Otol. 84
Extremities, gangrene of, following infectious disease, Child. 11, 12
——, painful necrosis of, case (James Galloway), Clin. 26
——, recovery of sufferers from plague of Athens after loss of, Child. 13
Eye and orbit, affections of, in relation to diseases of the nose and accessory sinuses, Laryng. 161-168
——, diseases of, inflammatory, due to oral sepsis (C. Goulden), Odont. 27-36
—— —— —— —— —— ——, method of infection, Odont. 27
—— —— —— —— —— ——, question of direct infection, Odont. 32, 35, 36
——, foreign bodies in globe of, skiagraphy of, precautions necessary, Electr. 91
——, influence of adenoid vegetations on, Laryng. 164
——, peripheral field anomalies not reflex in origin, Laryng. 165
—— —— —— contraction, in sinusitis, Laryng. 163-166
——, routes of extension of disease from nasal to ophthalmic regions, Laryng. 162
——, suppuration, necrosis and orbital cellulitis due to septic processes of nasal cavities, Laryng. 162
Eye-colour, factors deciding, Med. 6
Eyes, condition of, in amaurotic family idiocy (Tay-Sachs disease), Path. 151-152, 157-169
Eyre, J. W. H., Malta fever, quoted, Epid. 75

Face, multiple tumours of (G. W. Dawson), Derm. 3
——, nerve-distribution of skin of, Odont. 72, 73
——, warts and moles upon, liability to develop epithelioma or rodent ulcer, Electr. 51
——, see also *Muscles*, facial
Fæces, *Bacillus typhosus* in, Epid. 4
——, *Bacillus paratyphosus* B not found in, Epid. 55
——, coliform organism freshly isolated from, fermentation reactions practically identical with those of *Bacillus Grünthal*, Path. 105
—— (diarrhœal), presence of *Bacillus typhosus* (type Flexner) in, Epid. 126, 137, 145
——, excretion of, before and after taking sulphur water, Baln. 49
—— of typhoid carriers, bacteriological examination, Epid. 118
—— —— —— —— ——, frequency of, Epid. 14
—— (putrefying), with dilatation of colon associated with chronic (recurrent) tetany, Child. 17
Fagge, C. Hilton, contact infection in typhoid fever, quoted, Epid. 130

Fagge, C. H.—Discussion on fractures in neigbourhood of joints, Surg. 34
——, discussion on operative treatment of cancer of rectum, Surg. 128
—— —— on treatment of cleft palate, Surg. 181
——, inguinal method of radical cure for femoral herniæ, Surg. 165-168
Fahnestock, "statuvolism," quoted, Anæsth. 19
Faier, Thomas, on infant-feeding, quoted, Child. 112
——, on wet-nurses, quoted, Child. 113, 114
——, "The Regiment of Life" (third section "The Booke of Children"), 1567, Child. 110
Fairbank, H. A. T.—Case of endothelioma of the gum, Odont. 12
——, cyst of the jaw, the result of traumatism, Odont. 14-17
——, malformations of ribs in Sprengel's deformity, Electr. 70
Fallopian tube, pregnant, changes in, Obst. 295-298
—— ——, diseases of, communications (1909-10) (summarized), Obst. 16
—— ——, torsion of, in case of hæmatosalpinx; no tubal pregnancy (Miss A. L. McIlroy), Obst. 112
—— ——, pathological specimens exhibited (1909-10) (summarized), Obst. 16
Fallopian tubes, enlargement in tuberculous peritonitis, Child. 226
Families affected with multiple cartilaginous exostoses, Med. 4
Family nervous disease: case for diagnosis (C. A. Basker), Child. 198
—— predisposition to pulmonary tuberculosis, Epid. 37, 47, 48
—— —— —— —— and family infection, Epid. 49
—— —— —— ——, influence of, Epid. 37, 44, 45
—— prevalence of amaurotic idiocy (Tay-Sachs disease), Path. 147, 148, 149, 167, 168
—— —— —— —— —— —— sometimes absent, Path. 168
Faradism, intragastric (Borri's method), in treatment of vomiting of gastritis, Electr. 41
Faradization (of brain) in electro-therapy, Electr. 102, 109
Farado-massage, contra-indications for, Electr. 40
—— in extrusion of gall-stones, method, Electr. 42, 43
—— in treatment of gastric dilatation, Electr. 40
—— —— of obstinate dyspepsia, method of application, Electr. 38-40
——, restoration of gastric secretions by, Electr. 38, 39
Farr, W., progressive attenuation of epidemic virus, noted by, Epid. 195
Fats, absorption in rectal feeding, Therap. 66
—— —— —— ——, absent in case of chyluria, Therap. 66, 67
—— —— —— ——, in case of fistula of thoracic duct, Therap. 66
Fauces, posterior pillar of, recurrent pedunculated fibroma of (A. Bronner), Laryng. 33
——, tertiary syphilitic ulceration of, accompanying congenital syphilitic deafness, Otol. 8
Faulder, T. J.—Case for diagnosis and suggestions for treatment, Laryng. 14
——, case of aphonia, Laryng. 42
Fawcett, Professor, on appendiculo-ovarian ligament, Obst. 344
Fawcett, J.—Cystic disease of kidneys, Clin. 151
——, discussion on absence of abdominal respiratory movements in pericarditis, Med. 48
Fearnsides, E. G.—Case of syringomyelia, with symptoms precipitated by trauma (for Dr. Henry Head), Neur. 34
——, functional hysterical trophœdema, Derm. 84
Fedden, W. Fedde.—Discussion on case of benign cyst of tibia, Clin. 139
Feeble-mindedness associated with excessive hypertrophy of gums and alveolar processes, Odont. 23
Feeding (infant), history of, from Elizabethan times (D. Forsyth), Child, 110-141
—— ——, see also *Infants*, feeding of
Feeding-bottle, cow-horn earliest form of, Child. 131
——, early form of, dangers of, Child. 131, 132
——, glass, early form of, Child. 131
Felixstowe, health resort suitable for foreign invalids, Baln. 20
Female, incomplete dominance of condition of multiple cartilaginous exostoses in, discussed, Med. 7, 8, 22

Females, apparent excess of typhoid fever carriers among, Epid. 11, 12
——, preponderance of congenital syphilitic deafness among, Otol. 4
Femur, deformity (congenital) of, absence of upper epiphysis and of half shaft of left femur (Frank Kidd), Clin. 158
——, fracture of, cases for operative treatment, Surg. 38, 41, 42
—— ——, reasons for operative treatment, Surg. 32, 33
—— —— of lower end, complicated by condyloid separation, Surg. 16
—— —— —— ——, treatment, Surg. 16
—— —— —— —— —— by traction in line of limb, Surg. 16
——, fractures of neck of, Surg. 14
—— —— ——, diagnosis, Surg. 13
—— —— ——, impacted, Surg. 14
—— —— —— ——, ability to raise limb from bed, Surg. 14
—— —— —— ——, question of disimpaction, Surg. 14
—— —— ——, treatment, Surg. 14-16
—— —— —— —— by breaking down adhesions, Surg. 15, 16
—— —— —— —— by fixation of limb with Thomas's abduction frame, Surg. 14, 15
—— —— —— ——, routine of massage; Surg. 15
—— (left), purulent infiltration into cancellous tissue of neck of, Child. 203
——, malformation, case of (O. L. Addison), Child. 164
——, sarcoma of, case of (O. L. Addison), Child. 40
——, shaft of, surrounded by subperiosteal infiltrate in case of atypical chloroma, Path. 63
——, syphilitic osteitis of, case of (P. Maynard Heath), Child. 103
—— and tibia from case of infantile scurvy, Child. 204
——, traumatic myositis ossificans in neighbourhood of, Surg. 144-153
——, see also *Thigh*
Fenestræ shown in two specimens of quadrilateral cartilage (nasal septum); removed by submucous section (D. McKenzie), Laryng. 5
FENTON, W. J., and GALLOWAY, J.—Stokes-Adams symptoms in a lad, aged 18, Clin. 39
Fenwick, S., on secretion of sulphocyanate in saliva, quoted, Baln. 57
Fergusson, W., "The mercurial plan of treatment in dysentery," quoted, Med. 148
——, "Yellow fever and the remitting fevers of Europe and the East and West Indies," quoted, Med. 148
Fermentability of foodstuffs in relation to production of dental caries, Odont. 91
Fermentation, mixture of saliva with food in relation to, Odont. 91, 92
—— tests in differentiation of bacilli, Epid. 70
Ferments, constituents of, Baln. 60
Ferrannini's method of using positive galvanic pole in stomach as treatment for subacute or chronic gastritis with vomiting, Electr. 41
Ferreyrolles, Dr., method of giving arsenical waters of La Bourboule, Baln. 108
Fever, treatment among North American Indians (1683), Baln. 89
Fevers, treatment by cold baths, first advocacy of, Baln. 83
—— —— —— ——, rules of Giannini for, Baln. 88, 89
Fever hospitals, nurses in, contact-infection with typhoid fever. Epid. 128, 129, 139
Fibrillation, electric effects in, Med. 93
—— —— ——, auricular, Med. 93
—— —— ——, ventricular, Med. 93
Fibroid tumour of uterus complicating pregnancy, after-history of case (A. H. N. Lewers), Obst. 307
—— —— ——, intramural, myomectomy, case referred to, Obst. 12
—— —— superimposed on gravid uterus which retroflexed (C. E. Purslow), Obst. 289
Fibroid tumours complicating labour, procedure in, Obst. 190, 191
—— complicating operation of Cæsarean section, Obst. 204
—— of uterus, treatment by panhysterectomy, Obst. 116
Fibroma, diffuse, of ovary which has undergone central degeneration with extravasation of blood (R. H. Paramore), Obst. 248

Fibroma, diffuse, of ovary, with twisted pedicle (M. A. Scharlieb), Obst. 35
——, pedunculated, recurrent, of posterior pillar of fauces (A. Bronner), Laryng. 33
Fibromatosis of stomach, Path. 240
Fibromatous tumours in man, aged 55 (G. W. Dawson), Derm. 101
Fibromyoma of uterus causing acute intestinal obstruction (J. Barris), Obst. 108
—— ——, treatment by subtotal hysterectomy: forty additional cases (Alban Doran), Obst. 115
Fibromyomatous polypus of uterus in girl, aged 17 (G. Blacker), Obst. 63
Fibrositis essential feature of muscular rheumatism, Baln. 29
——, low blood-pressure in subjects of, Med. 73
Films from cervix uteri, examination to determine nature of micro-organisms, Obst. 171, 180, 206, 207
Finchley, outbreaks of milk-borne sore throat at, Epid. 90, 94
Finger, keratosis of, case of (A. W. Williams), Derm. 32
Fingers, clubbing of, in congenital heart disease, Child. 26
——, contraction of, unusual form of (H. Lewis Jones), Electr. 73
——, nodes on, see *Heberden's nodes*
——, osteo-arthritis of, lateral deviation in, Baln. 32
FINZI, N. S.—Case of malignant stricture of œsophagus, Laryng. 153
——, discussion on case of benign cyst of tibia, Clin. 139
—— —— on case of malignant disease of pharynx under treatment by radium, Laryng. 64
—— —— on treatment of cancer by X-rays and radium, Electr. 61, 69
——, œsophagoscopy of malignant growth and application of radium, demonstration, Laryng. 151
—— and WALTERS, C. F.—Tumour of the cheek, Laryng. 160
Fire, exposure to, see *Erythema* ab igne
FIRTH, A. C. D., and LEDINGHAM, J. C. G.—Atypical chloroma, Path. 60-69
Fish (blind), instance of, discussed, Path. 308
—— ——, provided with luminous or phosphorescent organs, Path. 308
—— (fried), outbreaks of typhoid fever due to, Epid. 134, 135, 141, 144, 145
Fistula, formation of, indication for radical operation in chronic frontal sinus suppuration, Laryng. 132
—— of ampulla of superior semicircular canal on left side; bilateral radical mastoid operation; instrumental exploration of left vestibule; total vestibular ablation; reactions (left side); cochlea unaffected (Sydney Scott), Otol. 73
—— (utero-abdominal) in contracted pelvis and protracted labour (septic cases), Obst. 163, 165, 173, 201
Fistulæ (recto-vaginal), repair of, methods described, Obst. 333-335, 336
Fistulous opening (large) into anterior wall of left maxillary antrum (J. Donelan), Laryng. 18
FITZWILLIAMS, DUNCAN C. L.—Case of congenital absence of the pectoral muscles, Child. 79
——, discussion on congenital dilatation of colon, Child. 77
Flack, auricular-ventricular bundle, quoted, Therap. 97
Flap method (Arbuthnot Lane's) in operating for cleft palate, Surg. 171, 173, 175, 176, 180, 184, 191-196, 197
Fleisch, relations between othæmatoma, syphilis and insanity, Otol. 104, 106
Fleming, A., "An inquiry into the physiological and medicinal properties of the aconitum napellus," Therap. 153
FLEMMING, A. L.—Discussion on nasal reflex during anæsthesia, Anæsth. 29
——, prolonged anæsthesia maintained by ether and gas alternately, Anæsth. 1-12
FLETCHER, H. MORLEY.—Case of infantilism with polyuria and chronic renal disease, Child. 95
——, case of infantilism with thyroid inadequacy (juvenile myxœdema), Child. 96
Flexion (acute) in treatment of fractures of collar-bone, upper arm and elbow, Surg. 23, 29, 45, 46
Flexner, experimental work on transmissibility of poliomyelitis, Epid. 199

Florence, baths at Spedale Santa Maria Nuova in, Baln. 89
Flour, foreign, transmission of poliomyelitis by, Epid. 216
——, steel-milled and stone-milled, comparative amounts of acid produced from mastication of bread made from, Odont. 91, 102, 103
Floyer, Sir John, "An inquiry into the right use and abuse of the hot, cold and temperate baths in England" (1697), Baln. 82, 103
Flügge, inhalation theory in phthisis, Child. 228
Fluids, localization of effects of diathermic currents in, Electr. 2, 3
FOERSTER, Professor O.—Resection of the posterior spinal nerve-roots in the treatment of gastric crises and spastic paralysis, Surg. 226-246
Fœtal life, saving of, how effected, Obst. 216
Fœtus, head of, pressure of pelvic bones on, alleged cause of facial asymmetry, Odont. 107
——, pelvic colon, rectum and anus of, Surg. 89-97
—— with congenital absence of sacrum (C. White), Obst. 279
—— at term, uterus of, cervical glands in (Herbert R. Spencer), Obst. 281
Folin's colorimetric process, estimation of creatinin by, Baln. 47
Folkestone, calcareous (hard) drinking water of, Baln. 119, 120
——, climate of, effects, Baln. 125, 126
——, prevalence of goitre at, Baln. 122
—— ——, typhoid fever (1893-1909), due to same carrier through milk distribution, Epid. 16, 18
—— —— —— —— —— —— —— —— —— ——, probability discussed, Epid. 115-118, 137, 138, 145, 146
——, water supply, analysis, Baln. 127, 128
Folds of Houston, Surg. 91, 92
Folliculitis decalvans, case of (E. G. Graham Little), Derm. 144
Foltz, Hans, barber and bath-keeper, Baln. 76
Food, articles of, calcium salts in, Baln. 123
—— (artificial), method of accustoming children to, after weaning, Child. 119
——, mixture of saliva with, in relation to fermentation, Odont. 91
——, presence of *Bacillus suipestifer* in, Epid. 58, 59
—— (solid), kinds allowed after weaning, in seventeenth and eighteenth centuries, Child. 119, 120
Food-bolting, influence on intermitting albuminuria, Med. 114
Food-poisoning, ætiology (F. A. Bainbridge), Epid. 58
—— after eating pork pies, at St. Anne's-on-Sea, Epid. 64
——, agency of private slaughterhouses in spreading, Epid. 69
——, bacteriology, Epid. 51, 52, 58
—— caused by bacilli, most closely connected with *Bacillus enteritidis* (Gaertner), Epid. 63
——, classification of strains of organisms from cases of, by complement-fixation method, Path. 253
—— due to consumption of meat infected with *Bacillus enteritidis* (Gaertner), Epid. 74
——, epidemiology (F. A. Bainbridge), Epid. 58
—— following eating of canned meats, toxins cause of, Epid. 65, 66
—— —— —— of pork pies at Derby, cause of, Epid. 65
——, outbreak at Aertryck, Epid. 53
—— —— at Astley Sanatorium following eating of canned meat, Epid. 66
—— —— at Wrexham, Epid. 65
—— ——, severe, following eating beef infected with Gaertner bacillus at Limerick, Epid. 68
——, relation of *Bacillus enteritidis* (Gaertner) to, Epid. 60, 61
—— —— of *Bacillus suipestifer* to, Epid. 59, 60
Foodstuffs, carbohydrate, initiation of caries at points in teeth favouring periodic stagnation of, Odont. 92
——, fermentability of, in relation to production of dental caries, Odont. 91
Foot, large cavernous nævus of (Sidney Boyd), Clin. 83

Foot, plantar area of, essential feature, Path. 310
Foot-and-mouth disease, transmission to man through cows' milk, Epid. 75
Foot-switch, double, for X-ray examination, Electr. 14, 26
FORBES, J. GRAHAM, and COATS, GEORGE.—Relation of *Meningococcus intracellularis* to pseudoglioma, Path. 242
Forearm, dislocation backwards, complicating fracture about elbow-joint, treatment by extension, supination, and acute flexion, Surg. 8
Foreign bodies in auditory meatus, Otol. 96
—— —— —— ——, injuries from introduction and attempted extraction, Otol. 81, 82
—— —— —— —— remaining without injury to ear, Otol. 82
—— —— causing œsophageal obstruction, Electr. 16
—— —— in ear, attempts to extract causing death, Otol. 97
—— —— in nasopharynx, Otol. 105
—— —— —— injuring mouth of Eustachian tube, Otol. 86
—— ——, removal of, from air-passages and œsophagus, specimens illustrating (H. Tilley), Clin. 152
—— ——, skiagraphy of, Electr. 91
Foreign countries, invalids from, advantages of British health resorts for, Baln. 11-22
Forestier, H., origin of "douche écossaise," explained by, Baln. 83
Formic acid produced by *Bacillus typhosus*, Path. 106
Fornet, proportion of typhoid fever carriers, quoted, Epid. 6
Forrest's needle, diathermy distinct from, Electr. 4
Forster and Kayser, isolation of *Bacillus paratyphosus* B from gall-bladder by, Epid. 56
FORSYTH, DAVID.—History of infant-feeding from Elizabethan times, Child. 110-141
Fossa (cranial, posterior), suppuration in, risk of sudden death in, Otol. 37
Fossil cetacea, periotic bones of, Otol. 114
Foster-mothers, Child. 112
Foulerton, A. G. R., and Bonney, V., micro-organisms causing puerperal infection, Obst. 167
Fowl recommended as food for young children after weaning, Child. 122, 123
—— (featherless), Path. 299
—— ——, thyroid gland of, Path. 301
FOWLER, F.—Discussion on treatment of cancer by X-rays and radium, Electr. 60
FOX, H. CLAYTON.—Discussion on case illustrating "dents de scie" of vocal cords, Laryng. 10
——, discussion on case of aphonia, Laryng. 16
—— —— —— of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 102
—— —— —— of papilloma of pharynx, Laryng. 62
—— —— —— of ulceration of left vocal cord, Laryng. 63
—— —— on equine larynx and "ventricle-stripping," Laryng. 91
—— —— on two specimens of quadrilateral cartilage (nasal septum) removed by submucous section, Laryng. 6
FOX, R. FORTESCUE.—Discussion on British health resorts for foreign invalids, Baln. 22
——, the Samuel Hyde memorial lectures: Outlines of medical hydrology—the science of water and of waters, with a plea for its practice and teaching in Britain. Lecture I: analysis and definitions, Baln. 63-80; Lecture II: modern hydrotherapeutics, a résumé and an appreciation, Baln. 81-100; Lecture III: the medicinal spring, the teaching of medical hydrology, Baln. 101-118
FOX, T. COLCOTT.—Acute pityriasis of the scalp, Derm. 140
——, case for diagnosis, Derm. 21, 34
——, discussion on case of angiokeratoma, Derm. 46
—— —— —— for diagnosis, Derm. 63
—— —— —— of granuloma annulare, Derm. 25
—— —— —— of non-ulcerating tuberculide (?) Boeck's miliary sarcoid, following lupus vulgaris, Derm. 68

FOX, T. COLCOTT.—Discussion on case of pemphigus foliaceus, Derm. 17
——, discussion on case showing keloids forming on cicatrices resulting from ulcerating syphilitic lesions, Derm. 75
—— —— —— of summer eruption, Derm. 4
—— —— on macular atrophy associated with lupus erythematosus, Derm. 144
—— —— —— —— in syphilitic patient, Derm. 125
—— —— on report on case for diagnosis (sarcoma cutis), Derm. 82
—— —— on small-spored ringworm of scalp, Derm. 67
——, epithelioma of the back of the hand of two years' duration, Derm. 142
——, generalized eruption, probably mycosis fungoides, in a man, aged 63, Derm. 20
——, ichthyosis in a child, aged 16 months, treated by thyroid extract, Derm. 72
——, macular atrophy in syphilis, quoted, Derm. 124
FOX, W.—Discussion on case of (?) granuloma inguinale, Derm. 137
—— and WARNER, F. A.—Case for diagnosis, Derm. 40
—— ——, report on case for diagnosis (sarcoma cutis), Derm. 81
Fractures about ankle-joint, treatment, Surg. 18-22
—— —— elbow-joint complicated by dislocations, treatment, Surg. 8
—— —— —— in children, treatment, Surg. 6
—— —— —— —— ——, difficulties in, Surg. 6
—— —— ——, treatment, Surg. 7, 8
—— —— —— —— by passive movements combined with massage at early stage injurious, Surg. 7
—— —— hip-joint, Surg. 12
—— —— ——, treatment, Surg. 14-16
—— —— knee-joint, treatment, Surg. 16
—— at ankle-joint, Surg. 4
—— (compound), of temporal bone, Otol. 101
——, diagnosis by X-rays, limitations, Surg. 1, 2, 31, 32
——, healed, of right temporal bone, showing line of fracture in meatus (A. H. Cheatle), Otol. 26
—— in neighbourhood of joints (R. Jones), Surg. 1-22, 44
—— —— ——, cases of epiphyseal injury, Surg. 30
—— —— ——, difficulty of after-treatment of hospital patients, Surg. 42
—— —— ——, disabilities accompanying, Surg. 3
—— —— —— indications for operative treatment, Surg. 34-36, 37, 38, 39-42
—— —— ——, passive movement condemned, Surg. 23, 27, 36, 37
—— —— —— —— ——, early and forced, deprecated, Surg. 22, 32, 43, 46
—— —— —— —— ——, method, Surg. 39, 40
—— —— ——, treatment, correct alignment in, Surg. 4
—— —— —— ——, restoration of function more important than appearance after, Surg. 3, 4, 9
—— —— —— —— by splints, Surg. 4
—— of and below olecranon, Surg. 10
—— of coronoid, Surg. 10
—— of femur (lower end), Surg. 16
—— of head of radius, Surg. 9
—— of humerus (anatomical neck), Surg. 4
—— —— (surgical neck), Surg. 4
—— —— (upper end), Surg. 5
—— of joints, replacement of bearing surfaces, Surg. 28
—— of osseous walls of auditory meatus, Otol. 82
—— of patella, transverse, without separation of the fragments (A. E. Johnson), Surg, 69
—— of petrous bone, Otol. 101
—— —— ——, aural complications, Otol. 89
—— of scaphoid, Surg. 3, 11

Fractures of skull, aural complications following, Otol. 88, 89
—— of temporal bone, specimens illustrating (G. J. Jenkins), Otol. 107
—— —— ——, types of, illustrated by fifteen specimens (A. H. Cheatle), Otol. 107
—— of upper arm, elbow and collar-bone, advantage of slinging wrist to neck with elbow acutely flexed, Surg. 23, 24, 29, 45
——, sesamoid bone fractured by direct violence (R. Morton), Electr. 96
——, treatment by calcium salts, Baln. 121
Fraenkel, involution cyst, quoted, Obst. 220
France, establishment and progress of hydrotherapy in, Baln. 87, 88, 97
——, sporotrichosis in, Derm. 114
——, teaching of medical hydrology in, Baln. 113
Frank, case of division of posterior spinal nerve-roots for gastric crises of tabes, Surg. 213
Frankenhäuser, Dr., lecturer on balneology at Berlin, Baln. 114, 115
——, re-hydration of skin by mineral baths, quoted, Baln. 16
——, sharp division of hydrotherapy and balneology in Germany, quoted, Baln. 78
FRENCH, HERBERT.—Discussion on case of spondylitis deformans, Clin. 30
——, discussion on use of belladonna in dyspepsia, Med. 32
—— —— on vaccine treatment of endocarditis, Clin. 20
——, pulsatile swelling in the third and fourth left intercostal spaces (? thoracic aneurysm), Clin. 22
FRENCH, J. GAY.—Discussion on origin of cancer in larynx, Laryng. 24
——, endothelioma of palate, with metastatic growth in neck, treated by radium, Laryng. 94
——, stricture of lower end of œsophagus (spasmodic ?), Laryng. 36
Freud, defence neuroses, quoted, Neur. 12
——, sexual memories as the cause of hysteria, quoted, Neur. 15
—— and Breuer, views on hysteria, quoted, Neur. 10
Friction sounds, position in rheumatic pericarditis, Med. 48
Fritsch's transverse method of fundal incision into uterus, Obst. 178
Froné, injection of chloral before chloroform, quoted, Anæsth. 44
Frontal and left temporo-sphenoidal lobes, abscesses in, in case of chronic suppurative mastoiditis (P. Watson-Williams), Otol. 134
—— region, right, and orbit, affected by morphœa (James Galloway), Clin. 27
—— sinus, disease of, effect on vision, Laryng. 163, 164-166
—— —— operations, osteo-plastic, radical, patients illustrating results of (P. Watson-Williams), Laryng. 154-157
—— ——, skiagraphy of, Electr. 90
—— —— suppuration, acute, following influenza, Laryng. 138
—— —— ——, chronic, complicated by meningitis, Laryng. 141
—— —— —— ——, condition of fronto-nasal canal in, Laryng. 131, 144
—— —— —— ——, packing cavity with aseptic wax after operation, Laryng. 146, 148
—— —— —— ——, treatment by autogenous vaccines, Laryng. 130, 138, 139
—— —— —— —— ——, intranasal, by lavage and local applications, Laryng. 129, 142, 144
—— —— —— —— ——, operative, Laryng. 129
—— —— —— —— —— ——, intranasal, Laryng. 130, 134, 139, 146, 147
—— —— —— —— —— —— ——, radical, by Delsaux's, Riedel's and Kuhnt's methods, Laryng. 134, 143
—— —— —— —— —— —— —— ——, by Killian's method, Laryng. 123, 134-138, 140, 143, 146, 147
—— —— —— —— —— —— —— ——, by Ogston-Luc method, Laryng. 134, 144, 146, 147, 148
—— —— —— —— —— —— —— ——, by Watson-Williams's osteo-plastic method, Laryng. 134, 135, 136, 137, 138, 148
—— —— —— —— —— —— —— ——, choice of method, Laryng. 134, 138
—— —— —— —— —— —— —— ——, complicated by supra-orbital neuralgia, Laryng. 139, 143
—— —— —— —— —— —— ——, results, Laryng. 132, 133, 134, 141
—— —— —— —— —— —— —— ——, influenced by variation in size of sinus, Laryng. 132, 133

Frontal sinus suppuration, chronic, treatment, operative, results influenced by variation in virulence of infecting organism, Laryng. 132

—— —— —— —— —— —— —— indications for, Laryng. 132, 144

—— —— —— —— —— —— ——, perfection in, cannot always be expected, Laryng. 133

—— —— —— —— —— ——, risks, Laryng. 141

—— —— ——, treatment by injection of mentholized liquid vaseline, Laryng. 147

—— —— —— ——, intranasal, by lavage and local applications, value of, Laryng. 130, 138

—— —— —— ——, operative, complicated by broncho-pneumonia, Laryng. 146

—— —— —— —— ——, method of avoiding infection after, Laryng. 144

—— —— —— —— ——, preceded by operation on all other involved sinuses, Laryng. 129, 134

—— —— —— —— ——, radical, complicated by osteomyelitis, Laryng. 134, 135, 139, 145, 146, 147

—— ——, variation in size as influencing result of operation, Laryng. 132, 133

—— sinusitis, acute, relief of pain in, Laryng. 127

—— ——, œdema of conjunctiva from (A. J. M. Wright), Laryng. 160

—— —— ——, treatment by aspiration with Sondermann's suction apparatus, Laryng. 146

—— —— —— —— by Brünings's light head bath, Laryng. 127, 128, 143

—— —— —— —— by counter-irritation, Laryng. 127, 128

—— —— —— —— by diaphoretics, Laryng. 127, 128

—— —— —— —— by plugging, Laryng. 142

—— —— —— —— —— with adrenalin and cocaine solutions, Laryng. 147

—— —— —— ——, intranasal, Laryng. 127, 128

—— —— —— ——, operative, conditions necessitating, Laryng. 128, 129, 147

—— —— —— —— ——, with preservation of mucous membrane, Laryng. 128

—— ——, œdema of conjunctiva following, Laryng. 160

Fronto-nasal canal more often patent than obstructed in chronic suppuration of frontal sinus, Laryng. 131, 144

—— ——, passage of drainage-tube through, to avoid infection after operation, Laryng. 144

Frosch, period of excretion of typhoid bacillus by carriers, quoted, Epid. 4

——, proportion of typhoid carriers, quoted, Epid. 20

Fuchs, Professor, on effect of disease of ethmoidal cells on retrobulbar portion of optic nerve, Laryng. 168

Fulguration distinct from diathermy, Electr. 4, 10

Function, bodily, one particular, liable to be affected by hysterical paralysis, Neur. 7, 8

—— determining anatomical variation, essential doctrine of Lamarckism, Path. 305

Functions, bodily, disability through hysteria, Neur. 7

—— ——, nature of, Neur. 7

Fungus from case of sporotrichosis, Derm. 117

GABELL, D. P.—Discussion on comparative elasticity and internal structure of cast and rolled gold plate, Odont. 66

——, discussion on a dentigerous cyst with cellular contents, Odont. 89

—— —— on distribution of nerve-fibres of dental pulp, Odont. 53

—— —— on erosion cavity on lingual aspect of lower incisor, Odont. 70

—— —— on relation between teeth and certain diseases of skin and mucous membrane, Odont. 83

Gaehtgens, paratyphoid carriers, reported by, Epid. 56

Gaertner's bacillus, see *Bacillus enteritidis* (Gaertner)

Gag (Geffer's) with tongue-depressor, in operating for cleft palate, Surg. 174, 177

—— used in operation on cleft palate, Surg. 180

Gairdner, Sir W. T., case of polypoid thrombus, quoted, Child. 201

Gait, peculiar, in child (? functional ataxia) (J. A. Wright), Child. 191

Galabin, A. L., and Blacker, G. F., alternatives for Cæsarean section, quoted, Obst. 166

Gall-bladder, *Bacillus paratyphosus* B found in, Epid. 56

Gall-bladder, cancer of (primary), gall-stones as factor in causation of, Path. 93, 94
—— —— —— —— —— ——, question discussed, Path. 94, 95
—— —— ——, incidence at Guy's Hospital, Path. 91
—— —— —— —— in insane, Path. 91, 92
——, microscopical appearances at necropsy of cases of congenital obliteration of bile-ducts, Child. 45
——, relation of typhoid carrier condition to, Epid. 134, 141
——, tumour in right side with symptoms referable to, skiagram of (A. D. Reid), Electr. 74
Galloway, James.—Case of painful necrosis of extremities, Clin. 26
——, case of pulmonary stenosis (congenital?), Clin. 82
——, discussion on carcinomatous degeneration in a chronic dermatosis, Clin. 159
—— —— on case of actinomycosis of abdominal wall, Clin. 80
—— —— —— of congenital pulmonary stenosis, Clin. 95
—— —— —— —— xanthoma (endothelioma), Derm. 77
—— —— —— of cystic disease of kidneys, Clin. 151
—— —— —— of (?) granuloma inguinale, Derm. 135
—— —— —— of ichthyosis in child treated by thyroid extract, Electr. 72
—— —— —— of parakeratosis variegata (?), Derm. 31
—— —— —— of splenomegaly (? congenital family cholæmia), Clin. 130
—— —— —— of spondylitis deformans, Clin. 29
—— —— —— of sycosiform syphilide, Derm. 106
—— —— —— of symmetrical gangrene of limbs and nose, Clin. 80
—— —— on cases of familial pigmentary dermo-fibromatosis, Clin. 74
—— —— —— of Henoch's purpura, Clin. 50
—— —— on monilithrix in male child, aged 16 months, Derm. 71
—— —— on peculiar affection of ankles in lad, aged 18, Derm. 4
—— —— on report on case for diagnosis (sarcoma cutis), Derm. 82
—— —— on two cases of adenoma sebaceum in mother and son, Derm. 70
—— —— on vaccine treatment of endocarditis, Clin. 19
——, morphœa affecting right frontal region and orbit, Clin. 27
——, multiple cutaneous telangiectases of recent origin, Clin. 42
—— and Fenton, W. J.—Stokes-Adams symptoms in a lad, aged 18, Clin. 39
Gall-stones as factor in causation of primary malignant disease of gall-bladder and bile-ducts, Path. 93, 94
—— —— —— —— —— —— —— and biliary ducts, question discussed, Path. 94, 95
——, extrusion by farado-massage, Electr. 42-44
—— —— ——, with internal administration of cholelysin, Electr. 44
——, formation of, possibly concomitant with and dependent upon presence of malignant disease, Path. 95
—— ——, time needed for, Path. 95
—— incidence in the insane (J. P. Candler), Path. 87
—— —— —— greater than in the sane, Path. 88, 89
—— —— —— —— ——, explanations to account for, Path. 91
—— ——, increases at advancing age-periods, Path. 89, 90
—— ——, percentage in post-mortem examinations, Path. 88
——, relation of typhoid carrier condition to, Epid. 12, 19
Galvanism followed by induced current in treatment of severe functional dyspepsia, Electr. 36
—— in treatment of gastritis (subacute or chronic), Electr. 41
Galvano-cautery, removal by, of nodules from cords (indirect method), cases illustrating results (E. A. Peters), Laryng. 40
Galvanometer (string) confirms facts made known by polygraph, Med. 97
—— ——, description of, Med. 81-83
—— ——, diagnostic uses, Clin. 41
—— —— indicating electric contraction of muscle-strip, Med. 82, 83
—— ——, installation in hospitals and physiological laboratories a necessity, Med. 98

Galvanometer (string), value of, Med. 98
—— ——, working principles of, Med. 81-84
Gametes, generative cells, Med. 6
Gamgee, stab-wound of ear, case quoted, Otol. 82
Ganglion, sympathetic (inferior cervical), section of, from case of amaurotic idiocy (Tay-Sachs disease) (photomicrograph), Path. 186
Ganglion cell from case of amaurotic idiocy (plate) (Tay-Sachs disease), Path. 190
Gangrene of extremities following diphtheria, prophylaxis against, Child. 13
—— —— —— —— —— infectious disease, Child. 11, 12
—— —— —— —— —— —— ——, prognosis, Child. 13
—— of leg following diphtheria (J. D. Rolleston), Child. 10
—— (symmetrical) with extensive lupus erythematosus, (G. W. Dawson), Derm. 101
—— —— of both legs, fingers of both hands, and nose (C. C. Choyce), Clin. 77
—— of vagina, probably due to chemical effect of pink rubber ring pessary (A. E. Johnson), Obst. 323
Gant, statistics of permanent cure after operation for rectal cancer, quoted, Surg. 110
GARDNER, H. BELLAMY.—Discussion on anæsthesia in post-pharyngeal abscess, Anæsth. 36
——, discussion on nasal reflex during anæsthesia, Anæsth. 30
—— —— on use of drugs before inhalation anæsthesia, Anæsth. 53
GARDNER, T. F.—Air and water: is there anything new under the sun? presidential address, opening of session of Balneological and Climatological Section, Baln. 1-10
——, discussion on balneological treatment of neurasthenia, Baln. 44
Gargel in sheep, Surg. 48
Garrigou, F., colloidal metallic solutions in thermal waters, quoted, Baln. 104, 105
——, occupant of chair of medical hydrology at University of Toulouse, Baln. 113
GARROD, A. E., F.R.S.—Case of spondylitis deformans, Clin. 29
——, discussion on case of congenital obliteration of bile-ducts, Child. 50
—— —— —— of pulmonary stenosis, Clin. 95
—— —— —— of pulmonary stenosis (congenital ?), Clin. 83
—— —— —— of rheumatoid arthritis, Child. 37
—— —— —— of symmetrical lipomatosis, Clin. 6
—— —— on cases of adiposis dolorosa, Clin. 6
—— —— on pulsatile swelling in left intercostal space, Clin. 24
——, on auscultation of joints, Med. 35-39
——, suggestion as to estimation of calcium content of blood in congenital heart disease, quoted, Clin. 93
Garrow, Dr., epidemics of poliomyelitis at Maryport, Workington and Barrow-in-Furness, Epid. 212-215, 226
Gas, production of, by intestinal bacteria from alcohol entirely or mainly undiminished, Path. 102, 103, 105, 109
—— —— —— —— from sugars, or sugar-yielding bodies, abolished entirely or almost, Path. 102, 103, 105, 109
—— —— —— ——, summary and conclusions, Path. 109
—— —— —— ——, variability in (W. J. Penfold), Path. 97
—— —— in bacilli isolated from urine in various sugar-media, compared with production of acid, Path. 97, 98
—— —— in dextrose-peptone-water culture of *Bacillus coli*, diminished by benzoates, Path. 98
Gas-tube, anaerobic, growth of variant *Bacillus coli* in, Path. 107
Gases contained in waters, Baln. 108
Gaskell, W. H., F.R.S., necessity for presence of auriculo-ventricular bundle first maintained by, Therap. 97
Gaskoin, hairless mice, quoted, Path. 292, 293
Gastein, radio-activity of waters at, Baln. 108
Gastric crises in ? tabes, division of posterior roots (C. M. Hinds Howell), Neur. 48
—— crises, treatment by division of posterior spinal nerve-roots, results, Surg. 227, 228, 253

Gastric and intestinal hæmorrhages (probably of septic origin) after abdominal operations (J. Hutchinson), Surg. 58-64
—— juice, acidity of, constancy of, Med. 26
—— ——, secretion of, diminished by administration of atropine, Med. 23, 24
—— —— ——, during rectal feeding, Therap. 72
—— —— —— —— —— ——, neutralization of, Therap. 72
—— ——, mode of action of belladonna on, Med. 33, 34
—— secretion, restoration by gastric lavage, Electr. 38, 39
Gastritis (chronic), treatment by electricity, Electr. 41
—— (septic), case of (W. P. Herringham), Clin. 140
—— —— ——, bacteriological examination, Clin. 140
—— (subacute), treatment by electricity, Electr. 41
Gastro-duodenal lavage following ingestion of saline waters, Baln. 40
Gastro-enteritis caused by ingestion of milk from cows with mastitis, Epid. 76
—— in cows, Epid. 74
——, milk-borne outbreak of, Epid. 97, 98
——, outbreaks due to *Bacillus paratyphosus* B, Epid. 56, 57
Gastro-intestinal disease, success in, of local applications in electro-therapy, Electr. 108
Gastro-succorrhœa, how controlled electrically, Electr. 41
Gastroscopy, combined direct and indirect, Hill-Herschell method of, by means of new type of œsophago-gastroscope, demonstration on living subject (W. Hill), Laryng. 69
——, hypodermic injection of scopolamorphine, before inhalation anæsthesia for, Anæsth. 52
Gastrotoxic serum, experimental production of acute ulcer of stomach by, in cats, Path. 53, 54, 55
—— ——, method of production, Path, 54, 55
Gatschenburger, Dr., hydrotherapy and balneotherapy first taught at University of Würzburg by, Baln. 115
Gauthier, Professor V., lecturer on hydrology at University of Naples, Baln. 116
Gautier, A., on classes of medicinal springs, quoted, Baln. 106
Geffer's gag, with tongue-depressor, in operating for cleft palate, Surg. 174, 177
Geigel, Professor, lecturer on physical therapy at University of Würzburg, Baln. 115
GEMMELL, J. E.—Ovarian teratoma, Obst. 285
Gengou and Bordet, differentiation of *Bacillus typhosus* from *Bacillus coli* through complement fixation, by, Path. 251
Genital organs (female, diseases of), habitual constipation associated with, Obst. 328
—— —— —— —— ——, interfering with functions of rectum, Obst. 327
—— tract, anaerobic putrefactive bacteria infective in, Obst. 170
—— ——, micro-organisms saprophytic in, Obst. 170
Genito-mesenteric fold, Obst. 346
Geographical distribution of angiokeratoma, Derm. 45, 46
—— —— of epidemic poliomyelitis, Epid. 200-206
—— —— of paratyphoid fever A, Epid. 53, 54
Germ-cells, changes in fundamental origin of, variations due to, Path. 307
Germ-plasm, accessibility to body-cells, Path. 307
——, possible influence of climate on, Path. 307
Germain and Dwight, cases of oral sepsis causing thrombosis of cavernous sinus, quoted, Odont. 113, 116
Germany, clinics and institutes for hydrotherapy in, Baln. 86, 87, 97
——, establishment of hydrotherapy upon scientific basis in, Baln. 85, 97
——, mineral water baths of, re-hydration of skin effected by, Baln, 16
——, scopolamorphine prepared in, advantages over English production, Anæsth. 54
——, sharp division between hydrotherapy and balneology in, Baln. 78
—— (South-west), campaign of preventive measures against typhoid fever in (1903), Epid. 2, 10, 12, 20
——, teaching of medical hydrology in, Baln. 114

Germany, see also *Army* (German)

Gestation sac (interstitial) of three to four months' development removed unruptured with body of uterus by abdominal hysterectomy (A. H. N. Lewers), Obst. 154

Giannini, "Della natura delle febbri et del miglior metodo di curarle" (1805), Baln. 88

—— —— —— —— —— —— —— ——, rules for cold baths in fevers, Baln. 89

Gibson, G. A., blood examinations in case of supposed mediastinal pericarditis and case of splenomegalic polycythæmia by, Clin. 91

——, interference with action of diaphragm as indicating pericarditis, quoted, Med. 45, 46

Gibson, T., delimitation of rectum, quoted, Surg. 86

Giddiness, see *Vertigo*

Gigantism, disturbed functions of thyroid gland associated with, Odont. 26

—— (partial), multiple cartilaginous exostoses associated with, Med. 3

Gilberti, stomatitis complicated by necrosis of jaw, case ending fatally, Child. 31

Giles, A. E.—Cessation of menstruation after hysterectomy, quoted, Obst. 116, 117

——, discussion on hysterectomy followed by double phlebitis, Obst. 225

—— —— on ovarian tumour obstructing delivery, Obst. 256

—— —— on six cases of "utriculoplasty" for uterine hæmorrhage, Obst. 277

——, hydatid of the uterus, Obst. 245

Gilford, Hastings.—Case of asexual ateleiosis, Clin. 34

——, see also *Hastings Gilford* form of symptomatic infantilism

Gilgit valley, endemic goitre in, Child. 78, 79

Gill, Richard.—Anæsthesia in post-pharyngeal abscess, Anæsth. 34

——, discussion on anæsthesia by ether and gas alternately, Anæsth. 7

—— —— on asphyxia under chloroform, Path. 223

—— —— on nasal reflex during anæsthesia, Anæsth. 32

Gillivray, case of hypertrophy of gums and alveolar processes, quoted, Odont. 22

Gimbernat's ligament, Surg. 166, 167

—— ——, division of, in inguinal method of radical cure of femoral hernia, Surg. 167

Giraffe, callosities in, Path. 283

—— ——, capacity to acquire, alone transmitted, Path. 309

——, hair-mats in, Path. 283

Girdansky, method of viewing skiagrams stereoscopically, quoted, Electr. 92

Giunti, Professor L., lecturer on hydrology at Cagliari, Baln. 116

Glanders in horses identical with sporotrichosis, Derm. 115

Glands (axillary), cutaneous scent of, causing sexual excitation, Path. 287

—— (cervical) in uterus of fœtus at term (H. R. Spencer), Obst. 281

—— (cutaneous), function in antelope, Path. 286

—— (ductless), function of, disturbed; presumed connexion with excessive hypertrophy of gums and alveolar processes, Odont. 26

—— (enlarged) and post-pharyngeal tumour obstructing œsophagus, Electr. 22

—— ——, important early sign of human trypanosomiasis, Path. 13, 14, 18

—— found in vagina, Obst. 22

—— (gastric), acidogenic and pepsinogenic functions of, quasi-independence of, Med. 27

——, general enlargement of, in case of congenital syphilitic deafness, Otol. 8

—— (lymphatic), see *Lymphatic* glands

—— (mesenteric), see *Mesenteric* glands

—— of neck, see *Neck*, glands of

—— (scrofulous), treatment by calcium salts, Baln. 122

—— (tuberculous), disease of, marine climate indicated for, Baln. 17

Glandular areas, specialized in antelopes, Path. 310

—— fever, case of (W. H. Bowen), Child. 195

Glasgow, outbreak of milk-borne scarlet fever at, Epid. 87

Glasgow Maternity Hospital, results and technique of Cæsarean section at, Obst. 205, 211

Glasgow, see also *Belvidere Hospital*

Glénard's disease, see *Proptosis*, visceral

Glendining, B.—Cervical glands in the uterus of fœtus at term, Obst. 282
——, discussion on vaginal secretion in infants, Obst. 31
——, and Bonney, Victor.—Adenomatosis vaginæ: a hitherto undescribed condition, Obst. 18
Glia-proliferation, distribution of, in human trypanosomiasis, Path. 28, 29
Glinski, acute leukæmia with infiltrates, quoted, Path. 68
"Globus hystericus," Neur. 5
Glossina palpalis, carrier of human trypanosomiasis, Path. 7-12
—— ——, infectivity due to reservoir of infection, Path. 9, 10
—— ——, period of infectivity, Path. 7-10
Glossitis, chronic ? cause (P. Watson-Williams), Laryng. 158
—— in "Addisonian" anæmia, Clin. 38
Gluteal districts, hairless, in monkeys, Path. 310
Glycerine as mouth-wash during rectal feeding objectionable, Therap. 72
—— plugs in dilatation of cervix before operation, Obst. 312
Glycogen, no effect on low blood-pressure, Med. 70
Glycogoll, Path. 98
Glycosuria, simple, success of electrotherapy in, Electr. 108
Goadby, K. W.—Discussion on inflammatory eye conditions due to oral sepsis, Odont. 34
Goats, gastrotoxic serum obtained from, Path. 54, 55
——, inoculation with streptococcus from fatal case of acute lymphadenitis, Epid. 85
——, mastitis in, caused by inoculation of *Streptococcus mastitidis*, Epid. 80, 85
—— ——, not caused by inoculation of *Streptococcus anginosus*, Epid. 80, 85
Go-cart, early kind of, Child. 117
Godlee, R. J.—Discussion on fractures in the neighbourhood of joints, Surg. 43
——, discussion on inguinal method of radical cure for femoral hernia, Surg. 168
—— —— on large congenital diverticulum of bladder, Surg. 165
—— —— on operative treatment of cancer of rectum, Surg. 122, 131
—— —— on resection of posterior spinal nerve-roots, Surg. 247, 254
—— —— on septic hæmorrhages, Surg. 68
—— —— on transverse fractures of the patella, Surg. 84
——, myositis ossificans traumatica, Surg. 144-156
——, torus palatinus, quoted, Odont. 39
——, two unusual cases of thyroid tumour, Clin. 119
Goetzky, on lordotic type of adolescent albuminuria, Med. 126
Goitre caused by drinking melted snow water, Baln. 122
—— (endemic) in Chitral and Gilgit valleys, Child. 78, 79
—— (exophthalmic), association with rheumatoid arthritis, Child. 36
—— ——, connexion with menopause, Clin. 122, 124
—— —— in boy, commencing at age of 10 (W. Essex Wynter), Clin. 155
—— ——, sex-limited disease, Med. 20
—— ——, suggestion before operation for, Anæsth. 22, 23
—— ——, case treated by medical and afterwards surgical methods (C. H. Pring and A. Evans), Clin. 122
—— ——, treatment by calcium salts, Baln. 121, 122
—— —— ——, medical, Clin. 122, 124
——, prevalence at Folkestone, Baln. 122
Gold-miners, prevalence of phthisis among, how explained, Child. 218
Gold plate (cast), microscopical structure, Odont. 63, 65
—— —— —— and rolled, comparative elasticity and internal structure of (H. J. Morris and A. McWilliam), Odont. 57
—— —— —— ——, comparative methods discussed, Odont. 66, 67
—— —— —— ——, dynamic test employed to prove comparative strength of, Odont. 60
—— ——, properties of, important factor in determining, Odont. 67
—— —— (rolled), microscopical structure, Odont. 62
—— —— (soldered), microscopical structure, Odont. 64

Goldstein and Parhon, first case of amaurotic idiocy (Tay-Sachs disease) in Roumania observed, reported by, Path. 169
Goldthwaite, bony formations of infective origin in and around joints, quoted, Baln. 24
Golf at British health resorts, Baln. 14, 21
Gonococcal infections leading to bony formations in and around joints, Baln. 24
Gonococcus in puerperal infection, Obst. 169, 177
—— —— ——, dangers of, Obst. 169
Gonorrhœa causing iritis, Odont. 28, 29
——, presence of, in protracted labour and contracted pelvis contra-indication to classical Cæsarean section, Obst. 169
—— —— —— —— —— ——, indication for removal of whole uterus, Obst. 190
Gonorrhœal keratosis of hands and feet (A. Winkelried Williams), Derm. 12
GOODALL, E. W.—Discussion on typhoid carriers and contact infection, Epid. 139
GOODHART, Sir James F., Bt.—The after-history of cases of albuminuria occurring in adolescence, Med. 109-120, 144-146
——, scheme of hand-feeding for infants, Child. 138
Gordon, cases of amaurotic idiocy (Tay-Sachs disease), Path. 169
Gordon, T. E., adoption of inguinal method of radical cure of femoral hernia, quoted, Surg. 166
Gordon, W., phthisis morbidity in places exposed to strong, prevalent, rain-bearing winds, Baln. 17
GORE, W. RINGROSE.—The climatic and balneological treatment of neurasthenia, Baln. 37-44
GOSSAGE, A. M.—Adiposis dolorosa (?), Clin. 4
——, discussion on after-history of albuminuria in adolescence, Med. 141
—— —— on the electrocardiographic method, Med. 99
——, and CARLING, E. ROCK.—Multiple cartilaginous exostoses, Med. 1-22
Göttingen, University of, courses of lectures on medical hydrology given at, Baln. 114
Gottstein, instrument for patient's use after total laryngectomy, designed by, Laryng. 56
Gougerot and de Beurmann, on sporotrichosis, quoted, Derm. 114
Gouget, mortality from measles at Hôpital Claude-Bernard, Paris, Child. 174
GOULD, Sir A. PEARCE, K.C.V.O.—Discussion on case of multiple cutaneous telangiectases of recent origin, Clin. 45
——, discussion on case of septic gastritis, Clin. 140
—— —— —— of symmetrical lipomatosis, Clin. 5-6
—— —— —— of urgent dyspnœa and œdema of glottis, due to hæmorrhage into thyroid, Clin. 25
—— —— on cases of adiposis dolorosa, Clin. 5-6
—— —— on spondylitis and on rigidity of the spine, Clin. 150
—— —— on treatment of splenic anæmia by splenectomy, Clin. 69
GOULDEN, CHARLES.—Some inflammatory eye conditions due to oral sepsis, Odont. 27-36
Goullioud, cyst involution, quoted, Obst. 220
Gout, administration of hypoxanthin to patients suffering from, effects of, Baln. 59
——, ætiology discussed, Baln. 119, 120
——, atonic, treatment by subthermal baths, Baln. 97
——, carbonate of lime contained in mineral waters beneficial to, Baln. 123
——, chronic, sulphur treatment, Therap. 15, 16
——, connexion of testicle with (presumed), Baln. 36
——, effect of alcohol on, Med. 78
——, no immunity to, from drinking soft water, Baln. 124
——, output of uric acid in, Baln. 59
——, production of, due to climate rather than water, Baln. 119, 120
——, question of diagnosis by auscultation of joints, Med. 39
—— (recurrent), percentage of diuresis during taking of sulphur water, Baln. 49
——, relation to osteo-arthritis, Baln. 25
——, treatment by diathermic current, Electr. 7

Gout, see also *Dyspepsia* (gouty)
——, see also *Osteo-arthritis* (gouty)
Gow, W. J.—Discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 195
Graham, Dr., on artificial feeding of infants and children, quoted, Child. 136
GRAHAM, CECIL.—Bilateral tuberculous disease of tonsils with tuberculous laryngitis, Laryng. 67
——, chronic ulcer of nasal septum, Laryng. 56
——, party-wall pharyngeal cancer, Laryng. 55
GRAHAM, J. C. W.—Case of chronic cataract, Child. 197
——, case of hydrocephalus and buphthalmos, Child. 197
—— —— of hypermetropic astigmatism with macular changes, Child. 198
GRANT, J. DUNDAS.—Case of secondary syphilis of external ear, quoted, Otol. 10
——, discussion on case of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 102
—— —— —— of chronic laryngitis, Laryng. 109
—— —— —— for diagnosis, Laryng. 57
—— —— —— illustrating "dents de scie" of vocal cords, Laryng. 11
—— —— —— of party-wall pharyngeal cancer, Laryng. 56
—— —— —— of tonsillolith in left supra-tonsillar fossa, Laryng. 12
—— —— on diseases of nose and accessory sinuses in relation to affections of eye and orbit (written contribution), Laryng. 167
—— —— on empyema of maxillary antrum in boy, Laryng. 32
—— —— on equine larynx and "ventricle-stripping," Laryng. 91
—— —— on functional aphonia in a boy, aged 15, Laryng. 115
—— —— on laryngeal tuberculosis in a boy, aged 10, Laryng. 116
—— —— on lupus of larynx in woman, Laryng. 35
—— —— on stricture of lower end of œsophagus, Laryng. 37
—— —— on tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 111
—— —— on the treatment of frontal sinus discharge, Laryng. 146
Grantown, districts round, suitable for neurasthenia, Baln. 42
Granule cells ("Körnchenzellen"), immense numbers present throughout cortex of brain, in cases of amaurotic idiocy (Tay-Sachs disease), Path. 177, 190, 193
Granuloma annulare, case of (E. G. Graham Little), Derm. 24
—— ——, nomenclature, Derm. 24
——, inguinale, case of (A. Shillitoe and J. E. R. McDonagh) Derm. 133
—— ——, connexion with syphilis, Derm. 136
—— ——, ulcerating, case of, Derm. 135
Graves's disease, see *Goitre* (exophthalmic)
GRAY, A. A.—Discussion on periotic bones of fossil Cetacea, Otol. 114
—— —— on case of persistence of disease in large sinus tympani, Otol. 55
Great Britain, epidemics of poliomyelitis in, Epid. 209-218
—— ——, health resorts of, advantages for foreign invalids, Baln. 11-22; see also *Health resorts* (British)
—— —— —— ——, need of increase in amenities, Baln. 80
—— ——, mortality of towns supplied with hard water less than that of those supplied with soft, Baln. 123
—— ——, sporotrichosis recently introduced into, Derm. 113
—— ——, West coast of, climatic advantages, Baln. 20
Green tint in chloroma, partial distribution, Path. 67, 68
—— —— ——, striking feature, but not of first importance, Path. 68
GREENWOOD, M., jun.—Discussion on notification of disease, and on epidemiology of poliomyelitis, Epid. 224, 225
——, discussion on tuberculosis and its prevention, Epid. 44
—— —— on typhoid carriers and contact infection, Epid. 137

GREENWOOD, M., jun., and BULLOCH, W.—The problem of pulmonary tuberculosis considered from the standpoint of disposition, Epid. 147-177
——, and Lamb, Major, seasonal prevalence of plague, quoted, Epid. 225
Greifswald, University of, courses of lectures on medical hydrology given at, Baln. 114
Greig, case of typhoid carrier, Epid. 123
——, isolation of *Bacillus typhosus* A in India, Epid. 53
—— and Semple, proportion of typhoid carriers, Epid. 4, 15, 19-21, 119, 120, 121
Grey-Edwards, C., and Severn, W. D., outbreak of milk-borne sore throat in Anglesey, Epid. 91
Griffith, A. S., feeding experiments with tubercle bacilli, quoted, Child. 217
GRIFFITH, W. S. A.—Discussion on an ovarian tumour obstructing delivery, Obst. 259
——, discussion on case of adenomatosis vaginæ, Obst. 25
—— —— on indications for, and technique of, Cæsarean section and its alternatives, Obst. 203
—— —— on rupture of gravid uterus, Obst. 153
—— —— on six cases of "utriculoplasty" for uterine hæmorrhage, one of which was followed by pregnancy and labour, Obst. 277
—— —— on subtotal hysterectomy for fibromyoma uteri, Obst. 143
—— —— on vaginal secretion in infants, Obst. 31
Grinders, prevalence of phthisis among, how explained, Child. 218
Gross, S. D., rarity of hypertrophy of gums and alveolar processes, quoted, Odont. 22
—— —— —— —— —— ——, case quoted, Odont. 22
Group reaction, presence of, in differentiation of bacteria, Path. 252
Grouse Commission, Child. 218
Grouse disease due to nematode invasion associated with presence of *Bacillus coli* in intestines, Child. 218
GROVES, E. W. H.—A new operation for the radical treatment of flat pelvis, Obst. 264, 270
——, on the division of the posterior spinal nerve-roots for pain, Surg. 199-206
—— —— —— —— —— for spasm, Surg. 213-225
—— —— —— —— —— for visceral crises, Surg. 206-213
Growth, inordinate, influence of, on intermitting albuminuria, Med. 114
——, pedunculated, on elongated uvula (H. Tilley), Laryng. 107
Gruber, pressure that can be withstood by tympanic membrane, Otol. 94
"Grunting" in horses, Laryng. 91
Guéniot, emergence of thermal waters, Baln. 107
Guibert de Saint-Brieux, morphine injection before chloroform inhalation in parturition, Anæsth. 43
Guildford, outbreak of milk-borne sore throat at, Epid. 93
Guinea baboon, ischial callosities in, Path. 288, 289
Guinea-pigs, epizootic of, mutation of intestinal micro-organisms in, Epid. 124, 136
——, production of carrier-state in, Path. 70-72
Guisez, treatment of cancer with radium, quoted, Electr. 64
Guleke's method of resection of posterior spinal nerve-roots, Surg. 229
Gull, Sir W., advocacy of surface-therapeutics by, Baln. 99
Gullet, see *Œsophagus*
Gum, endothelioma of (H. A. T. Fairbank), Odont. 12
——, injection of adrenalin into, effect of, Therap. 60
Gumma, bursting of, during chloroform anæsthesia, causing death by inspiration of gummatous material (W. J. McCardie), Anæsth. 25
—— of brain, case of (C. A. Ballance), Clin. 47
—— of mediastinum, Electr. 21, 29, 30
Gummatous condition, diffuse, of knee, in case of congenital syphilitic deafness, Otol. 8
—— infiltration about knee-joint, Otol. 20
—— ulceration of external ear mistaken for epithelioma, Otol. 12, 19
Gums and alveolar processes of jaws, excessive hypertrophy, case of, successful surgical and prosthetic treatment (F. Sleep), Odont. 18-26
—— —— —— —— —— —— —— —— —— —— ——, value demonstrated by report of another case (F. Sleep), Odont. 25

Gums and alveolar processes of jaws, excessive hypertrophy, cases reported, account of, Odont. 22
—— —— —— —— ——, feeble-mindedness associated with, Odont. 23
—— —— —— —— ——, presumed connexion with disturbed functions of ductless glands, Odont. 26
Gundelach, radiator tubes, Electr. 63
Gun-drill, performance of, rehearsed by subject of hysteria formerly injured during, Neur. 5, 6
Gunshot injuries of auditory meatus uncommon, Otol. 82
—— —— to ear, Otol. 98, 99, 101, 102, 104
Gunstock deformity, treatment, Surg. 10
Gut, post-allantoic, Surg. 93, 94, 95, 97
GUTHRIE, L. G.—Congenital insufficiency of soft palate, Child. 23
——, discussion on case of cerebral athetotic diplegia, Child. 78
—— —— —— of congenital absence of pectoral muscles, Child. 79
—— —— —— of peculiar gait, Child. 191
—— —— —— of post diphtheritic paralysis and hemiplegia, Path. 196
—— —— —— of simple atrophic type of myopathy, Child. 102
—— —— on specimens from case of "delayed chloroform poisoning," Child. 83
——, juvenile tabes (case shown by Dr. W. Harris), Neur. 50
——, two cases of muscular atrophy of the peroneal type (Charcot-Marie, Tooth) in father and son, Clin. 126
——, and BATTEN, F. E.—Cases of hemiplegia on one side associated with optic nerve atrophy on other, Child. 196
Guy's Hospital, department of electro- and actino-therapy at, Baln. 93
—— ——, incidence of primary carcinoma of gall-bladder and biliary passages as ascertained at, Path. 91
—— ——, percentage incidence of gall-stones based upon post-mortem records of, Path. 88, 89, 90
Gynæcological results of electro-therapy, Electr. 111, 112
Gynæcology, electro-therapy in, communications on (summarized), Obst. 3
——, importance of rectum in (E. Stanmore Bishop), Obst. 327-336

Hackney, outbreak of milk-borne sore throat at, Epid. 91
Hæmangio-lymphadenoma, see *Lymphoma*
Hæmatemesis after abdominal operations, of grave prognosis, Surg. 62
Hæmatocele, communications on, summarized (1909-10), Obst. 13
Hæmatocolpos, three cases, report on (W. Blair Bell), Obst. 234
—— fluid, nature of, with report of three cases of hæmatocolpos (W. Blair Bell), Obst. 234
Hæmatoma developing in early stage of traumatic myositis ossificans, Surg. 133, 140
Hæmatosalpinx with torsion of the tubes; no tubal pregnancy (Miss A. L. McIlroy), Obst. 112
Hæmodynamics of circulation, anticipation of knowledge of (1811), Med. 151
Hæmodynamometer, observations on blood-pressure made with, Med. 76
Hæmoglobin, oxygen capacity of, Clin. 86
Hæmolysis, results as to, in complement-fixation experiments, Path. 258-262, 264-271, 273-275
Hæmolytic system in complement-fixation experiments, Path. 256
Hæmophilia, sex-limited disease, Med. 20
—— —— —— (alleged), Med 7
——, treatment by calcium salts, Baln. 121
—— and osteo-arthritis, Baln. 24
Hæmoptysis, danger of, in middle-aged subjects of phthisis at high altitudes, Med. 60, 76
——, use of ergot in cases of, Therap. 147, 148

Hæmorrhage after menopause, significance of, in diagnosis of cancer of uterus, Obst. 86, 105, 107
—— (capillary), treatment by calcium salts, Baln. 121
——, control of, during operation for rectal cancer, Surg. 111, 117
——, fatal, from internal carotid, in case of sloughing tonsil, Child. 204
—— from ear following fracture of petrous bone, Otol. 90
—— (interstitial) in appendicitis, Obst. 347
—— (*post-partum*) and use of ergot, Therap. 141
—— (pulmonary), action of ergot in, Therap. 146-148
——, use of ergot in, Therap. 116, 141-143
—— (uterine), six cases of "utriculoplasty" for, one followed by pregnancy and labour (V. Bonney), Obst. 270-278
Hæmorrhages, gastric and intestinal, after abdominal operation, non-septic cases, Surg. 62, 64, 66
—— —— —— (probably of septic origin), after abdominal operations (J. Hutchinson), Surg. 58-64
—— of septic origin, cases for treatment with an auto-vaccine, Surg. 56
—— —— ——, diseases complicated by, Surg. 49, 50
——, severe capillary and parenchymatous, of septic origin, septicæmia hæmorrhagica (W. G. Spencer), Surg. 48-57
—— (vascular) in human trypanosomiasis, Path. 31
Hæmorrhagic cellulitis, cases of, Surg. 50-56
Hæmorrhoids, anatomical factor in causation of, explained, Obst. 332, 333
——, diagnosis from other diseased conditions aided by rectal examination, Obst. 331, 332
Hahnemann, experiments with aconite, Therap. 152
Hair, growth of, imperfect, in scalp of child, aged 18 months (J. M. H. MacLeod), Derm. 54
——, specimens from aboriginal Indian girl (J. M. H. MacLeod), Derm. 149
Hairless areas in monkeys, Path. 309-311
Hairlessness, see *Atrichosis* (congenital)
Hair-mats, glandular, Path. 284
—— in antelopes, over carpus, Path. 284
—— in giraffe, Path. 283
Hair-pins impacted in vagina, probably causing suppurating ovarian cyst (H. J. Paterson), Obst. 259
Hajek, treatment of acute frontal sinusitis, quoted, Laryng. 127
Haldane, J. S., and Douglas, C. G., estimation of total oxygen capacity and total blood-volume in case of congenital pulmonary stenosis, Clin. 88, 89
—— and Lorrain Smith's carbon-monoxide method, Clin. 89
HALL, F. DE HAVILLAND.—Discussion on cases of Henoch's purpura, Clin. 49
——, discussion on treatment of splenic anæmia by splenectomy, Clin. 58
Hall, Marshall, mortality among dry-nursed children, quoted, Child. 129, 130
Hall and Thomas, case of division of posterior spinal nerve-roots for gastric crises of tabes, Surg. 212
HALL EDWARDS, J.—Discussion on diathermic treatment, Electr. 9
——, notes on the treatment of cancer by means of the X-rays and radium, Electr. 47-69
Halle, advocacy of intranasal operations in chronic frontal sinus suppuration, Laryng. 130
Halle, University of, courses of lectures on medical hydrology given at, Baln. 114
Halliburton, W. D., F.R.S., "Chemical Side of Nervous Activity," quoted, Anæsth. 2
HAMER, W. H.—Discussion on notification of disease and epidemiology of poliomyelitis, Epid. 223
——, discussion on paratyphoid fever and food-poisoning, Epid. 69
——, typhoid carriers and contact infection; some difficulties suggested by study of recent investigations carried out on "living lines," Epid. 105-146
Hamilton, paratyphoid carriers, quoted, Epid. 56
HAMPSON, W.—Case of achondroplasia (shown for E. Pritchard), Child. 1

Hampson's method of apparatus for production of undulatory current, Electr. 35
Hancocke, John, "Febrifugum magnum, or Common Water the Best Cure for Fevers," (1723), Baln. 82
Hand, palmar area of, essential feature, Path. 310
—— (left), epithelioma of back of (T. Colcott Fox), Derm. 142
Hand-feeding, modern development of, Child. 139
——, scheme of, Child. 138
Handley, W. Sampson.—Advocacy of widely extended operation for rectal cancer, Surg. 102
——, discussion on case of thyroid tumour, Clin. 125
—— —— on operative treatment of cancer of rectum, Surg. 118, 122, 131
—— —— on subsequent report on case of excision of larynx, lower part of pharynx, and upper end of œsophagus for malignant disease (squamous-celled carcinoma), Clin. 144
——, muci-carmine staining method to show spread of rectal cancer, Surg. 102
Hands, deformity of, two cases (C. W. Chapman), Clin. 159
Harbitz, Professor F., latent tuberculosis, quoted, Epid. 34
Hard's farinaceous food, Child. 136
Harden, chemical activities of *Bacillus coli*, quoted, Path. 106, 107
Hare-lip associated with cleft palate, Surg. 180, 181
—— —— —— ——, age for operation, Surg. 179, 180, 181
de la Harpe, Dr., lecturer on hydrology at University of Lausanne, Baln. 116
Harris, "De Morbis Acutis Infantum," quoted, Child. 116
Harris, W.—Discussion on case of congenital brain defect, with stamping gait and tendency to fall backward, Neur. 34
——, discussion on case of progressive spinal muscular atrophy of infants (Werdnig-Hoffmann), Clin. 116
—— —— —— of syringo-bulbia, Neur. 27
—— —— —— of syringomyelia, with symptoms precipitated by trauma, Neur. 39
—— —— on cases of spondylose rhizomélique, with progressive muscular contracture, Neur. 44
—— —— on diathermic treatment, Electr. 9
——, juvenile tabes (for Dr. Guthrie), Neur. 50
——, spondylitis, with progressive muscular contracture, Neur. 42
—— —— —— —— —— —— (? myositis fibrosa), Clin. 145
——, tic douloureux illustrating the anæsthesia produced by alcohol injection of the superior maxillary and supra-orbital nerves, Neur. 52
——, traumatic neuritis of inner cord (Klumpke-paralysis): prefixed type of plexus, Neur. 31
——, and Clayton-Greene, W. H.—The treatment of acute suppression of urine by incision and drainage of the kidneys, Clin. 161
Harrison, Captain L. W.—Discussion on cases illustrating benefit from Ehrlich-Hata's "606," Derm. 7
Harrison, Reginald, renal tension and its treatment by surgical means, quoted, Clin. 162
Harrison, Ross, specific autonomic inherited energy of neurone, quoted, Path. 175
Harrogate, baths at, effect on low blood-pressure, Med. 71, 72
——, elevation in relation to treatment of circulatory disorders at, Baln. 69
——, mineral waters at, Baln. 15
——, neurasthenia benefited at, Baln. 42
——, some pharmacological effects of the strong sulphur water of (D. Brown), Baln. 45-62
Hartmann, H., sex-incidence of appendicitis, quoted, Obst. 349
——, primary source of combined disease of appendix and adnexa, quoted, Obst. 355
Harvey, isolation of *Bacillus typhosus* A in India by, Epid. 53
Harvieston (near Tillicoultry, Clackmannanshire), epidemic of poliomyelitis at, Epid. 217
Hastings, Somerville.—Case of early myasthenia gravis with laryngeal symptoms, Laryng. 39
——, case of ulceration on the left tonsil, Laryng. 74
——, new form of burr for use in antral operation by nasal route, Laryng. 105

Hastings, health resort suitable for foreign invalids, Baln. 20
Hastings Gilford's form of symptomatic infantilism (H. Batty Shaw), Clin. 33
Haug, case of foreign body in ear, quoted, Otol. 96
HAWTHORNE, C. O.—Discussion on low blood-pressure, Med. 75
Hay, Professor, on decline of phthisis rate, quoted, Epid. 171
Hay, M., outbreak of milk-borne gastro-enteritis and diarrhœa at Aberdeen, Epid. 97
——, outbreaks of typhoid fever at Aberdeen, Epid. 110-114
Hay's pharyngoscope, improved form (H. Tilley), Laryng. 108
HAYNES, G. S.—Case of congenital heart disease (? patent ductus arteriosus) (shown for Dr. E. Lloyd Jones), Child. 191
—— case of congenital heart disease (pulmonary stenosis), Child. 191
—— —— of mutism, Child. 192
——, infantile spastic paraplegia (shown for Dr. Charles Searle), Child. 192
——, skull from a case of idiocy of the Mongolian type, Child. 204
Hazarihagh district (Bengal), thermal springs of, Baln. 117
HEAD, HENRY, F.R.S.—Association of areas of bodily surface with deeply seated organs, quoted, Baln. 93
——, case of syringomyelia with symptoms precipitated by trauma (shown by Dr. Fearnsides), Neur. 34
——, researches on hyperæsthesia, alluded to, Surg. 227
—— —— on surface therapeutics, alluded to, Baln. 99
Head, injuries to, symptoms following, Otol. 91
——, nerve distribution of skin of, Odont. 72, 73
—— (fœtal), pressure of pelvic bones on, alleged cause of facial asymmetry, Odont. 107
—— of embryo, position *in utero*, Odont. 107
Headache, persistence of, indication for radical operation in chronic frontal sinus suppuration, Laryng. 132, 141, 144
——, treatment by calcium salts, Baln. 121
Health resorts (British), advantages for foreign invalids, Baln. 11-22
—— ——, amusements at, deficiency of, Baln. 15, 21
—— ——, amenities of, improvements in, needed, Baln. 13, 80
—— ——, climate of, compared with climate of Continental, Baln. 15
—— ——, complementary, not rivals, to Continental, Baln. 12
—— ——, favourable to out-door exercise during summer months, Baln. 14
—— ——, international repute of deficient, Baln. 12, 13, 14
—— —— —— —— ——, requisite, Baln. 13
—— —— (inland), Baln. 20, 21
—— ——, limited knowledge of, possessed by Americans and Canadians, Baln. 21, 22
—— ——, open-air galleries instead of winter-gardens at, advocated, Baln. 15
—— —— (marine), class suitable for foreign invalids, Baln. 18
—— —— ——, climates, compared with that of German littoral, Baln. 18, 19
—— —— —— ——, compared with those of French coasts, Baln. 18
—— —— —— ——, variety in, Baln. 17
—— —— ——, winter stations as summer resorts for foreign invalids, Baln. 20
—— (Continental), complementary, not rivals, to British, Baln. 12
—— ——, unsuited for out-door exercise in summer months, Baln. 14
—— (French, marine), climates of, compared with those of coasts of Great Britain, Baln. 18
—— (German, marine), climates compared with those of British marine health resorts, Baln. 18, 19
—— (marine), residence or stay at, indications for, Baln. 17
——, residence or stay at, contra-indications for, Baln. 17
Hearing, organ of, damaged by loud noises and explosions, Otol. 93
Heart, action of atropine on, Anæsth. 54, 55
—— —— of diathermic current on, Electr. 6, 7

Heart, action increased at high altitudes, factor in causation of rise in low blood-pressure, Med. 59
——, dilatation of, differential diagnosis from pericardial effusion, Med. 44, 48
—— ——, low blood-pressure accompanying, Med. 73
——, disease of, action of aconite on pulse-rate in, cases showing, Therap. 158-162, 165-167
—— ——, congenital, case of (P. G. Lee), Child. 26
—— —— ——, clubbing of fingers in, Child. 26
—— —— ——, in infant, specimen showing, Child. 204
—— —— ——, orthostatic albuminuria in, Clin. 93, 95
—— —— —— ? patent ductus arteriosus (G. S. Haynes, for E. Lloyd Jones), Child. 191
—— —— —— (pulmonary stenosis) (G. S. Haynes), Child. 191
—— —— —— —— ——, with cerebral hemiplegia (J. A. Wright), Child. 190
—— —— ——, secondary polycythæmia in, Clin. 93
—— —— —— —— ——, cause of, Clin. 90, 91, 92
—— —— ——, with congenital malformation of external ear (F. P. Weber), Child. 161
—— —— ——, without murmur, and with family history of congenital cyanosis (F. P. Weber), Child. 159
—— ——, effect of rest on, Therap. 26, 35
—— ——, indications for use of cardiac tonics due to accurate clinical records, Therap. 7-12
—— ——, pulse-rate in, effect of atropine on, in cases under influence of digitalis (M. D. Silberberg), Therap. 192-210
—— —— (rheumatic), association of delirium cordis with, Med. 92, 96
—— —— ——, value of digitalis group of drugs, Therap. 26
——, long axis of, increased obliquity in aortic dilatation, Electr. 25, 27-29
——, malformation of, case (F. Langmead), Child. 39
—— ——, congenital case of, pathological specimen of heart from (T. R. Whipham), Child. 3
—— —— —— ——, with polycythæmia, Child. 3, 4
——, specimen showing infective polypoid thrombus in pulmonary artery and extending into right ventricle, mistaken for patent ductus artericsus (T. R. Whipham), Child. 199
Heart-beat, mammaiian, origin of, Med. 86, 87
——, normal, electrocardiogram of, Med. 85
—— of isolated auricles and ventricles, Therap. 97
—— of isolated ventricle, Therap. 97
Heart-block, Med. 94
——, stages of, Med. 94
—— ——, electrocardiogram showing, Med. 91, 95
Heart-failure during anæsthesia, Anæsth. 27, 28
—— from reflex cardiac inhibition, Path. 206-214
—— in acute disease, cause of, Baln. 93
—— on change from recumbent to sitting posture during chloroform anæsthesia, Anæsth. 29
——, spontaneous, in asphyxia under chloroform, Path. 215-217
Heat, *minus* applications of, Baln. 95
——, resistance of certain bacteria to, in canned meat, Epid. 66
——, second common factor in spa treatment, Baln. 72, 73
HEATH, CHARLES.—Discussion on three specimens of chronic middle-ear suppuration, Otol. 113
——, eight cases of mastoid operation, Otol. 125-134
Heath, Christopher, case of hypertrophy of gums and alveolar process, quoted, Odont. 22
HEATH, P. MAYNARD.—Case of traumatic myositis ossificans, quoted, Surg. 149, 156
——, deformity of the cervical spine, Child. 105
——, discussion on myositis ossificans traumatica, Surg. 156
——, multiple exostoses, Child. 106
——, syphilitic osteitis of the femur, Child. 103
Heberden, William, on virtues of cow-horn as sucking-bottle, quoted, Child. 131
Heberden's nodes, compensatory, not pathological, Baln. 32
—— ——, development where strain is greatest, Baln. 32

Heberden's nodes, occurrence of, Baln. 23, 28, 29, 32, 35
Hebosteotomy, substitute for Cæsarean section, Obst. 165
Hebrides (The), Baln. 18
Hecking, sequel to conservative Cæsarean section performed in woman with gonorrhœa, Obst. 169
Hedgehog (spineless), specimen of, Path. 296
Heel, painful, osteomata or exostoses, as cause of, Med. 3
Heidelberg University of, clinic for hydrotherapy at, Baln. 87
—— ——, courses of lectures on medical hydrology given at, Baln. 114
Hektoen and Perkins, case of sporotrichosis, quoted, Derm. 114
Heliopolis, health resort for neurasthenia, Baln. 42
Helium in medicinal waters, Baln. 108
Helix, ulceration of, in tertiary syphilis of external ear, Otol. 12
von Helmont, use of cold water in disease, Baln. 89
Helouan, health resort for neurasthenia, Baln. 42
Hemiatrophy (facial), multiple cartilaginous exostoses associated with, Med. 3
—— from sclerodermia, case of (P. C. Knapp), Neur. 28
Hemiplegia (cerebral) with pulmonary stenosis (congenital heart disease) (J. A. Wright), Child. 190
——, deafness in, Otol. 13
—— on one side associated with optic atrophy on other, Child. 196
—— and paralysis (post-diphtheritic), case of, Child. 195
—— (right), slowly progressive, suggesting tumour (operation—no tumour found) (F. E. Batten), Neur. 45
Hemisine and cocaine solution, plugging middle meatus with, in acute frontal sinusitis, Laryng. 142, 146
Hendaye, Baln. 18
Hendon, outbreak of milk-borne scarlet fever at, Epid. 86, 87, 100
Henoch's purpura, see *Purpura*, Henoch's
Henry, Dr., "Experiments on the Urine discharged in Diabetes Mellitus," quoted, Med. 152
Heredity in amaurotic idiocy (Tay-Sachs disease), Path. 174, 192
—— in cretinism, question of, Med. 8
—— in multiple cartilaginous exostoses, Med. 4
—— in pulmonary tuberculosis, Epid. 147-184
——, sex-limited, Med. 20
Herman, C., disinfection after measles, quoted, Child. 175
Hermaphroditism, supposed, case of (D. Drew), Obst. 109
——, unilateral, Obst. 109
Hernia (femoral), recurrences after operations for, Surg. 166, 167, 168
—— ——, treatment by inguinal method of radical cure (C. H. Fagge), Surg. 165-168
—— —— —— by Kocher's operation for radical cure, Surg. 166, 167
—— (interstitial), case of (O. L. Addison), Child, 40
Herpes of neck followed by internal ear deafness and facial paralysis, Otol. 17
HERRINGHAM, W. P.—Case of septic gastritis, Clin. 140
HERSCHELL, G.—Discussion on new type of œsophago-gastroscope, Laryng. 73
——, and HILL, W.—Demonstration of Hill-Herschell method of œsophago-gastroscopy, Laryng. 151, 152
Herschell's method of applying electricity in functional dyspepsia, Electr. 35
Herter, C. A., influence of benzoates on cultures of *Bacillus coli*, quoted, Path. 98
HERTZ, A. F.—Discussion on Röntgen ray examination of œsophagus, Electr. 28
——, discussion on use of belladonna in dyspepsia, Med. 31
—— —— on varix of right superficial epigastric vein, Clin. 51
Heterocephalus glaber, *H. phillipsi* (hairless rodent), Path. 298, 304
HETT, G. SECCOMBE.—Deformity of left auricle, Otol. 42
——, discussion on case of bilateral tuberculous disease of tonsils, Laryng. 68
—— —— on specimen of temporal bone from case of thrombosis of cavernous sinus, Otol. 28

HEWITT, Sir F. W., M.V.O.—Discussion on nasal reflex during anæsthesia, Anæsth. 31
von der Heyden, use of cold water in disease, quoted, Baln. 89
Hick, transplantation of ovaries, alluded to, Obst. 116
Hidrologia medica, commencement of journal (1896), Baln. 79
HIGGS, F. W.—Discussion on case of malformation of the heart, Child. 39
High-frequency current administered by auto-condensation in treatment of gouty dyspepsia, Electr. 41
—— ——, conductivity to, little affected by structure of tissue, Electr. 3, 4
—— —— in treatment of atonic dyspepsia with gastric dilatation, Electr. 41
—— —— —— of neurasthenia, Baln. 41
—— ——, thermal effects produced by (F. Nagelschmidt), Electr. 1-8
Highmore, antrum of, see *Antrum*, maxillary
Higier, name "Tay-Sachs disease" first proposed by, Path. 167
Hildebrand, division of posterior spinal nerve-roots for pain, quoted, Surg. 204
Hill and Barnard's instrument for recording blood-pressure, Med. 77, 78
HILL, W.—Carcinoma of deep pharynx with extension to gullet and to posterior margin of larynx, Laryng. 44
——, carcinoma of œsophagus, Laryng. 113
—— —— ——, dysphagia treated by a new form of intubation apparatus, Laryng. 9
—— —— —— ? temporary cure under radium treatment, Laryng. 43
——, demonstration on a living subject of the Hill-Herschell method of combined direct and indirect gastroscopy by means of a new type of œsophago-gastroscope, Laryng. 69
——, discussion on case of malignant stricture of œsophagus, Laryng. 83
—— —— on stricture of lower end of œsophagus, Laryng. 36
—— —— on treatment of cancer by X-rays and radium, Electr. 64
——, fungating carcinoma of œsophagus, disappearance of fungation and ulceration after treatment by radium, Laryng. 8
——. improved œsophago-gastroscope, combining the methods of direct and indirect vision, Laryng. 58
——, new pattern expanding œsophagoscope, Laryng. 58
——, permanent styletted oro-œsophago-gastric tube which had been in situ for thirteen weeks, Laryng. 113
——, sarcomatous growth of both tonsils, with multiple sarcomata in and beneath the skin, Laryng. 57
——, tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 109, 112
——, tuberculosis of the nose and cheek, case of, Laryng. 85
——, and HERSCHELL, G.—Hill-Herschell method of œsophago-gastroscopy, demonstration of, Laryng. 151, 152
Hildanus, Fabricius, explosions and loud noises injuring hearing, Otol. 93
—— ——, methods of removal of foreign bodies from auditory meatus, Otol. 96
HILLIARD, HARVEY.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 9, 12
——, discussion on nasal reflex during anæsthesia, Anæsth. 32
Hindhead, health resort suitable for foreign invalids, Baln. 21
Hip-joint, flexed and adducted, forcible manipulation of, followed by ossification of both iliaci, Surg. 133
——, fractures about, Surg. 12
—— —— in adolescents, Surg. 12
—— —— ——, separation of epiphysis in, Surg. 12, 13
—— —— ——, followed by traumatic coxa vara, Surg. 12
—— —— ——, types of, Surg. 12
—— ——, treatment, Surg. 14-16
——, osteo-arthritis of, following tuberculous disease of same joints, Baln. 24
—— ——, formation of osteophytes in, Baln. 29, 33
——, tuberculous disease of, followed by osteo-arthritis of same joints, Baln. 24
Hippocrates, "Airs, waters, and places," quoted, Baln. 1, 2

Hippocrates, epidemics and other diseases, and states of the weather, Epid. 189
—— on phthisical diathesis, Epid. 151
——, spa treatment in days of, description, Baln. 9
Hirsch (1866) on tuberculous contagion and disposition, quoted, Epid. 167
Hirschfelder, experimental work on auriculo-ventricular bundle, Therap. 97, 98
His, bundle of, Therap. 97-113
—— ——, see also *Auriculo-ventricular bundle*
HOBDAY, FREDK.—Specimens of equine larynx and records of cases illustrating "ventricle-stripping" operation for relief of "whistling" and "roaring" in horses; demonstration of instrument, Laryng. 87
Hoffa, tuberculous disease of hips and knee followed by osteo-arthritis of same joints, quoted, Baln. 24
Hoffmann, case of progressive spinal muscular atrophy of infants and and young children, quoted, Neur. 68
Hoffmann and Ultzmann, "Atlas der Harnsedimente," Path. 135
Hoffmann, Friedrich, "Essay on the nature and properties of water" (1761), Baln. 82
Hog-cholera, mutation of intestinal micro-organisms in, Epid. 124, 136
Holland, hydrotherapy in, Baln. 89
HOLLAND, C. T.—Discussion on treatment of cancer by X-rays and radium, Electr. 63
Holmes's "System of surgery" (case of fatal hæmorrhage from lateral sinus after injury, quoted), Otol. 90
HOLMES, GORDON M.—Pathology of amaurotic family idiocy, Path. 171
——, pathological anatomy of a case of amaurotic family idiocy, Path. 199-204
——, and Stewart, T. Grainger, cerebellar tumours, referred to, Otol. 37
Holst, outbreaks of acute gastro-enteritis from use of raw milk from cows with mastitis, quoted, Epid. 76
"Holy wells," supposed benefits obtained from, Baln. 82, 102
HOME, Fleet-Surgeon W. E., R.N.—Discussion on notification of disease and epidemiology of poliomyelitis, Epid. 225
Homes of refuge for advanced consumptives, Epid. 31, 44, 47
Hop, common, contains sulphur, Therap. 23
HOPE, C. W. M.—Case of malignant stricture of œsophagus, Laryng. 153
——, demonstration of œsophagoscopy of malignant growth and application of radium, Laryng. 151
HOPEWELL-SMITH, A.—Discussion on dentigerous cyst with cellular contents, Odont. 89
——, discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 46
—— —— —— —— —— —— —— —— ——, report on case, Odont. 43
—— —— on cyst of jaw, Odont. 16
—— —— on distribution of nerve-fibres of dental pulp, Odont. 54
Hôpital Claude-Bernard (Paris), mortality from measles at, Child. 174
—— Hérold (Paris), mortality from measles at, Child. 174
—— Pasteur (Paris), mortality from measles at, Child. 174
Hopkins, F. G., F.R.S., output of uric acid in gout, quoted, Baln. 59
Hopkins's process, estimation of uric acid by, Baln. 47
Horder, T. J., and Andrewes, F. W., organisms causing sore throat in man, quoted, Epid. 79
Hormone, term defined, Path. 305
HORNE, W. JOBSON.—Case illustrating "dents de scie" of the vocal cords, Laryng. 10
——, discussion on case for diagnosis, Laryng. 57
—— —— —— of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 103
—— —— —— of complete stenosis of larynx from epithelioma in man, aged 23, Laryng. 121
—— —— —— of epithelioma on right palatoglossal fold, Laryng. 60
—— —— —— of nasopharyngeal fibrous polypus, Laryng. 61
—— —— —— of papilloma of pharynx, Laryng. 62
—— —— —— of small tumour of soft palate, Laryng. 13

Horne, W. Jobson.—Discussion on case of ulceration of left vocal cord, Laryng. 63
——, discussion on case of ulceration of soft palate, Laryng. 12
—— —— on ectopia of pituitary, Laryng. 54
—— —— on equine larynx and "ventricle-stripping," Laryng. 91
—— —— on functional aphonia in boy, aged 15, Laryng. 115
—— —— on improved form of Hay's pharyngoscope, Laryng. 108
—— —— on new pattern expanding œsophagoscope, Laryng. 58
—— —— on specimen of pedunculated growth on an elongated uvula, Laryng. 107
——, laryngeal disease in a man, aged 45, probably tuberculous, but presenting appearances of epithelioma, Laryng. 117
——, laryngeal tuberculosis in a boy, aged 10, Laryng. 116
——, perforation of the soft palate due to syphilis, but presenting unusual features, Laryng. 116
——, periotic bones of fossil Cetacea, Otol. 114
——, simplified insufflator for use in the treatment of diseases of the throat, nose, and ear, Laryng. 117
Horrocks, conversion of *Bacillus typhosus* into organism of non-fermenting type, quoted, Epid. 142, 146
Horse, auricular fibrillation in, Med. 100
—— epizootic lymphangitis in, identical with sporotrichosis, Derm. 115
—— glanders in, identical with sporotrichosis, Derm. 115
——, "grunting" in, Laryng. 91
——, larynx of, specimens of, and records of cases illustrating results of "ventricle-stripping" operation for relief of "whistling" and "roaring" in horses; demonstration of instruments (F. Hobday), Laryng. 87
——, lead-poisoning in, symptoms of, Laryng. 91
—— sporotrichosis infection from, Derm. 115
Horsford, Cyril.—Case of aphonia for diagnosis, Laryng. 16
——, discussion on case illustrating "dents de scie" of vocal cords, Laryng. 10
—— —— on cases illustrating results of removing nodules from cords by means of galvano-cautery (by indirect method), Laryng. 41
—— —— on two specimens of quadrilateral cartilage (nasal septum) removed by submucous section, Laryng. 5
——, laryngeal tuberculosis, cure (?), Laryng. 43
Horsley, Sir V., F.R.S., danger of morphine injection before chloroform inhalation, quoted, Anæsth. 43
Horvath, case of ossification of both iliaci following forcible manipulation of hip-joint, quoted, Surg. 133
Hospice des Enfants Assistés (Paris), mortality from measles at, Child. 174
Hospital for Sick Children, Great Ormond Street, results of operation for cleft palate at, Surg. 171, 172, 191-196
Hospital patients, difficulty of after-treatment for fractures about joints, Surg. 42
Hospitals, installation of string galvanometer in, a necessity, Med. 98
—— (general), incidence of gall-stones among patients at, less than among general population, Path. 90, 91
—— —— —— —— shown by post-mortem records at, Path. 88, 89
—— —— —— —— —— —— ——, compared with similar records from lunatic asylums, Path. 88, 89
Hot-air baths, antiquity of, Baln. 85
Hot fluids, introduction into ear, injuring tympanic membrane, Otol. 84
"Hot-houses," treatment of leprosy in, Baln. 85
Hot water and potassium permanganate as mouth-wash during rectal feeding, Therap. 72
Houses, damp, rheumatic disorders endemic in, Baln. 28
Houston, folds of, Surg. 91, 92: see also *Plica* transversalis recti
Houston, A. C., typhoid bacilli in London drinking water, quoted, Epid. 126

Howarth, W.—Case of chronic laryngitis, Laryng. 108
——, discussion on new pattern expanding œsophagoscope, Laryng. 58
—— —— on removal of denture from œsophagus, Laryng. 55
——, tuberculosis of soft palate, pharynx, and larynx in a girl, aged 18, Laryng. 66
Hovell, T. Mark.—Discussion on bacteriology of chronic post-nasal catarrh, Otol. 52
Howell, C. M. Hinds.—Case of gastric crisis in ? tabes, division of posterior roots, Neur. 48
——, discussion on resection of posterior spinal nerve-roots, Surg. 253
Hübener and Conradi, association of *Bacillus typhosus* B with diseases other than paratyphoid fever and presence in healthy individuals, quoted, Epid. 54, 55, 70, 71
—— ——, ingestion of *Bacillus suipestifer* with impunity, maintained by, Epid. 58, 59
Huber, absorption of peptonized eggs in rectal feeding, Therap. 64
Huggenberg, case of typhoid carrier, quoted, Epid. 17
Hughes, Cecil.—Note on a case of "Cheyne-Stokes" respiration during anæsthesia, Anæsth. 37
Humerus, fracture of anatomical neck, treatment, Surg. 4
—— —— ——, cases for operative treatment, Surg. 37, 38, 41
—— —— ——, supra-condylar, reasons for operative treatment, Surg. 35
—— —— —— ——, treatment by flexion of elbow, Surg. 8, 9
—— —— of surgical neck, treatment, Surg. 4, 5
——, fractures of, advantage of slinging wrist to neck with elbow acutely flexed, Surg. 23, 24, 29, 45, 46
——. fractures of upper end complicated by dislocation of head, treatment, Surg. 5, 6
——, lower epiphysis of, separation with displacement (D. Drew), Child. 9
—— myeloma of upper end of, Child. 203
——, suppurative epiphysitis of upper end of, followed by chronic pyæmia of five years' duration (D. Drew), Child. 162
Humphris, F. Howard.—Discussion on Bauer's qualimeter, Electr. 72
——, discussion on British health resorts for foreign invalids, Baln. 22
—— —— on low blood-pressure, Med. 76
—— —— on value of electricity in abdominal diseases, Electr. 45
Humphry, G. P., and Rolleston, H. D.—Rigidity of the spine, Clin. 146
Humphry, L.—Case of post-diphtheritic paralysis and hemiplegia, Child. 195
Hunt, E. L., and Latham, A.—A case of malignant or infective endocarditis treated with an autogenous vaccine administered by the mouth, showing the disappearance of the infecting organisms from the blood within twelve months from the commencement of treatment, together with marked improvement in the clinical symptoms, Clin. 14-21
Hunter, W.—Case of "Addisonian" anæmia (idiopathic pernicious anæmia), Clin. 37
Hutchinson, Sir J., F.R.S., case of hypotrichosis, quoted, Path. 302
——, sex-incidence in congenital syphilitic deafness, quoted, Otol. 4
Hutchinson, J.—Discussion on transverse fractures of the patella, Surg. 83
——, gastric and intestinal hæmorrhages (probably of septic origin) occurring after abdominal operations, Surg. 58-64, 67
Hutchinson, J. R., case of typhoid carrier, quoted, Epid. 17
Hutchinsonian type of teeth in inherited syphilis, Child. 67
Hutchison, Robert.—Case of splenomegaly (? congenital family cholæmia), Clin. 129
——, discussion on absence of abdominal respiratory movement in pericarditis, Med. 47
—— —— on case of Graves's disease, Clin. 125
—— —— —— of von Jaksch's anæmia, Child. 157
—— —— on congenital heart disease without murmur, and with family history of congenital cyanosis, Child. 160
—— —— on low blood-pressure, Med. 77
——, two cases of secondary anæmia associated with achylia, with comments, Clin. 141
Hutinel, Professor, mortality from measles at Hospice des Enfants Assistés, Child. 174
Huxham, cold and tepid applications employed by, Baln. 83
Huxley, foundation of Darwinian varieties, quoted, Path. 306

Hydatid of uterus (A. E. Giles), Obst. 245
—— ——, microscopical examination, Obst. 246
Hyde, Samuel (1849-1899), biographical notice of (R. Fortescue Fox), Baln. 64-66
—— ——, founder of British Balneological Society (1895), Baln. 65, 66
—— —— —— of *Journal of British and Foreign Health Resorts*, Baln. 65
——, see also *Samuel Hyde Memorial Lectures*
Hydrocephalus and buphthalmos, case of (J. C. W. Graham), Child. 197
Hydrological treatment of neurasthenia, Baln. 39
Hydrology, definitions of, Baln. 79, 80
—— (medical), Baln. 78, 79
—— ——, definition of, according to modern usage, Baln. 80, 81
—— ——, earliest mention of, Baln. 78, 79
—— ——, examinations in, Med. 116, 117
—— ——, intensive method at foundation of, Baln. 111
—— ——, preference for term instead of "balneology," Baln. 99
—— ——, teaching of, Baln. 113
—— —— —— of, as obligatory branch of study, Baln. 114
—— —— —— in Austro-Hungary, Baln. 115
—— —— —— in Belgium, Baln. 116
—— —— —— in British Isles, absence of, Baln. 113, 117, 118
—— —— —— in France, Baln. 113
—— —— —— in Germany, Baln. 114
—— —— —— in Italy, Baln. 115
—— —— —— in United States, Baln. 116
—— —— —— in Switzerland, Baln. 116
Hydronephrosis, due to stricture of ureters complicating cancer of uterus, Obst. 77, 102
Hydropathy, Baln. 76, 77
——, rise of, Baln. 84, 85
Hydrosalpinx with ovarian peritoneal cysts (H. Macnaughton-Jones), Obst. 221
Hydrostatic pressure, factor of, in immersion baths, Baln. 73
Hydrotherapeutics (hydrotherapy), capacities of, Baln. 91
—— ——, climate in relation to, Baln. 70
—— ——, clinics and institutes for, in Germany, Baln. 86, 87, 97
—— ——, definition of, Baln. 77
—— ——, distinct from balneology in Germany, Baln. 78
—— ——, earliest use of term, Baln. 77
—— ——, establishment and progress of, in France, Baln. 87, 88, 97
—— —— —— upon scientific basis, Baln. 85, 86
—— —— general extension of, Baln. 84
—— —— in Belgium, Baln. 89
—— —— in England, empiric basis of, Baln. 99
—— —— ——, first serious advocacy of, by Sir J. Floyer, Baln. 82
—— —— ——, needs to be placed on scientific basis, Baln. 97, 117
—— —— in Holland, Baln. 89
—— —— in Italy, Baln. 88, 89, 97
—— —— in Russia, Baln. 89
—— —— in Scandinavia, Baln. 89
—— —— in Switzerland, Baln. 116
—— —— in United States, Baln. 89, 90
—— ——, laws of reaction in, Baln. 92
—— ——, lectures on, at University of Berne, Baln. 116
—— ——, methods applicable to acute and chronic illness, Baln. 109
—— ——, modern (R. Fortescue Fox), Baln. 81
—— ——, *plus* followed by *minus* applications underlying principle in many forms of, Baln. 95

Hydrotherapeutics (hydrotherapy), teaching of, at Columbia University (New York) and Johns Hopkins University (Baltimore) and at Chicago, Baln. 116
Hygiene, personal, instruction of school children in, Epid. 41
Hygienic measures to contract predisposition to pulmonary tuberculosis in children, Epid. 41, 42
Hylobates haïnanus (Hainan Gibbon), Path. 291
Hyperæmia following application of diathermic current, Electr. 5, 6
Hyperchlorhydria and gastric motor insufficency of functional dyspepsia, treatment by electricity, Electr. 35
——, contra-indication for farado-massage, Electr. 40, 45
——, control of, by administration of belladonna, Med. 26, 29, 33
——, treatment by sinusoidal current applied by Abrams's method, Electr. 45
Hyperkeratosis of palms and soles, case of (H. G. Adamson), Derm. 18
—— —— ——, treatment by X-rays, Derm. 18
Hypnoid state, definition of, Neur. 13
—— ——, hysteria originating as, Neur. 13, 14
—— ——, production of, through terror, Neur. 13
Hypnotism, alleviation of general anæsthesia under, Anæsth. 22
——, amnesia during, Neur. 7
——, analgesia produced by, Anæsth. 16
—— and hysteria, no essential difference between, Neur. 9, 10
——, Braid's method of, Anæsth. 22
——, induction to state of active somnambulism under, Anæsth. 16
——, painless extraction of teeth under, Anæsth. 14, 19, 20
——, production of surgical anæsthesia by (E. Ash), Anæsth. 13
—— —— —— ——, see also under *Anæsthesia* (surgical), production by hypnotism
——, somnambulism artificially produced under, Neur. 10
——, subjects for, best, Anæsth. 18
—— ——, bad, Anæsth. 20
—— ——, good, Anæsth. 20
——, suggestion distinct from, Anæsth. 23
——, susceptibility to, Anæsth. 23
——, see also *Post-hypnotic influence*
Hypochlorhydria and gastric motor insufficiency, treatment by electricity, methods, Electr. 36
Hypodermic administration of medicinal waters, Baln. 108
Hypotrichosis, human, examples of, Path. 301-304
—— in family of Australian aborigines, Path. 303
Hypoxanthin, administration to patients suffering from gout, effects of, Baln. 59
——, effect on excretion of uric acid, Baln. 51
Hyslop, T. B.—Discussion on preliminary communication on syphilis and mental deficiency, Child. 170
Hysterectomy (abdominal) for removal (unruptured) of interstitial gestation sac (A. H. N. Lewers), Obst. 154
—— compared with "utriculoplasty" for uterine hæmorrhage, Obst. 276
—— (extraperitoneal), Obst. 201, 215
—— followed by double phlebitis (H. Macnaughton-Jones), Laryng. 223
—— —— by thrombosis, Obst. 224
—— in contracted pelvis and protracted labour (septic cases), Obst. 165, 166
—— performed sixty-three hours after labour, case summarized, Obst. 12
—— (subtotal), continuation of menstruation after, Obst. 117
—— —— in treatment of fibromyoma uteri; 40 additional histories (Alban Doran), Obst. 115
—— —— —— —— ——, results as to menopause, Obst. 124-134, 136, 137
—— —— —— —— —— —— when both ovaries removed (Alban Doran), Obst. 124
—— —— —— —— —— —— when both ovaries saved (Alban Doran), Obst. 134
—— —— —— —— —— —— when one ovary saved (Alban Doran), Obst. 129

Hysterectomy (subtotal), in treatment of fibromyoma uteri, summary of complete series of 100 cases (Alban Doran), Obst. 142
—— (supravaginal), Obst. 211
—— —— (Porro's), with extraperitoneal treatment of stump in cases of contracted pelvis and protracted labour, comparative safety of, and reduction of mortality under, Obst. 177, 187
—— —— —— —— —— —— —— —— —— —— ——, suitable where gonococcal infection is present, Obst. 177
—— —— —— —— —— —— —— —— —— —— ——, with virulent infection, Obst. 177
—— ——, with intraperitoneal treatment of stump in cases of contracted pelvis and protracted labour where infection was suspected, Obst. 175, 176
—— —— —— —— —— —— —— —— —— ——, with virulent infection, Obst. 175
—— (total), cessation of menstruation after, Obst. 116
—— —— in treatment of uterine fibroid, Obst. 116
—— ——, statistics needed, Obst. 117, 143
—— —— (abdominal) and Cæsarean section in cancer complicating labour, Obst. 191
—— —— —— in cases of contracted pelvis and protracted labour, with virulent infection, Obst. 178
—— —— —— —— —— —— —— —— —— ——, risks of, Obst. 178
—— —— —— —— —— —— —— —— —— —— ——, how to avoid, Obst. 178
—— —— —— —— —— —— —— —— —— ——, sterilization of vagina during, Obst. 178
—— —— —— —— —— —— —— —— —— ——, technique of, Obst. 178
—— —— —— in contracted pelvis and protracted labour (septic cases), Obst. 163, 188, 200, 211, 213
—— —— —— —— —— —— ——, with gonorrhœa, Obst. 190
—— —— ——, Wertheim's method, cases operated on by (C. P. Childe), Obst. 79-100
—— —— —— —— —— —— ——, age of patients, Obst. 79
—— —— —— —— —— —— ——, examination under anæsthetic, Obst. 87
—— —— —— —— —— —— ——, general condition of patients, Obst. 88
—— —— —— —— —— —— ——, previous confirmation of presence of cancer by microscopical report, Obst. 88
—— —— —— —— —— —— ——, stage of disease, Obst. 87
—— —— —— —— —— —— ——, statistics, tabulated, Obst. 80-85. 97
—— —— —— —— ——, complicated by sepsis attacking abdominal or pelvic wound, Obst. 92, 93
—— —— —— —— ——, isolation of ureters in, Obst. 89
—— —— —— —— ——, primary mortality, Obst. 94-96
—— —— —— —— —— —— —— compared with that from vaginal hysterectomy, Obst. 94-96. 103
—— —— —— —— ——, reasons for preference to vaginal hysterectomy, Obst. 99
—— —— —— —— ——, removal of lymphatic glands in, Obst. 90, 91
—— —— —— —— ——, shock during or following, Obst. 91
—— —— —— —— ——, technique, points in, Obst. 104
—— —— —— —— ——, ultimate results, Obst. 98
—— —— —— —— —— —— —— compared with those of vaginal hysterectomy, Obst. 99
—— —— —— —— —— vaginal cuff in, Obst. 78
—— (vaginal), advantages of, Obst. 101
—— ——, primary mortality, Obst. 101
—— —— —— —— compared with that from abdominal panhysterectomy (Wertheim's method), Obst. 94-96, 103
—— ——, reasons for preference of Wertheim's method to, Obst. 99
—— ——, ultimate results compared with those of Wertheim's method (abdominal panhysterectomy), Obst. 99
Hysteria and hypnotism, no essential difference between, Neur. 9, 10
—— and neuromimesis, Neur. 2

Hysteria and paralysis from idea, Neur. 2
——, "conversion of the affect" in (substitution of bodily phenomenon for mental distress), Neur. 11, 12, 15, 16, 18
—— a defence-neurosis, Neur. 12
——, definitions of, Neur. 2
——, defined as psychical trauma, Neur. 10
——, disability of bodily functions from, Neur. 7
——, distinction from neurasthenia (or psychasthenia), Neur. 9
——, emotions exhibited by subjects of, relate to past circumstances, Neur. 5
——, field of vision restricted in subjects of, Neur. 4
——, fit of, comparable with somnambulism, Neur. 5
—— ——, disappearance of personal consciousness during, Neur. 5
——, fixed idea of local pain or tenderness a symptom of, Neur. 8, 9
——, "ideogenous" nature of, presumed, Neur. 2
——, manifestation of, following typhoid fever, Neur. 8
——, mental association in, inappropriate instances of, Neur. 12
—— —— theory of, Neur. 17
——, origin of, sexual memories in relation to, Neur. 15, 16, 18
—— —— of term, Neur. 2
——, originating as defence-neurosis, Neur. 12, 13
—— —— as hypnoid state, Neur. 13, 14
——, subconscious states of mind and, Neur. 3
——, subjects of, hypnotism of, Anæsth. 20
——, symptoms of, trophic or vasomotor, Neur. 17
—— (traumatic), dramatic behaviour of subject of, Neur. 5, 6
——, treatment, method described, Neur. 14, 15
——, see also *Anæsthesia* (hysterical); *Mind*, hysterical
Hysterical trophœdema, functional (E. G. Fearnsides), Derm. 84
Hysterotomy in contracted pelvis and protracted labour, Obst. 182

Ice, sucking of, during rectal feeding, objectionable, Therap. 72
Ichthyol, Therap. 22
Ichthyosis follicularis, case of (J. M. H. MacLeod), Derm. 131
—— hystrix linearis, syn. nævus verrucosus, case of, Derm. 66
—— —— —— (F. P. Weber), Child. 32
—— in child, treatment by thyroid extract (T. Colcott Fox), Derm. 72
—— simulating fading roseola in case of secondary syphilis (H. G. Adamson), Derm. 33
Idea, hysterical paralysis due to, Neur. 7
——, paralysis from, and hysteria, Neur. 2
Idiocy and cerebral sclerosis (E. Cautley), Child. 184
—— (amaurotic—Tay-Sachs disease), absence of choline and cholesterol from cerebrospinal fluid in, Path. 155, 157, 161
—— —— —— ——, age at which disease proves fatal, Path. 168
—— —— —— —— —— —— —— —— ——, extension beyond the two years' limit, Path. 168
—— —— —— ——, Babinski's sign in, Path. 153
—— —— —— ——, cases of (H. B. Carlyll), Path. 147-172
—— —— —— —— ——, clinical histories, Path. 149-167
—— —— —— —— ——, histological and chemical examination of the nervous structure (F. W. Mott, F.R.S., and S. A. Mann), Path. 147-198
—— —— —— —— ——, post-mortem examination results, Path. 157, 166
—— —— —— ——, cellular changes in brain in, Path. 177
—— —— —— ——, chemical examination of brain in two cases, Path. 195-198
—— —— —— —— —— —— —— —— ——, comparison with normal brain, Path. 195-198
—— —— —— ——, "claw" hand in, Path. 150, 151, 155, 157, 159, 160

Idiocy (amaurotic—Tay-Sachs disease), condition of eyes in, Path. 151-153, 157-161, 163, 165, 167-169
—— —— —— —— —— of nervous system in, Path. 153-166
—— —— —— ——, congenital, Path. 168
—— —— —— ——, deformity of limbs in, Path. 150, 151
—— —— —— ——, examination of retina, Path. 188, 189
—— —— —— ——, family prevalence, Path. 147, 148, 149, 167, 168
—— —— —— —— —— —— sometimes absent, Path. 168
—— —— —— ——, fatty degeneration of cytoplasm of neurones in, Path. 176, 178, 193
—— —— —— ——, heredity in, Path. 174, 192
—— —— —— ——, historical summary, Path. 167-172
—— —— —— ——, Kernig's sign in, Path. 154, 160, 161
—— —— —— ——, lumbar puncture and examination of cerebrospinal fluid in, Path. 154, 155, 157, 161, 165
—— —— —— ——, pathogenesis, hypothesis as to, Path. 174, 192
—— —— —— —— ——, morphological and chemical investigations in relation to, Path. 174, 192
—— —— —— ——, pathological anatomy of case (G. M. Holmes), Path. 199-204
—— —— —— ——, pathology of, Path. 169-172
—— —— —— ——, rare disease occurring among Jewish children, Path. 147, 148, 174
—— —— —— ——, sections from specimens, detailed description of figures and photomicrographs, Path. 179
—— —— —— —— —— ——, methods of staining, Path. 177-194
—— —— —— ——, syphilis not a factor in, Path. 168
—— (Mongolian), skull from case of (G. S. Haynes), Child. 204
Idioglossia, case of (Eric Pritchard), Child. 52
Ileum, tuberculous ulcer of (perforated) (L. E. C. Norbury), Clin. 50
Iliac vein, external, and obturator nerve, large gland lying between, Obst. 75
Iliaci, both, ossification of, following forcible manipulation of flexed and adducted hip-joint, Surg. 133
Illegitimate children, mothers of, selected as wet-nurses by preference, Child. 124
Immersion baths, subthermal, Baln. 96
Imschanitzky, experimental work on auriculo-ventricular bundle, Therap. 98
Incisor (central), preceded by multiple supernumerary teeth, case of (A. A. H. Johnson), Odont. 42
—— (lower), erosion cavity on lingual aspect of (C. F. P. Baly), Odont. 69
—— (temporary), dislocation of pulp of (C. F. P. Baly), Odont. 69
India, medicinal springs of, Baln. 117
——, outbreaks associated with *Bacillus paratyphosus* in, Epid. 117
——, prevalence of *Bacillus paratyphosus* A in, Epid. 53
Indian girl (aboriginal), specimens of hair from (J. M. H. MacLeod), Derm. 149
Indican, estimation by colorimetric adaptation of Bouma's indigo red method, Baln. 47
——, excretion before and after taking sulphur water, Baln. 55
Indoxyl group, administration of calomel increases excretion of bodies of, Baln. 56
Induced current preceded by galvanic current in treatment of severe functional dyspepsia, Electr. 36
Induction coil, new, brushes fitted to, Electr. 80, 81, 83
—— —— ——, connexions of, Electr. 80, 81
—— —— ——, description of (Professor E. and W. H. Wilson), Electr. 79-83
—— —— ——, "make" and "break" of current with, Electr. 80, 81, 82
Infancy, operation for cleft palate in, advocated, Surg. 170-172, 192-194, 195
—— —— —— ——, mortality, Surg. 195, 197
—— —— —— ——, objected to, Surg. 183, 190
Infant, congenital heart disease in, specimen showing, Child. 204
—— —— xanthoma in, Derm. 76

Infant, nævo-lipomata in (E. G. Graham Little), Derm. 23
Infant feeding pot, Child. 126
Infantilism, case of (H. E. Symes-Thompson), Clin. 45
—— (pancreatic), test for, Child. 60
—— —— and *Bacillus bifidus*, Child. 61
—— (symptomatic), Hastings Gilford type of, cases of (H. Batty Shaw), Clin. 33
——, two cases of (Kenneth Kellie), Child. 59
—— with polyuria and chronic renal disease, case of (H. M. Fletcher), Child. 95
—— with thyroid inadequacy (juvenile myxœdema) (H. M. Fletcher), Child. 93
Infants, drugging of, by monthly nurses, Child. 141
——, feeding of, dawn of scientific methods in, Child. 125
—— ——, during Middle Ages, Child. 110, 111
—— ——, earliest books on, Child. 110
—— ——, in Elizabethan period, Child. 111-116
—— ——, history from Elizabethan times (D. Forsyth), Child. 110-141
—— —— (artificial), advocates of enlightened methods, Child. 136-138
—— —— ——, first mention not till eighteenth century, Child. 111
—— —— ——, impossible in London in Tudor period owing to insanitation, Child. 115, 116
—— —— ——, in early nineteenth century, Child. 128, 129
—— —— ——, success of (1850-1900), Child. 136
—— (hand-fed), mortality compared with that of those suckled, Child. 129
——, hand-feeding of, scheme for, Child. 138
——, infection from subjects of tuberculosis, Child. 222, 223
——, mortality (1750-1850), Child. 129, 130
—— —— at Foundling Hospital, Child. 129, 130
—— —— of, during reign of Charles II, Child. 117, 118
—— (newly born), Erb's paralysis in, how produced, Child. 9
——, rearing of, during Stuart period (seventeenth century), Child. 116
——, vaginal secretion in (G. Drummond Robinson), Obst. 26
—— and young children, progressive spinal muscular atrophy of (F. E. Batten), Neur. 53-84
Infection, mechanism of production of agglutinins in absence of, Path. 71
Infectious disease, gangrene of extremities following, Child. 11, 12
Infective diseases, empirical treatment of, example of vaccine therapy (C. Riviere), Therap. 39
Infiltrate (subperiosteal) surrounding femur shaft, from case of atypical chloroma, Path. 63
Infiltrates in chloroma, distribution, Path. 67, 68
Inflammatory exudations, absorption under subthermal bath treatment, Baln. 96
Influenza attacking brain and nervous system, Epid. 192, 223
—— complicated by pneumonia, Epid. 192, 223
——, connexion of outbreaks of poliomyelitis and cerebrospinal meningitis with, Epid. 224
—— —— with neurasthenia, Baln. 44
—— followed by frontal sinus suppuration, Laryng. 138
Infundibulo-pelvic ligament, Obst. 345
Ingals, Fletcher, advocacy of intranasal operations in chronic frontal sinus suppuration, Laryng. 130, 139
Inguinal method of radical cure for femoral hernia (C. H. Fagge), Surg. 165
—— —— —— —— —— ——, approximation of conjoint tendon and Cooper's ligament in, Surg. 166, 168
—— —— —— —— —— ——, division of Gimbernat's ligament in, Surg. 167
Inhaler (new) for administration of nitrous oxide gas by nasal method (J. F. Trewby), Anæsth. 12
—— —— —— —— —— —— —— ——, advantages, Anæsth. 12
Inhibition, reflex cardiac, influence of chloroform and asphyxia upon, Path. 206-214
Injury, formation of bone in periosteum separated by (Sir A. A. Bowlby), Surg. 142
Insane (the), incidence of gall-stones in (J. P. Candler), Path. 87
—— —— ——, greater than in the sane, Path. 90

Insane (the), incidence of gall-stones in, greater than in the sane, explanations to account for, Path. 91
—— —— of primary carcinoma of gall-bladder and biliary passages in, Path. 91, 92
—— occurrence of othæmatoma among, Otol. 104
Insomnia, treatment by subthermal baths, Baln. 97
Instruments for intravenous injection of salvarsan ("606"), demonstration of (W. A. Lieven), Laryng. 45
Insufflator, simplified, for use in treatment of diseases of throat, nose, and ear (W. J. Horne), Laryng. 117
Insurance (life), see *Life insurance*
Intensive method, foundation of hydrological medicine, Baln. 111
Inter-iliac lymphatic glands, see *Lymphatic glands*, inter-iliac
Intermittences, irregular pulse due to, Med. 87, 88
International Congress of Laryngology, Berlin, notice respecting exhibition of instruments, &c., Laryng. 87
Interrupter of new induction coil apparatus, Electr. 79
Intestinal obstruction, acute, caused by fibromyoma of uterus (J. Barris), Obst. 108
Intestine (large), power to digest foods slight, Therap. 63
—— (small), effects of tryptophane when administered by, experimentally, Baln. 56
—— —— and omentum, suppurating ovarian tumour perforated into, successfully removed with affected bowel, omentum, and appendix (H. R. Spencer), Obst. 157
—— ——, sarcoma of, specimen, Child. 203
Intestines, skiagraphy of, in bismuth cases, Electr. 91
——, support of, necessary under treatment of gastric dilatation by farado-massage, Electr. 40
——, tuberculous infection by way of, Child, 216
——, see *Micro-organisms* (intestinal)
Intracranial complications, indication for radical operation in chronic frontal sinus suppuration, Laryng. 129, 132
Intranasal disease, conjunctivitis due to, Laryng. 103
—— ——, mucocele due to, Laryng. 103
—— operation in chronic frontal sinus suppuration, Laryng. 130, 134, 139, 140
—— —— —— —— —— ——, advantages, Laryng. 130
—— —— —— —— —— ——, disadvantages and contra-indications, Laryng. 131, 140
Intubation apparatus, new form of, treatment of dysphagia by, in carcinoma of œsophagus (W. Hill), Laryng. 9
Invalids, foreign, advantages of British health resorts for, Baln. 11-22
—— ——, British health resorts for, winter marine, in summer season, Baln. 20
—— ——, class of British marine watering-place suitable to, Baln. 18
Involution cysts, ovarian, Obst. 219, 220
Iodine, weak solutions of, in intranasal treatment of chronic frontal sinus suppuration, Laryng. 129
Iodoform, keratin-coated pearls of, in diagnosis of pancreatic infantilism, Child. 60
Ionic medication in neurasthenia, Baln. 44
Ionization in water, Baln. 107
Iredell, C. E.—Epithelioma on lupus vulgaris in a girl, aged 13 (shown by H. G. Adamson), Derm. 139
Iridocyclitis due to oral sepsis, Odont. 29
—— due to syphilis, Odont. 28, 29
Iritis due to diabetes, Odont. 28, 29
—— —— to gonorrhœa, Odont. 28, 29
—— —— to syphilis, Odont. 28, 29
—— (recurrent), in case of hypertrophied middle turbinal, Laryng. 167
Iron, iodide of, in splenomegaly of inherited syphilis of children, Child. 68
—— and tannin staining method, to demonstrate distribution of nerve-fibres of dental pulp, Odont. 53

Irrigation treatment of bronchitis, Therap. 43, 46, 47
Irritation one of commonest factors in causation of cancer, Electr. 51
Irthlingborough, epidemic of poliomyelitis at (1910), Epid. 214, 215
Ischæmia, local, effect of, in local anæsthesia by drugs, Therap. 53
Ischial callosities of monkeys, Path. 287
Isoamylamine, reaction of arteries to, Therap. 89
Isodulcite, mutation of *Bacillus typhosus* in respect of, Epid. 142
Italy, diploma in hydrology obtainable in, Baln. 117
——, hydrotherapy in, Baln. 88, 89, 97
——, teaching of medical hydrology in, Baln. 115, 116
IVENS, Miss F.—Demonstration of a very young tubal ovum, Obst. 313

Jacob, Dr. P., lecturer on physical therapy at von Leyden's medical clinic, Baln. 115
Jacobi, motility of particles of matter and metabolism, Baln. 107
Jacobs, Professor, practice as regards removal of appendix in pelvic operations, Obst. 353
Jacobsen, typhoid bacillus described by, showing two distinct types of colony, Path. 98
Jacoby, division of posterior spinal nerve-roots for pain, quoted, Surg. 204
Jacquet, on connexion of baldness with dental trouble, quoted, Odont. 75
Jadassohn, local inflammation preceding macular atrophy, quoted, Derm. 123
Jaffrey, F., nephrostomy for suppression from acute nephritis, quoted, Clin. 162
Jahrbuch für Balneologie, Hydrologie, und Klimatologie, commencement of (1871-77), Baln. 79
von Jaksch's anæmia, case of (S. Neave), Child. 155
JAMES, W. W.—Dentigerous cyst with cellular contents, Odont. 85
——, cases of oral sepsis causing thrombosis of cavernous sinus, quoted, Odont. 117
——, discussion on case of absorption of permanent molar due to unerupted premolar, Odont. 38
—— —— —— —— of multiple supernumerary teeth preceding central incisor, Odont. 49
—— —— on endothelioma of gum and cyst of jaw, Odont. 15
—— —— on infection of cavernous sinus due to oral sepsis, Odont. 117
—— —— on inflammatory eye conditions due to oral sepsis, Odont. 32, 35
—— —— on model of maxilla with exostoses, Odont. 41
—— —— on the relation between the teeth and certain diseases of the skin and mucous membrane, Odont. 83
Janet, Professor, hypothesis of restricted field of personal consciousness, Neur. 6, 17
——, nature of emotion, quoted, Neur. 5
Japan, natural hyperthermal baths of, Baln. 94
——, prevalence in, of tuberculosis, with small consumption of milk, Child. 222, 231
——, subthermal spas in, Baln. 97
Jardine, indications for craniotomy, quoted, Obst. 166
Jaundice (acholuric), chronic, with splenomegaly, Clin. 112
—— (epidemic), causation of, Epid. 192
—— (persistent), case of (Kenneth Kellie), Child. 61
—— (recurrent), with splenomegaly, ending in hepatic cirrhosis and ascites (F. P. Weber), Child. 62
—— (splenomegalic), Clin. 131
—— ——, blood-count in, Clin. 131
——, see also *Ulcers* (icteric)
Jaw, cyst of, resulting from traumatism (H. A. T. Fairbank), Odont. 14
—— (lower), extensive necrosis of, case (H. S. Clogg), Child. 30
—— —— —— ——, specimen from case of (H. S. Clogg), Child. 53
—— necrosis of, stomatitis complicated by, Child. 31
—— ——, treatment, local, Child. 31
—— (upper), malformation (unusual) of, plaster casts of (H. Tilley), Laryng. 75
—— ——, removal of, in man, aged 71 (H. J. Davis), Laryng. 86
Jaws, alveolar processes of, and gums, excessive hypertrophy, case of, successful surgical and prosthetic treatment (F. Sleep), Odont. 18-26

Jaws, alveolar processes of, and gums, excessive hypertrophy, see also under *Gums* and *Alveolar* processes of jaws
——, compression of, in operative treatment for cleft palate, instrument for, Surg. 178
Jellett, H., practice as regards removal of appendix in pelvic operations, Obst. 353
Jena, University clinic for hydrotherapy at, Baln. 87
——, University of, courses of lectures on medical hydrology at, Baln. 114, 115
JENKINS, G. J.—Case of deafness in hemiplegia, quoted, Otol. 13
——, labyrinthine vertigo (Menière's symptoms—non-infective) treated by operation, Otol. 116
——, specimens of fractured temporal bone, Otol. 107
——, and CHEATLE, A. H.—Case in which disease in a large sinus tympani caused persistence of discharge after the radical operation; caloric tests applied to the exposed inferior crus of the posterior semicircular canal, Otol. 54
Jensen, outbreaks of milk-borne gastro-enteritis and diarrhœa at Stockholm, reported by, Epid. 98
Jews, children of, amaurotic idiocy (Tay-Sachs disease) occurring among, Path. 147, 148, 167, 174
Johansson and Moritz, case of chloroma, quoted, Path. 68
Johns Hopkins University (Baltimore), teaching of hydrotherapy at, Baln. 116
JOHNSON, A. A. H.—Case of multiple supernumerary teeth preceding a central incisor, Odont. 42
——, discussion on case of absorption of permanent molar due to unerupted premolar, Odont. 38
JOHNSON, A. E.—Case of gangrene of the vagina, probably due to the chemical effect of a pink rubber ring pessary, Obst. 323
——, discussion on fractures in the neighbourhood of joints, Surg. 42
——, transverse fractures of the patella without separation of the fragments, Surg. 69-82, 84
JOHNSTON-LAVIS, H. J.—Discussion on action of aconite on pulse-rate, Therap. 190
——, prevention of formation of calcium oxalate stones, quoted, Baln. 124
Johnstone, R. W., second report on typhoid fever prevalence at Folkestone (1893-1899), Epid. 16, 115, 137
Joints, auscultation of (A. E. Garrod, F.R.S.), Med. 35-39
—— ——, elimination of extraneous sounds, Med. 35, 36
—— ——, how to determine meaning of sounds, Med. 36, 37
——, bony formation in and around, of infective origin, Baln. 24
——, conditions tending to lowered resistance to osteo-arthritis in, Baln. 28
—— (diseased), diagnosis by ausculation, Med. 35-39
—— ——, silent under auscultation when effusion present, Med. 37
——, diseases of, acute, treatment by diathermic current indicated in, Electr. 7
—— ——, influence of unfavourable climatic conditions on, Baln. 28, 29
—— ——, strain as factor in production of, Baln. 28
—— ——, suggested appointment of committee to formulate and describe types of, Baln. 36
——, fractures about, diagnosis by X-rays, Surg. 16-19, 21, 36
—— —— ——, X-ray indications against operative treatment, Surg. 36
—— —— in neighbourhood of (R. Jones), Surg. 1-22, 44
—— —— ——, indications for and against operative treatment, Surg. 39-42
—— —— of, cases of epiphysial injury, Surg. 30
—— —— of, replacement of bearing surfaces, Surg. 28
——, function of, impairment, Surg. 2
—— (stiff), "scrunch" heard in, by auscultation, Med. 38
—— (tuberculous) silent under auscultation, Med. 37
—— ——, synovial crackle in later stages, Med. 37
JOLY, SWIFT.—Discussion on large congenital diverticulum of bladder treated by resection, Surg. 163
JONES, E. LLOYD.—Case of congenital heart disease (? patent ductus arteriosus) (shown by Dr. G. S. Haynes), Child. 191

JONES, F. WOOD.—The delimitation of the rectum and its subdivisions: an anatomical introduction to a discussion on the operative treatment of cancer of the rectum, Surg. 85-98
JONES, H. E.—Discussion on cast of cavity in the left mastoid process filled with cholesteatoma, Otol. 124
——, discussion on eight cases of mastoid operation (Case I), Otol. 126 (Case III), Otol. 129
JONES, H. LEWIS.—Congenital elevation of the shoulder (Sprengel's deformity), Electr. 69
——, discussion on diathermic treatment, Electr. 8
—— —— on new induction coil, Electr. 82
—— —— on a sesamoid bone fractured by direct violence, Electr. 98
—— —— on success or failure in electro-therapy, Electr. 113
——, modification of induced current, quoted, Electr. 36
——, unusual form of contraction of the fingers, Electr. 73
JONES, HUGH.—Discussion on case of fistula of the ampulla, Otol. 76
Jones, Llewyllyn, see *Llewellyn*, R. Ll. Jones
JONES, ROBERT.—On fractures in the neighbourhood of joints: an address introductory to a discussion on the subject, Surg. 1-22, 30, 44
——, traumatic myositis ossificans in neighbourhood of elbow-joint, quoted, Surg. 154
Jonnesco, on pelvic colon, quoted, Surg. 86
JORDAN, A. C.—Discussion on pulsatile swelling in left intercostal spaces, Clin. 24
——, the Roentgen-ray examination of the œsophagus, Electr. 13-30
Jouaust and Soupault, dysmenorrhœa and appendicitis, quoted, Obst. 356
Journal of British and Foreign Health Resorts, founded by Dr. Samuel Hyde, Baln. 65
Jugular foramen, extradural cerebellar abscess tracking through, into neck (H. J. Davis), Otol. 71
—— vein (internal), ulceration of, Child. 204
Julliard, injection of morphine antecedent to ether, Anæsth. 44

Kaltenleutgehen, establishment of first clinic and institute of hydrotherapy at (1862), Baln. 86
——, lectures and demonstrations on hydrotherapy at, Baln. 115
Kammerer, recurrences after operations for femoral hernia, quoted, Surg. 167
Kangra district (Punjab), medicinal spring at (Tarvala Mukhi—"Flame's Mouth"), Baln. 117
Kaposi, acne cachecticorum, quoted, Derm. 36
Kappeler, morphine injection before chloroform inhalation, quoted, Anæsth. 43
——, oral administration of chloral hydrate antecedentally to ether, quoted Anæsth. 44
Karewski, multiple cartilaginous exostoses and facial hemiatrophy, quoted, Med. 3
Karyorrhexis of tumour-cells in case of atypical chloroma, Path. 63, 64, 66
Kastle, investigations as to typhoid carriers, quoted, Epid. 12, 13, 121
Kayser, investigations as to typhoid carriers, quoted, Epid. 4, 15, 19, 107, 108, 122, 123
—— and Forster, isolation of *Bacillus paratyphosus* B from gall-bladder by, Epid. 56
Keay, percentages of primary carcinoma of gall-bladder and biliary passages in cholelithiasis, Path. 94
Kehar Singh (Sirdar Sahib), researches on cretinism, quoted, Med. 8
Keibel, origin of amnio-embryonic cavity, Obst. 321
KEITH, Professor A.—Auriculo-ventricular bundle, quoted, Therap. 97
——, prehistoric example of bodily asymmetry, recorded by, Odont. 109
——, and TWEEDIE, ALEXANDER R.—Ectopia of the pituitary, with other congenital anomalies of the nose, palate, and upper lip, Laryng. 47-52
Keitum, island of, Baln. 19
KELLAS, A.—Report on chemical composition of red and black ring pessaries, Obst. 324-326
KELLIE, KENNETH.—Persistent jaundice, Child. 61
——, two cases of infantilism, Child. 59
KELLOCK, T. H.—Discussion on case of deformity of cervical spine, Child. 105
——, discussion on case of syphilitic osteitis of femur, Child. 103

KELLOCK, T. H.—Discussion on congenital nævoid condition of left thigh, Child. 194
——, discussion on the treatment of cleft palate, Surg. 189
Kelly, H. A., practice as regards removal of appendix in pelvic operations, Obst. 350
——, preservation of endometrium in operations for uterine tumours, Obst. 118, 139
——, and Cullen, T. S., preservation of ovaries in operation for myomata of uterus, Obst. 117, 118
Keloid, development of, in scars in case of trunk lesion of the fifth and sixth nerves (L. McGavin), Clin. 52
——, formation of, after application of diathermic current, Electr. 9, 11
—— (syphilitic), in man, aged 47 (G. W. Dawson), Derm. 3
Keloids forming on cicatrices resulting from ulcerating syphilitic lesions (J. M. H. MacLeod), Derm. 74
KELSON, W. H.—Brain of a patient who died from cerebellar abscess, Otol. 40
——, case of nasopharyngeal fibrous polypus in a man, aged 21, Laryng. 60
——, discussion on case of infiltration of both auricles, Otol. 38
—— —— —— of ulceration on left tonsil, Laryng. 75
—— —— on eight cases of mastoid operation (Case III), Otol. 129
—— —— on treatment of frontal sinus discharge, Laryng. 148
——, lupus of larynx in woman, Laryng. 35
——, microscopical section of cyst of tonsil, Laryng. 118
——, new form of burr for use in radical antral operation by intranasal route, Laryng. 106
——, growth of unknown nature in the typanum, Otol. 77
——, tumour of right side of neck involving pharynx, in woman, aged 30, Laryng. 99
Kelvin, Lord, on construction of the earth, quoted, Path. 143
Kelynack, incidence of gall-stones, quoted, Path. 88
Kenwood, Professor H. R., outbreak of milk-borne sore throat at Finchley, Epid. 90, 94
Keratitis due to oral sepsis, Odont. 34
—— (interstitial) accompanying congenital syphilitic deafness, Otol. 4, 6, 8, 19
Keratosis of finger, case of (A. W. Williams), Derm. 32
—— (gonorrhœal), of hands and feet (A. W. Williams), Derm. 12
Kernig's sign in amaurotic idiocy (Tay-Sachs disease), Path. 154, 160, 161
Kerr, J. Munro, indications for craniotomy, quoted, Obst. 166
——, justifiability of craniotomy, Obst. 214
——, practice as regards removal of appendix in pelvic operations, Obst. 353
Kholmogoroff, view as to suitability of extraperitoneal Cæsarean section, Obst. 165
KIDD, FRANK.—Case for diagnosis: imperfect osteogenesis (?), Clin. 106
——, congenital deformity of femur: absence of the upper epiphysis and of half the shaft of the left femur, Clin. 158
——, discussion on rigidity of the spine, Clin. 150
—— —— on specimens illustrating the removal of foreign bodies from the air-passages and the œsophagus, Clin. 154
——, hyperplastic tuberculosis of colon, quoted, Path. 240, 241
Kidd, F. W., practice as regards removal of appendix in pelvic operations, Obst. 353
KIDD, PERCY.—Discussion on absence of abdominal respiratory movement in pericarditis, Med. 46
Kidneys, cystic disease of (J. Fawcett), Clin. 151
——, disease of (chronic), and polyuria, associated with infantilism, case of (H. M. Fletcher), Child. 75
——, incision and drainage of, in treatment of acute suppression of urine (W. Harris and W. H. Clayton-Greene), Clin. 161
——, sclerosis of, due to alcohol, Med. 79
—— ——, leading to high blood-pressure, Med. 79
——, skiagraphy of, Electr. 90, 91
Killian, scarlet fever causing malignant forms of inflammation in sinuses, reported by, Laryng. 32

Killian's bridge operation in chronic frontal sinus suppuration, Laryng. 134, 135, 140, 143, 146, 147

—— —— —— —— —— —— ——, case illustrating, Laryng. 123

—— —— —— —— —— —— ——, risks of, Laryng. 140

—— —— —— —— —— —— ——, summary of cases treated by, Laryng. 136, 137, 138

—— —— —— in treatment of multi-sinusitis, case illustrating (I. Moore), Laryng. 124, 145

—— —— —— —— of unilateral pansinusitis, cases illustrating (StC. Thomson), Laryng. 123, 124

Kilroe, Laurence, and Moore, F. Craven.—The use of belladonna in dyspepsia, Med. 23-34

Kilworth camp, outbreak of typhoid fever at, Epid. 109

Kinetoplasm, Path. 175

——, process of, Path. 175

King, Preston.—Discussion on balneological treatment of neurasthenia, Baln. 43

Kingdon, E. C., case of amaurotic idiocy (Tay-Sachs disease), quoted, Path. 168

——, and Russell, J. S. Risien, infantile cerebral degeneration with symmetrical changes at the macula, quoted, Path. 169

Kionka, Professor, lecturer on balneology at Jena, Baln. 115

Kirchner and Baratoux, syphilitic endarteritis in blood-vessels of mucous membrane of tympanic cavity, case quoted, Otol. 18

Kirk, E. C., case of multiple supernumerary teeth preceding a central incisor, quoted, Odont. 45

Kjeldahl's process, estimation of total nitrogen in urine by, Baln. 47

Klein, subsequent recovery of diphtheria bacillus at site of inoculation in cows, reported by, Epid. 85

——, and Noble Smith, "Atlas of Histology," quoted, Obst. 285

Klinger, carriers of typhoid fever, quoted, Epid. 5, 123

——, period of excretion of typhoid bacillus by carriers, Epid. 3

——, statistics of typhoid fever, quoted, Epid. 10, 11

Klumpke-paralysis, see *Paralysis* (Klumpke's)

Knapp, condylomata of external ear, quoted, Otol. 11

——, division of posterior spinal nerve-roots for pain, Surg. 204

Knapp, P. C.—Notes of a case of hemiatrophy from sclerodermia, Neur. 28

Knee-cage, hinged, in treatment of avulsion of tubercle of tibia, Surg. 18

Knee-joint, amputation through, for benign cyst of tibia, Clin. 134

——, auscultation of, Med. 36

——, escape of synovial fluid from, in relation to ossific process, Surg. 140

——, fractures about, treatment, Surg. 16

——, gummatous infiltration about, in congenital syphilitic deafness, Otol. 8, 20

——, osteo-arthritis of, Baln. 29

—— ——, following tuberculous disease of same joints, Baln. 24

—— ——, formation of osteophytes in, Baln. 29

—— ——, incipient, abnormal sounds heard under auscultation, Med. 37

——, tuberculous disease of, followed by osteo-arthritis of same joint, Baln. 24

—— —— ——, old healed, with increase in length of limb (O. L. Addison), Child. 39

Knee-splint (Thomas's), Surg. 15

Knife, avoidance, except for surface incisions in treatment of cancer by diathermic currents, Electr. 11

Knox, R.—Demonstration of skiagrams, Electr. 76

Knuckle-joints, swollen and distorted, among labourers and charwomen, cause of, Baln. 28

Knuthsen, L. F. (shown by G. W. Dawson).—Case of purpura, Derm. 60

Koch, R., "Epidemiologie der Tuberkulose," quoted, Epid. 49

——, on phthisical disposition, quoted, Epid. 160

——, on portals of entry in tuberculosis, quoted, Child. 205, 227, 228

——, postulates of, compliance with, Epid 70, 71

Kocher's operation for radical cure of femoral hernia, Surg. 166, 167
Kohler, multiple cartilaginous exostoses and partial gigantism, quoted, Med. 3
Koller, case of amaurotic idiocy (Tay-Sachs disease); child alive at 4 years of age, Path. 168
Königsberg, University of, courses of lectures on medical hydrology at, Baln. 114
Koplik's spots in diagnosis of measles, Child. 153, 174, 180
von Koranyi, excess of red blood-corpuscles in cases of splenomegalic polycythæmia, Clin. 86
Korff, scopolamine and morphine as an anæsthetic, alluded to, Anæsth. 44
Kossel, search for typhoid carriers by, Epid. 121
Krahmer, earliest review of balneological literature, Baln. 75
Kraske, trans-sacral method of operation for rectal cancer, Surg. 99, 104, 108, 110, 112, 113, 116
Kraurosis and leukoplakic vulvitis, communications on (1909-1910) summarized, Obst. 4
Krause, epidemic of poliomyelitis in Westphalia (1908), Epid. 207
Kreatinin, estimation by Folin's colorimetric process, Baln. 47
——, excretion of, before and after taking sulphur water, Baln. 52, 53
Kronecker, experimental work on auriculo-ventricular bundle, Therap. 98
Krönig, anaerobic germs in puerperal infection, Obst. 167
——, correlation of appendicitis and adnexal disease, quoted, Obst. 348
——, injection of veronal before chloroform inhalation, Anæsth. 44
——, removal of normal appendix in cœliotomy, Obst. 348
Kryger, multiple cartilaginous exostoses and myositis ossificans progressiva, quoted, Med. 3
Kuhnt's operation in chronic frontal sinus suppuration, Laryng. 134, 143
Kussmaul, Professor, ignorance of hydrotherapy in Germany, quoted, Baln. 114
Küstner, primary mortality from vaginal hysterectomy, stated by, Obst. 101
Küttner, Professor, and Foerster, Professor O., division of posterior nerve-roots of spinal segments for gastric crises of tabes, quoted, Surg. 210

Laboratories (physiological), installation of string galvanometer in, a necessity, Med. 98
Laborde, injection of narceine before chloroform inhalation, Anæsth. 44
Labour, abdominal ovariotomy for removal of a dermoid tumour of the right ovary during (G. Blacker), Obst. 250
—— after "utriculoplasty" for uterine hæmorrhage (V. Bonney), Obst. 272
——, bacteriology of amniotic cavity during, after rupture of membranes, Obst. 169
—— —— of vagina during, Obst. 169
——, complicated by cancer, treatment, Obst. 190, 191
—— —— by fibroids, treatment, Obst. 190, 191, 204
——, ergot in, Therap. 124
—— ——, effect of medium chloroform anæsthesia on action, Therap. 132, 138
—— —— —— of opium on ecbolic action, Therap. 130-132
—— ——, general effect of, Therap. 124
—— ——, so-called disadvantages of use, Therap. 133-137
—— (neglected), method of treatment, Obst. 190
—— (obstructed), foresight of, important, Obst. 189-208
——, ovarian tumour obstructing delivery and fixed by suction in the pelvis (J. D. Malcolm), Obst. 253
——, ovariotomy during, cases recorded, Obst. 256-259
—— (premature), induction of, Obst. 185, 201
—— —— ——, late, Obst. 216
—— (protracted), with contracted pelvis, treatment by Cæsarean section and alternative operations, Obst. 161
—— —— —— —— —— —— —— and alternative operations in various types of suspected cases, Obst. 172
——, tumours obstructing Cæsarean section in, Obst. 252, 255

Labour pain, constitution of, Therap. 120
—— ——, frequency and duration of, Therap. 121
La Bourboule, hypodermic administration of arsenical waters of, Baln. 108
Labourers, cause of swollen and distorted knuckle-joints among, Baln. 28
Labyrinth, anæmia of, Otol. 123
—— (bony), sequestration of, in boy, aged 4 (F. H. Westmacott), Otol. 123
——, increased tension in, Otol. 115, 119
——, suppuration in, facial paralysis: complete "bridge" operation—recovery (W. Milligan), Otol. 63
—— ——, following fracture of skull, Otol. 89
——, tertiary syphilis affecting (probably), Otol. 14
Labyrinthine concussion, Otol. 103
Labyrinthitis, infective, chronic, case of (C. E. West), Otol. 39
—— (syphilitic), Otol. 20
Lack, H. Lambert.—Discussion on case of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 103
—— (?) lupus erythematosus of mucous membrane of cheeks of over twelve months standing, Laryng. 104
Lacorbière, "Traité du Froid" (1839), Baln. 87
Lactic acid, sterilization of mouth by weak solution of, Odont. 9, 10
—— —— bacillus inoculation in Cæsarean section, Obst. 203, 211
—— —— ——, possible production of dental caries by, Odont. 8, 9
Lacto-farinaceous diet, restriction to, effects of, Baln. 56
Lactose, *Bacillus coli* group fermenting, Epid. 125
——, fermentation by culture from pseudo-typhoid organism, Path. 43
—— —— by *Bacillus typhosus*, Epid. 70, 142
—— —— —— —— not found possible, Path. 51, 52
Laennec, introduction of stethoscope by, Med. 150
——, on tuberculous contagion, quoted, Epid. 163
Laidlaw, P. P.—Discussion on value of rectal feeding, Therap. 75
Lamarckism, chief feature of, Path. 280, 304
——, function determining anatomical variation, essential doctrine of, Path. 305
—— in relation to callosities (S. G. Shattock), Path. 279-312
—— inadequate to explain evolutionary phenomena, Path. 306
Lamb, Major G., and Greenwood, M., jun., seasonal prevalence of plague, quoted, Epid. 225
Lameris and von Harrevelt, outbreak of milk-borne diarrhœa in hospital at Stockholm, Epid. 98
Laminaria tent in dilatation of cervix before operation, Obst. 309
Laminectomy, best method of, in division of posterior spinal nerve-roots, Surg. 220
Landsteiner, experimental transmissibility of poliomyelitis, Epid. 199
Lane, W. Arbuthnot.—Treatment of cleft palate: an address introductory to a discussion on the subject, Surg. 169; (reply), Surg. 198
—— treatment of fractures, alluded to, Surg. 1, 31, 34
Lane's plates in fractures, Surg. 10
Lange, cases not regarded as belonging to Werdnig-Hoffmann type of progressive spinal muscular atrophy, Neur. 73
——, differential diagnosis of progressive spinal muscular atrophy of infants and young children, Neur. 73
Langenbeck's method of operating on cleft palate, Surg. 173, 175, 176, 177, 180, 181, 184, 189, 190
Langmead, Frederick.—Case of achondroplasia, Child. 107
—— —— of malformation of the heart, Child. 39
—— —— of myopathy (? affection of facial muscles), Child. 108
—— —— of rheumatoid arthritis, Child. 33
——, discussion on case of cerebral athetotic diplegia, Child. 78

LANGMEAD, FREDERICK.—Discussion on case of severe tuberculous peritonitis cured by laparotomy, Child. 187

——, discussion on chronic (recurrent) tetany, Child. 17

—— —— on congenital heart disease without murmur, and with a family history of congenital cyanosis, Child. 160

—— —— on specimens from case of "delayed chloroform poisoning," Child. 84

—— —— on two cases of infantilism, Child. 60

——, sclerema neonatorum, with myositis fibrosa, Child. 5

Lanz's point in location of base of appendix, Obst. 354

Laparotomy curing case of severe tuberculous peritonitis (P. Lockhart Mummery), Child. 186

Laryngeal symptoms in case of early myasthenia gravis (S. Hastings), Laryng. 39

Laryngectomy, total, instrument for patient's use after, Laryng. 56

Laryngitis, chronic, case of (W. Howarth), Laryng. 108

—— (tuberculous), with bilateral tuberculous disease of tonsils (C. Graham), Laryng. 67

Laryngology, medical education in relation to, Laryng. 1-5

——, Semon Lectureship in, Laryng. 149, 150

Laryngo-otology, degrees in, advocated, Laryng. 4

Laryngoscopy, hypodermic injection of scopolomorphine before inhalation anæsthesia for, Anæsth. 52

Larynx, cancer in, excess of mechanical strain as cause of (R. H. Scanes Spicer), Laryng. 19-23

—— —— of, case of (P. Watson-Williams), Laryng. 93

——, disease of, in man, aged 45, probably tuberculous, but presenting appearance of epithelioma (W. J. Horne), Laryng. 117

——, epithelioma of, further notes on case of (P. Watson-Williams), Laryng. 153

—— (equine), specimens of, and records of cases illustrating results of "ventricle-stripping" operation for relief of "whistling" and "roaring" in horses, demonstrations of instruments (F. Hobday), Laryng. 87

——, growth (large) in, case of (H. W. FitzGerald Powell), Laryng. 84

——, lower part of pharynx, and upper end of œsophagus, excision of, for malignant disease (squamous-celled carcinoma), subsequent report on case of (A. Evans), Clin. 142

——, lupus of, in woman (W. H. Kelson), Laryng. 35

——, neoplasm (malignant) of, case of (P. Watson-Williams), Laryng. 16

——, nose, and lungs, chronic tuberculosis of, case of (C. A. Parker), Laryng. 64

—— and œsophagus, cancer (party-wall) of, case of (L. H. Pegler), Laryng. 101

——, papilloma of, in child (D. McKenzie), Laryng. 80

——, posterior margin of, and œsophagus, extension of cancer of deep pharynx (W. Hill), Laryng. 44

——, soft palate, and pharynx, tuberculosis of, case of (W. G. Howarth), Laryng. 66

——, stenosis (complete) of, from epithelioma (StC. Thomson), Laryng. 119

——, tuberculous ulceration of, case of (L. H. Pegler), Laryng. 78

——, see also *Tuberculosis* (laryngeal)

Lasègue's phenomenon, Neur. 7

Lateral sinus, injury to, during operation, Otol. 85

—— —— thrombosis following acute otitis media in a boy; operation; recovery (W. J. Chichele Nourse), Otol. 56

—— —— —— (streptococcal), with early pulmonary metastasia; recovery (S. Scott), Otol. 30

LATHAM, A., and HUNT, E. L.—A case of malignant or infective endocarditis treated with an autogenous vaccine administered by the mouth, showing the disappearance of the infecting organisms from the blood within twelve months from the commencement of treatment, together with marked improvement in the clinical symptoms, Clin. 14-21

Laudanoscine, oxidation of, in preparation of lodal, Med. 71

Lausanne, University of, lectures on balneology, climatology and hydrotherapy at, Baln. 116

Lavage (gastric) and intragastric electrical treatment in treatment of functional dyspepsia (obstinate third stage with dilatation), Electr. 36-38

—— ——, necessity for, Electr. 45

Lavage (gastric) with electricity in treatment of ulcer of stomach, Electr. 41, 42
—— —— with static electricity in treatment of dilatation of stomach following operations or shock, Electr. 41
—— (intranasal) in chronic frontal sinus suppuration, Laryng. 129, 138, 142, 144
Lavage-tube for gastric lavage, Electr. 37
Law, Edward.—Report on the fatal termination of case of deafness and discomfort in the right ear as early symptoms in a case of epithelioma originating near the right Eustachian tube, Otol. 67
Läwen, cartilaginous exostoses and multiple enchondromata, quoted, Med. 3
Lawrence, T. W. P., on pseudo-hermaphroditism, quoted, Obst. 62
——, and Blacker, G. F., unilateral hermaphroditism, quoted, Obst. 109
Lea, Arnold W. W.—Discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 187
——, micro-organisms causing puerperal infection, recorded by, Obst. 167
Lead-poisoning, symptoms of, in horses and cattle, Laryng. 91
Leathem, A. N.—Determination of nature of pathogenic micro-organisms on uterine swabbings, Obst. 171, 180
——, discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 206
Le Brocq on local anæsthetics recommended as substitutes for cocaine, Therap. 57
Lecithin, effect of arsenic on, in protozoa, Path. 2
Le Couquet, Baln. 18
Lediard, H. A.—Benign cyst of the tibia, Clin. 132
Ledingham, J. C. G.—Bacteriological examination of fæces of typhoid carriers, quoted, Epid. 118, 119, 120
——, discussion on typhoid carriers and contact infection, Epid. 133
——, report of, to Local Government Board on carriers of typhoid fever, Epid. 107
——, and Firth, A. C. D.—Atypical chloroma, Path. 60-69
Leduc, Professor, on osmotic growths, quoted, Electr. 94
Lee, P. G.—Case of congenital heart disease, Child. 26
Leg, gangrene of, following diphtheria (J. D. Rolleston), Child. 10
—— and loin, left, angiomata of (E. I. Spriggs and J. B. de W. Molony), Child. 85
——, icteric ulcers of legs in pernicious anæmia (H. Batty Shaw), Clin. 110
——, obstinate callous ulcers on legs with sclerodermia, greatly improving under mercurial inunction (A. W. Williams), Derm. 13
Legg, J. Wickham, hæmophilia and osteo-arthritis, quoted, Baln. 24
Lehndorff, cases of chloroma, quoted, Path. 67
Leichtenstern, on ætiology of consumption, quoted, Epid. 175
Leipzig University, clinic for hydro-therapy at, Baln. 87
—— ——, lectures on hydrotherapy at, Baln. 115
Leitch, A.—On the pathological bases of operations for cancer of the uterus, Obst. 69-78
——, pathology of ureters in cancer of uterus, quoted, Obst. 78
Leith, mixed outbreak of milk-borne diphtheria and septic sore throat at, Epid. 96
Lemmon's biscuit powder, Child. 136
Lentz, paratyphoid carriers, quoted, Epid. 56
——, period of excretion of typhoid bacillus by carriers, stated by, Epid. 3
Léopold, presence of gonorrhœea contra-indication to classical Cæsarean section, Obst. 169
Leprosy, case of (H. J. Davis), Laryng. 25
——, commencing (case for diagnosis) (E. G. Graham Little), Derm. 62, 63
—— (maculo-anæsthetic), case of (J. M. H. MacLeod), Derm. 8
——, treatment by Chaulmoogra oil, Laryng. 27
—— —— by nastin, Laryng. 27
—— —— in "hot-houses," Baln. 85
——, two cases of (J. H. Sequeira), Derm. 109
—— with involvement of external ears, palate, pharynx, &c., case of (G. N. Biggs), Otol. 120

Leprosy, with involvement of external ears, palate, pharynx, &c., case of, treatment adopted, Otol. 120

Léri, ossification in spondylitis, quoted, Baln. 32

——, spondylose rhizomélique, quoted, Neur. 41. 44. 45

Lesage, mortality from measles at Hôpital Hérold, Paris, reported by, Child. 174

Lethargy in human trypanosomiasis caused by perivascularitis, Path. 34-37

Leube, absorption of meat and pancreas by rectum. Therap. 64

Leuchs, differentiation of *Bacillus typhosus* from *Bacillus paratyphosus*. Path. 252

Leucoderma syphiliticum, case of (E. G. Graham Little), Derm. 102

Leucorrhœa in chronic endometritis, results of treatment by curetting, Obst. 44, 45

Leukæmia, connexion of chloroma with, Path. 68. 69

Leukerbad, prolonged subthermal baths at, Baln. 97

Leukoplakia buccalis in relation to dental troubles, Odont. 79

Leukosarcomatosis, connexion of chloroma with, Path. 69

Levaditi, experimental work on transmissibility of poliomyelitis, Epid. 199

Levatores ani muscles, Surg. 95

Levertin, A., lecturer on balneology at University of Stockholm in 1880, Baln. 116

Levy, A. G.—Asphyxia under chloroform, Path. 205-222

Lewers, A. H. N.—After-history of a case of fibroid tumour of the uterus complicating pregnancy, Obst. 307

—— discussion on case of gangrene of vagina probably due to chemical effect of pink rubber ring pessary, Obst. 326

—— —— on indications for, and technique of, Cæsarean section and its alternatives, Obst. 192

—— —— on rupture of gravid uterus, Obst. 154

—— —— subtotal hysterectomy for fibromyoma uteri, Obst. 144

——, interstitial gestation sac of between three and four months' development removed unruptured with the body of the uterus by abdominal hysterectomy, Obst. 154

——, sarcoma of the vulva, Obst. 64

Lewis, experimental work on transmissibility of poliomyelitis, Epid. 199

Lewis, Percy G.—The influence of calcareous (hard) drinking water in health and disease, Baln. 119-128

Lewis, T.—Discussion on excitation and section of the auriculo-ventricular bundle, Therap. 111

——, the electrocardiographic method and its relationship to clinical medicine, Med. 81-100, 151

von Leyden, Professor, on physical methods of treatment, quoted, Baln. 71

Lice, transmission of typhus fever by, Epid. 225, 226

—— —— —— ——, experimental, Epid. 225

Lichen annularis in man, aged 40 (G. W. Dawson), Derm. 3

—— planus in relation to dental troubles, Odont. 79

—— —— of unusual distribution, case of (J. H. Sequeira), Derm. 80

—— scrofulosorum, case of (Haldin Davis), Derm. 19

—— ——, experimental production of tuberculosis by inoculation of material from, Derm. 49

—— —— imitating psoriasis (F. P. Weber), Derm. 93

—— —— and phthisis with atrophodermia striata et maculata (H. G. Adamson), Derm. 1

—— spinulosus, case of (J. L. Bunch), Child. 86

—— variegatus, synonym for parakeratosis variegata, Derm. 31

Lichtenthal, Pietro, "Idrologia medica" (1838), Baln. 79

Liddell, J.—Discussion on British health resorts for foreign invalids, Baln. 21

Liebreich, R., theory to account for facial asymmetry, Odont. 105-108

Liefmann and Nieter, proportion of typhoid carriers in asylums, given by, Epid. 119, 120

Lieven, W. A.—Demonstration of instruments for the intravenous injection of salvarsan ("606"), Laryng. 45

Life insurance in cases of adolescent albuminuria, Med. 115, 137

Ligament, see *Cooper's*, *Gimbernat's*, *Poupart's* ligaments

Ligaments, see *Broad* ligaments ; *Sacro-uterine* ligaments
Light head bath (Brünings's) in treatment of acute frontal sinusitis, Laryng. 127, 128, 143
Lightning-stroke causing rupture of membrana tympani, Otol. 84, 85
Limbs, deformity of, in amaurotic idiocy (Tay-Sachs disease), Path. 150, 151
Lime, carbonate of, in mineral waters beneficial to gout, Baln. 123
Lime salts in bread not harmful to teeth, Odont. 97, 98
—— ——, see also *Calcium salts*
Limerick, severe outbreak of food-poisoning following consumption of beef infected with Gaertner bacillus at, Epid. 68
Lincoln, outbreaks of milk-borne sore throat at, Epid. 92
——, typhoid fever at (1881-1909), Epid. 7
—— —— ——, epidemic at (1905), cases reported subsequently as an indication of "carrier" transmission, Epid. 8, 9
—— —— —— ——, secondary infections, Epid. 132
Lindner and Mally, epidemic of poliomyelitis in Steyr (1908), reported by, Epid. 209
Lingual thyroid ; thyroid gland in neck not palpable (W. G. Spencer), Laryng. 13
Linossier, argument against importance of typhoid carrier as infective agency, Epid. 19
Lip, elephantiasis of, case of (J. H. Sequeira), Derm. 56
—— (lower), whole of, and entire skin of chin, epithelioma involving, showing close clinical resemblance to actinomycosis (A. W. Williams), Derm. 14
—— (upper), congenital anomalies of (A. R. Tweedie and A. Keith), Laryng. 47-54
—— ——, tumour of, report on (J. D. Rolleston and N. S. Macnaughtan), Clin. 114
Lipomata, diffuse, sex-incidence, Clin. 7
Lipomatosis, symmetrical, case of (H. D. Rolleston), Clin. 1
—— ——, relation to adiposis dolorosa and thyroid insufficiency, Clin. 7
Liquor amnii, bacteriological examination in suspect cases, Obst. 216
—— ——, good culture medium for pathogenic bacteria, Obst. 170, 195, 196
—— ——, infection of, Obst. 195, 209, 215
—— —— —— distinct from infection of patient, Obst. 198
—— —— ——, fatal consequences of, Obst. 196
——, pancreaticus, predigestion of rectal feed by, Therap. 63
Lister Institute, outbreak of typhoid fever at, Epid. 136
LITTLE, E. G. GRAHAM.—Acne scrofulosorum in a girl, Derm. 35
——, cases for diagnosis, Derm. 52, 60, 63, 73, 145
—— —— of acute lupus erythematosus, Derm. 50
—— —— of granuloma annulare, Derm. 24
—— —— of leucoderma syphiliticum, Derm. 102
—— —— of nævus pigmentosus, verrucosus et pilosus, Derm. 64
—— —— of pityriasis rubra pilaris, Derm. 23
——, discussion on case for diagnosis, Derm. 91, 95
—— —— —— of acute lupus erythematosus, Derm. 92
—— —— —— of cheilitis exfoliativa, Derm. 84
—— —— —— of lichen scrofulosorum, Derm. 20
—— —— —— non-ulcerating tuberculide (?) Boeck's miliary sarcoid, following lupus vulgaris, Derm. 68
—— —— —— of pemphigus foliaceus, Derm. 17
—— —— —— of pityriasis rubra pilaris, Derm. 100
—— —— —— of unilateral pigmentary nævus, Derm. 60
—— —— —— showing keloids forming on cicatrices resulting from ulcerating syphilitic lesions, Derm. 75
—— —— on a series of ringworm cultures, illustrating the eczematoid ringworm of the hands and feet, Derm. 112
——, disseminated tuberculous nodules of skin, Derm. 48
——, erythema ab igne, Derm. 88
——, folliculitis decalvans in a young lady, aged 19, Derm. 144

LITTLE, E. G. GRAHAM.—Herpes zoster in a man, aged 50, Derm. 87
——, macular atrophy associated with lupus erythematosus, Derm. 143
——, nævi of peculiar appearance in girl, aged 12, Derm. 74
——, nævo-lipomata in an infant, Derm. 23
——, sarcomatosis cutis of the "Perrin" type in a man, aged 25, Derm. 22
——, two cases of nævus unius lateris, Derm. 64
—— ? urticaria pigmentosa in an adult, Derm. 104
——, urticaria pigmentosa of the macular type in an infant, Derm. 103
Little, W. G., solubility of salts of uric acid, quoted, Baln. 122
Livedo reticularis or erythema ab igne with pigmentation (H. G. Adamson), Derm. 46
Liveing, R., local inflammation preceding macular atrophy, quoted, Derm. 123
Liver, cirrhosis of, and ascites, following splenomegaly with recurrent jaundice (F. P. Weber), Child. 62
——, enlargement of, case of (T. R. Whipham), Child. 54
——, microscopical appearances at necropsy of case of congenital obliteration of bile-ducts, Child. 45
Llandrindod Wells, chalybeate spring at, tonic effect in neurasthenia, Baln. 40
—— ——, elevation in relation to treatment of circulatory disorders at, Baln. 69
—— ——, mineral waters at, Baln. 15
—— ——, neurasthenia benefited at, Baln. 42
—— —— —— —— by hydrological treatment, Baln. 39
—— ——, saline waters of, action, Baln. 39, 40
Llewellyn, R. Ll. Jones, association between rheumatoid arthritis and Graves's disease, quoted, Child. 36
—— bony formation of infective origin in and around joints, quoted, Baln. 24
Lobule of ear, "cleft," Otol. 81
——, deformities of, from wearing heavy ornaments dependent from, Otol. 81
——, piercing of, deformities resulting from, Otol. 81
Local applications, *minus*, Baln. 92
—— ——, *plus*, Baln. 92, 94
Local Government Board, new order as to notification of disease issued by, Epid. 185, 196, 222
LOCKYER, C.—Discussion on cases of adeno-carcinoma of uterus and ovaries, and of carcinoma of rectum, Obst. 361
——, discussion on operation for cancer of uterus, Obst. 102
Lodal, composition of, Med. 71
——, effect on low blood-pressure, Med. 71
Loeb, parthogenesis in sea-urchins' eggs, recorded by, Path. 175, 192, 193
Logarrhœa, definition of, Neur. 8
Loin and leg, left, angiomata of (E. I. Spriggs and J. B. de W. Molony), Child. 85
Lombard, therapeutic effects of aconite, stated by, Therap. 152
Lombroso, asymmetry as stigma of degeneracy, quoted, Odont. 106
Lommel, excess of red blood-corpuscles in cases of splenomegalic polycythæmia, quoted, Clin. 86
Lommius (1560) on tuberculous contagion, quoted, Epid. 161
London, epidemic cholera more severe in, than in Manchester, Epid. 192
—— (City of), insanitary conditions of, in Elizabethan period, Child. 115
—— —— —— —— —— ——, rendering artificial feeding of infants impossible, Child. 115
—— ——, liberties and out-parishes, infantile mortality in, during reign of Charles II, Child. 117, 118
—— ——, prohibition of new buildings within walls of, by Queen Elizabeth, Child. 115
——, cowhouses in (1850), insanitary condition, Child. 135
——, distribution of cases of poliomyelitis in (1904), Epid. 217, 218, 219
——, hydrotherapy in, Baln. 98
——, milk supply of, in 1850, foul condition of, Child. 134
——, teaching of medical hydrology in, advocated, Baln. 117

London County Council, veterinary inspector's reports on mastitis in cows, Epid. 77
Lordosis, influence on adolescent albuminuria, Med. 126, 140
Löschner, first publication of "Beiträge zür Balneologie" (1845), Baln. 75
Lothiessen, adoption of inguinal method of radical cure of femoral hernia, quoted, Surg. 165, 166, 167
Lotio nigra, see *Black lotion*
Lucas, experimental work on transmissibility of poliomyelitis, Epid. 199
Lucas, R. Clement.—Discussion on after-history of albuminuria in adolescence, Med. 121
——, discussion on congenital nævoid condition of left thigh, Child. 194
—— —— on portals of entry in phthisis, Child. 223
Luciani, experiments on cerebellum, quoted, Neur. 20
Ludwig-Salkowski process, estimation of uric acid by, Baln. 47
Lumbago, treatment by diathermic current indicated in, Electr. 7
Lumbar lymphatic glands, see *Lymphatic glands*, lumbar
Lumbar puncture in amaurotic idiocy (Tay-Sachs disease), Path. 154, 155, 157, 161, 165, 178
Lumbo-sacral disease, with tabes, case of (E. F. Buzzard), Clin. 15
Lumsden, investigations as to typhoid fever carriers, quoted, Epid. 12, 13, 121
Lunatic asylums, incidence of gall-stones shown by post-mortem records at, Path. 88, 89
—— —— —— —— —— —— —— compared with similar records from general hospitals, Path. 88, 89
Lung, cancer of (primary), local prevalence in cobalt mines at Schneeberg, Path. 78
—— —— ——, pneumokoniosis predisposes to, Path. 78
—— —— ——, rarity, Path. 78
——, foreign bodies in, skiagraphy of, Electr. 91
—— (right), beef-bone expelled from, by coughing, Laryng. 97.
Lungs, air-borne bacteria entering, experiments to prove, Child. 207
——, aspiration of bacteria into, during feeding experiments, Child. 212, 213
——, diseases of, cause of unilateral inhibition of action of diaphragm, Med. 40
——, general congestion of, Med. 107
——, infection with tuberculosis by alimentary tract, Child. 221
——, nose, and larynx, chronic tuberculosis of, case of (C. A. Parker), Laryng. 64
——, pneumococcal infection, conception and explanation, Med. 108
——, tumours of, pressure from, causing œsophageal obstruction, Electr. 21, 22
——, see *Artery* (pulmonary)
——, see *Hæmorrhage* (pulmonary)
Lupus erythematosus (acute), cases of (J. H. Sequeira), Derm. 91
—— —— —— (Sir Malcolm Morris and S. E. Dore), Derm. 106
—— —— associated with macular atrophy (E. G. Graham Little), Derm. 143
—— —— —— —— ——, due to exposure to sun's rays, Derm. 144
—— —— (extensive) associated with symmetrical gangrene (G. W. Dawson), Derm. 101
—— —— —— associated with ulceration, case of (J. M. H. MacLeod), Derm. 53
—— —— of mucous membrane of cheeks ? case of (H. Lambert Lack), Laryng. 104
—— —— (nodular), coloured drawing of (G. Pernet), Derm. 108
—— ——, sex-incidence, Derm. 92
—— ——, treatment, Derm. 92
—— —— ——, methods suggested, Derm. 51
—— of larynx, case of (W. H. Kelson), Laryng. 35
—— ——, starting point of tuberculosis, Child. 227
—— ——, very extensive (A. W. Williams), Derm. 96
—— vulgaris, epithelioma upon, case of (G. N. Meachen), Derm. 132
—— —— —— in girl, aged 13 (H. G. Adamson, for C. E. Iredell), Derm. 139
—— ——, followed by non-ulcerating tuberculide (?) Boeck's miliary sarcoid (J. H. Sequeira), Derm. 67
Lymphadenitis (acute), streptococcus from fatal case inoculated into goat, Epid. 85
Lymphangitis, chronic, ? case of infiltration of both auricles in woman, aged 32 (Dan McKenzie), Otol. 37

Lymphangitis, epizootic in horses identical with sporotrichosis, Derm. 115
—— from uterine sepsis, Obst. 73
—— —— —— as cause of so-called "syphilitic stricture of rectum," Obst. 73
Lymphatic gland, large, lying between external iliac vein and obturator nerve, Obst. 75
—— —— on uterine artery, Obst. 102
—— glands, extirpation of, course of common iliac artery compared to axillary region in regard to, Obst. 75
—— ——, histological changes in, in trypanosomiasis, Path. 18-20
—— —— (inguinal), involvement in uterine cancer, Obst. 75, 76
—— —— (inter-iliac), Obst. 73-75
—— —— ——, extirpation in uterine cancer, Obst. 74, 75
—— —— —— connexion with lumbar glands, Obst. 73
—— —— (lumbar), Obst. 73
—— —— (pelvic), distribution shown by dissection of pelvis in case of cancer of uterus, Obst. 74, 75
—— —— ——, removal in abdominal pan-hysterectomy (Wertheim's method), Obst. 90, 91
—— —— ——, tracts of lymphatic vessels from uterus to, contained within broad ligaments, Obst. 76
—— —— permeated by cancer-cells do not coincide with channels marked out by artificial injection, Obst. 76
—— —— seat of latent tuberculous infection, Epid. 34
—— "knot," juxta-cervical, Obst. 106
—— structures, morbid changes in, in human trypanosomiasis, Path. 20, 21
—— vessels, involvement in cancer of rectum, Surg. 107, 121
—— ——, middle tract of Poirier, Obst. 73
—— —— of pelvis, tract of, Obst. 72
—— —— tracts of, from uterus to pelvic glands, contained within broad ligaments, Obst. 76
Lymphoma invading each orbit and causing double proptosis (L. V. Cargill), Clin. 53

Maar, W., and Bie, W., dry residue of blood and blood-serum in case of cyanosis and polycythæmia, amount stated by, Clin. 91
Macacus, ischial callosities in, Path. 288, 290
——, newborn, vertical sections of palm and dorsum of hand, compared, Path. 311
Macalister, Professor A., F.R.S., views on appendiculo-ovarian ligament, Obst. 346
McBurney's method of reduction of dislocation of head of humerus complicating fractures of upper end, Surg. 6
—— point in location of base of appendix, Obst. 354
McCann, F. J.—Discussion on operation for cancer of uterus, Obst. 104
——, discussion on retroflexion of gravid uterus with superimposed fibroid, Obst. 290
McCardie, W. J.—A death due to inspiration of gummatous material from a gumma which had burst during chloroform anæsthesia, Anæsth. 25
——, discussion on hypnotism in relation to surgical anæsthesia, Anæsth. 21
—— —— on nasal reflex during anæsthesia, Anæsth. 33
—— —— on note on case of "Cheyne-Stokes" respiration during anæsthesia, Anæsth. 40
—— —— on use of drugs before inhalation anæsthesia, Anæsth. 54
McCarrison, Captain R., I.M.S., investigations of endemic goitre, quoted, Child. 78, 79
——, researches on cretinism, quoted, Med. 8
Macclesfield, outbreak due to milk at, comprising cases of scarlet fever, shown to be septic sore throat and diphtheria, clinically, Epid. 101
McConkey, epizootics associated with *Bacillus enteritidis*, noted by, Epid. 61
MacCormac, H., post-mortem statistics of uterine cancer, quoted, Obst. 71, 73
McDonagh, J. E. R.—Case of congenital xanthoma (endothelioma), Derm. 76
—— —— of idiopathic atrophy of the skin, Derm. 128
—— —— of syphilitic affection of the eighth nerve, Derm. 147

McDonagh, J. E. R.—Case of syphilitic auto-reinfection, Derm. 129
——, convenient method of giving "606" intravenously, Derm. 36
——, description of lesion and serological examination in case of sporotrichosis, Derm. 118
——, discussion on case showing keloids forming on cicatrices resulting from ulcerated syphilitic lesions, Derm. 75
—— —— —— of (?) granuloma inguinale, Derm. 137
—— —— —— of ringworm of the toe-nails, Derm. 151
—— —— on syphilis in relation to otology, Otol. 22, 23
——, pathological report on case of (?) granuloma inguinale, Derm. 134
——, rare form of syphilitic onychia, Derm. 55
——, three cases illustrating benefit derived from Ehrlich-Hata's "606," Derm. 5
McGavin, L.—Discussion on specimens illustrating removal of foreign bodies from air-passages and the œsophagus, Clin. 155
——, trunk lesion at fifth and sixth nerves; keloid development in scars, Clin. 52
——, varix of right superficial epigastric vein, Clin. 51
McIlroy, Miss A. Louise.—Demonstration on origin of follicle cells of ovary, Obst. 226
——, hæmatosalpinx, with torsion of the tubes, Obst. 112
Mackay, M.—Discussion on diseases of nose and accessory sinuses in relation to affections of eye and orbit, Laryng, 164
Mackenrodt, primary mortality from Wertheim's operation, quoted, Obst. 95
——, ultimate results of Wertheim's operation, quoted, Obst. 98
McKenzie, Dan.—Antrum suppuration, nasal antrostomy followed by blocking of nasal duct, Laryng. 102
——, case of unilateral deafness, probably complete, associated with vertigo, Otol. 60
——, cicatricial obliteration of nasopharynx (hereditary syphilis), Laryng. 104
——, discussion on case for diagnosis, Laryng. 57
—— —— —— of acute inflammation of mastoid treated by Bier's method, Otol. 59
—— —— —— —— vertigo, with repeated falls always to left, Otol. 72
—— —— —— of bilateral tuberculous disease of tonsils, Laryng. 68
—— —— —— of caries and necrosis following mastoiditis due to scarlet fever, Otol. 70
—— —— —— of fistula of ampulla, Otol. 76
—— —— —— of leprosy, Laryng. 27
—— —— —— of persistence of disease in large sinus tympani, Otol. 55
—— —— —— of small tumour of soft palate, Laryng. 13
—— —— —— of temporary deafness and blindness due to intestinal toxæmia, Otol. 123
—— —— on empyema of maxillary antrum in boy, Laryng. 32
—— —— on injuries of ear, Otol. 103
—— —— on labyrinthine vertigo (Menière's symptoms—non-infective), treated by operation, Otol. 119
—— —— on syphilis in relation to otology, Otol. 22
—— —— on three specimens of chronic middle-ear suppuration, Otol. 113
—— —— on treatment of frontal sinus discharge, Laryng. 144
——, infiltration of both auricles (? chronic lymphangitis) in a woman, aged 32, Otol. 37
——, papilloma of larynx in a child, Laryng. 80
——, two specimens of the quadrilateral cartilage (nasal septum) showing fenestræ removed by submucous section, Laryng. 5
Mackenzie, J., effect of digitalis on blood-pressure, quoted, Med. 68
McKerron, R., ovarian tumours obstructing delivery, quoted, Obst. 256
Mackey, L. G. J., association of secondary polycythæmia of congenital heart disease with erythroblastic reaction in bone-marrow, quoted, Clin. 93
McLaren, causal relationship between appendicitis and dysmenorrhœa, stated by, Obst. 356
MacLean, H., report on specimen of ovarian teratoma made by, Obst. 286
MacLeod, J. M. H.—Case of maculo-anæsthetic leprosy, Derm. 8
——, complete albinism in a girl, aged 6, with total absence of pigment in the skin, hair, choroid, and iris, Derm. 7

MacLeod, J. M. H.—Discussion on case of acute lupus erythematosus, Derm. 51, 93
——, discussion on case of acute pityriasis of scalp, Derm. 141
—— —— —— of cheilitis exfoliativa, Derm. 84
—— —— —— for diagnosis, Derm. 43, 63, 90
—— —— —— of (?) granuloma inguinale, Derm. 135
—— —— —— of hyperkeratosis of palms and soles, Derm. 18
—— —— —— of ichthyosis in child treated by thyroid extract, Derm. 73
—— —— —— of lichen scrofulosorum, Derm. 20
——, extensive lupus erythematosus associated with ulceration, Derm. 53
——, icthyosis follicularis in a woman, aged 18, Derm. 131
——, imperfect hair-growth in the scalp of a girl, aged 18 months, Derm. 54
——, keloids forming on the cicatrices, resulting from ulcerating syphilitic lesions, Derm. 74
——, small-spored ringworm of the scalp in a woman, aged 23, Derm. 67
——, specimens of hair from an aboriginal Indian girl, Derm. 149
Macnaughtan, N. S., and Rolleston, J. D.—Familial pigmentary dermo-fibromatosis, Clin. 71
—— ——, report on tumour of the upper lip, Clin. 114
Macnaughton-Jones, H.—Discussion on cases of adeno-carcinoma of uterus and ovaries, and of carcinoma of rectum, Obst. 360
——, discussion on case of fibromyoma uteri causing acute obstruction, Obst. 109
—— —— —— of gangrene of vagina, probably due to chemical effect of pink rubber ring pessary, Obst. 326
—— —— —— of hæmatosalpinx with torsion of the tubes, Obst. 114
—— —— —— of pseudo-hermaphroditism, Obst. 61
—— —— —— of supposed hermaphroditism, Obst. 111
—— —— on cervical glands in the uterus of the fœtus at term, Obst. 285
—— —— on chronic endometritis, Obst. 51
—— —— on demonstration on origin of follicle cells of ovary, Obst. 227
—— —— on demonstration of very young human ovum, Obst. 322
—— —— on hydatid of uterus, Obst. 247
—— —— on indications for, and technique of, Cæsarean section and its alternatives, Obst. 212
—— —— on interstitial gestation sac removed unruptured by abdominal hysterectomy, Obst. 157
—— —— on nature of hæmatocolpos fluid, Obst. 244
—— —— on new operation for the radical treatment of flat pelvis, Obst. 269
—— —— on operation for cancer of uterus, Obst. 101, 105
—— —— on ovarian cyst in child, Obst. 263
—— —— on rectum in gynæcology, Obst. 335
—— —— on retroflexion of gravid uterus with superimposed fibroid, Obst. 290
—— —— on six cases of "utriculoplasty" for uterine hæmorrhage, one of which was followed by pregnancy and labour, Obst. 277
—— —— on specimen of fœtus with congenital absence of sacrum, Obst. 280
—— —— on subtotal hysterectomy for fibromyoma uteri, Obst. 145
—— —— on suppurating ovarian tumour, perforating into small intestine and omentum, successfully removed with affected bowel, omentum, and appendix, Obst. 160
—— —— on use of drugs before inhalation anæsthesia, Anæsth. 50
—— —— on uterine mucosa in menstruation and pregnancy, Obst. 306
——, hydrosalpinx with ovarian and peritoneal cysts, Obst. 221
——, hysterectomy followed by double phlebitis, Obst. 223
——, Presidential Address, "Lessons of a session," Obst. 1-17
—— —— —— (review of work of Obstetrical and Gynæcological Section, 1910-11), Obst. 337-340
——, the relation of the appendix to the causation and surgical treatment of affections of the adnexa, Obst. 341-358
—— (for Professor Schottländer), cases of uterine polypi undergoing malignant degeneration, Obst. 31
—— and Atkins, case of atrichosis, quoted, Path. 302

McPhail and Caverly, epidemic of poliomyelitis in Rutland (Massachusetts) Epid. 200
Macula lutea, hypermetropic astigmatism with change at (J. C. W. Graham), Child. 198
——, symmetrical changes in each eye prominent symptom in amaurotic idiocy (Tay-Sachs disease), Path. 167, 169
McWEENEY, Professor E. J.—Discussion on paratyphoid fever and food-poisoning, Epid. 67
McWILLIAM, ANDREW, and MORRIS, H. J.—Comparative elasticity and internal structure of cast and rolled gold plate, Odont. 57
Magnesium sulphate, tests for presence of, in urine, in the Christie Hospital, Therap. 20
—— ——, value of, in dysentery, Therap. 21
MAHOMED, G.—Discussion on osteo-arthritis and chronic rheumatism, Baln. 36
Maidstone, typhoid fever at (1881-1909), Epid. 7
—— —— —— epidemic (1897), cases reported subsequently as an indication of "carrier" transmission, Epid. 8, 9
—— —— —— ——, secondary infections, Epid. 132
Mair, L. W. Darra, outbreaks of milk-borne sore throat at Lincoln, Epid. 92, 93
Mair, W., acid and gas-forming power of bacillus isolated from urine, Path. 97, 107
MAKINS, G. H., C.B.—Traumatic myositis ossificans, Surg. 133
Mal de coit, or dourine, transmission of, Path. 2, 11
Malaria suspected in first stage of human trypanosomiasis, Path. 4, 13
MALCOLM, J. D.—Discussion on subtotal hysterectomy for fibromyoma uteri, Obst. 144
——, an ovarian tumour obstructing delivery and fixed by suction in the pelvis, Obst. 253
Male, complete dominance of condition of multiple cartilaginous exostoses in, discussed Med. 7, 8
Males, hæmophilia alleged to be limited to, Med. 7
——, incidence of multiple cartilaginous exostoses greater in, Med. 3, 4
—— —— —— —— —— ——, explanation, Med. 7
——, typhoid fever incidence greater among, Epid 11
Malformations, ante-natal (acquired *in utero*) distinguished from congenital, Path. 306
Malignant degeneration of resulting scar-tissue after incised wound of auricle, Otol. 80
—— disease in ear resulting from injury, Otol. 83
—— ——, cases failing to react to X-rays, Electr. 58
—— ——, treatment with radium, Electr. 52
—— —— —— with X-rays, Electr. 52
—— growths originating from prolonged and unscientific exposure to X-rays, Electr. 53
—— papillomatous cysts of both ovaries, removal followed six years later by carcinoma of rectum (M. A. Scharlieb), Obst. 358
—— stricture of œsophagus, case of (P. Watson-Williams), Laryng. 83
—— tumour, infiltrating, on lateral wall of nasopharynx, course and symptoms, Otol. 68, 69
—— ulceration (extensive) of pharynx and tonsils, case of (L. H. Pegler), Laryng. 78
Malingering in form of self-injury to walls of auditory meatus, Otol. 82
—— —— —— —— —— —— and membrana tympani, Otol. 84
Malignancy in ovarian tumours, communications on (1909-10), summarized, Obst. 10
Mallory, output of uric acid in gout, stated by, Baln. 59
Mally and Lindner, epidemic of poliomyelitis in Steyr (1908), reported by, Epid. 209
Malnutrition, prevalence of pulmonary tuberculosis due to, Epid. 48
Malta fever and paratyphoid fever, mixed infections of, Epid. 118
—— —— and typhoid fever, mixed infections of, Epid. 118
—— ——, possibility of spread by cow's milk, Epid. 75
Malthus, on foundling hospitals as best means of stopping growth of population, Child. 130
Malum coxæ senile, Baln. 34
Malvern, hydropathic institution started at, Baln. 84
Mamma as feeding bottle, Child. 134
Man, evidence for auricular fibrillation in, Med. 100
——, ingestion of *Bacillus suipestifer* by, without harmful results, Epid. 58, 59
Manchester, epidemic cholera more severe in, than in St. Marylebone, Epid. 192

Manchester, diseases of air-passages more common in, than in St. Marylebone, Epid. 192, 193
—— measles more prevalent in, than in St. Marylebone, Epid. 192, 193
Manchester and Salford Sanitary Association system of registration of disease established by, Epid. 186, 187
MANDERS, H.—Discussion on diathermic treatment, Electr. 9
——, discussion on treatment of cancer by X-rays and radium, Electr. 66
——, the value of electricity in treatment of some abdominal diseases, Electr. 32-46
Mandrill, hairless area in, Path. 310
——, ischial callosity in, Path. 290
Manganic acid, ions of, beneficial action on gastric ulcer, Electr. 42
Mangiagalli, Professor, practice as regards removal of appendix in pelvic operations, Obst. 353
Mania (acute) complicating epilepsy, successful method of treatment, Baln. 7
Manipulation in treatment of dislocation of head of humerus, Surg. 5
MANN, SYDNEY A.—The chemical examination of the brain in two cases of amaurotic idiocy, and comparison with the normal brain, Path. 195-198
Manometer (Oliver's compressed air) for recording blood-pressure, Med. 51
MANTLE, A.—Discussion on British health resorts for foreign invalids, Baln. 22
——, discussion on pharmacological effects of the strong sulphur water, Baln. 62
MANUEL, A.—Case of reinfection with syphilis after five and a half years, Clin. 30
Marcet, A., F.R.S., prior to C. Schmidt in regarding cerebrospinal fluid as secretion, Med. 153
——, "Various dropsical fluids," quoted, Med. 153
Marchand, on meaning of word "disposition," Epid. 150
Margate, Baln. 17
——, health resort suitable for foreign invalids, Baln. 20
——, rainfall of, compared with that of Borkum, Baln. 19
Marie, ossification in spondylitis, quoted, Baln. 32
Marinesco, process termed "kinetoplasm" by, Path. 175
Marital infection in pulmonary tuberculosis, Epid. 45, 47
Marmalade, see *Bread and marmalade*
MARRIAGE, H. J.—Discussion on case of lateral sinus thrombosis, Otol. 33
——, large tuberculous polyp removed from middle ear of infant, aged 7 months, Otol. 41
Marshall, transplantation of ovaries, quoted, Obst. 116
Martin, Blair, Gram-negative cocci, quoted, Epid. 70
Martin, C., practice as regards removal of appendix in pelvic operations, Obst. 353
MARTIN, C. J.—Discussion on the problem of disposition in pulmonary tuberculosis, Epid. 182
Martin, E., procedure in cases of protracted labour with contracted pelves, aseptic or septic, Obst. 168
Martin, L., mortality from measles at Hôpital Pasteur, Paris, Child. 174
Maryport, epidemic of poliomyelitis at (1910), Epid. 212
Massachusetts, epidemics of poliomyelitis in (1907-08-09), Epid. 206
Massage, early, importance of, in Colles's fracture, Surg. 29, 30
——, early and gentle, in recent fractures about elbow-joint, useful, Surg. 7
—— in conjunction with faradism in treatment of obstinate dyspepsia, Electr. 38; see also *Farado-massage*
—— in neurasthenia, Baln. 41
—— kept up during treatment of fractures about hip-joint, Surg. 15
——, with passive movements, in early stages of fractures about elbow-joint injurious, Surg. 7
Massini, mutation of micro-organisms, quoted, Epid. 124
Mastitis (acute) in cows, causing atrophy of quarters of udders, Epid. 77
—— in cows, Epid. 76
—— ——, association of outbreaks of milk-borne septic conditions with, Epid. 88
—— ——, bacteriology of, Epid. 77-81, 103
—— ——, existence of, as constitutional disease, Epid. 102
—— —— infecting milk, Epid. 76, 99
—— ——, infectious to man when due to *Streptococcus pyogenes*, Epid. 95

Mastitis in cows, pathological investigations upon, Epid. 77-87
—— ——, reports of Veterinary Inspector, London County Council, Epid. 77
—— in goats, caused by inoculation of *Streptococcus mastitidis*, Epid. 80, 85
—— —— not caused by inoculation of *Streptococcus anginosus*, Epid. 80, 85
Mastoid, inflammation (acute) of, treated by Bier's method, case of, (G. C. Cathcart), Otol. 58
—— operation, delayed healing of wound after, in a syphilitic, Otol. 13
—— ——, eight cases of (C. J. Heath), Otol. 125
—— ——, radical bilateral, in case of fistula of ampulla of superior semicircular canal (S. Scott), Otol. 73
—— process (left), cast of cavity in, of woman, aged 75. filled with cholesteatoma—smooth polished surfaces (F. H. Westmacott), Otol. 124
Mastoiditis followed by caries and necrosis of temporal parietal and occipital bones, due to scarlet fever, in girl, aged 5; operation; recovery (H. J. Davis), Otol. 70
——, suppurative (chronic), with abscesses in left temporo-sphenoidal and frontal lobes (P. Watson-Williams), Otol. 134
Matlock, hydropathic institution started at, Baln. 84
Matter, free motility of particles of, essential to metabolism. Baln, 107
Mavor, F. F., and Burness, A. G., experiments as to action of aconite, made by, Therap. 157
Maxilla with exostoses, model of (P. J. Proud), Odont. 41
Maxwell, R. Drummond.—Clinical history of case of involution of ovarian cyst, associated with vesicular mole, Obst. 219
——, discussion on indications for and technique of Caesarean section and its alternatives, Obst. 197
—— —— on new operation for radical treatment of flat pelvis, Obst. 269
—— —— on retroflexion of gravid uterus with superimposed fibroid, Obst. 290
——, microscopical examination of specimen of sarcoma of vulva, Obst. 66
——, prophylactic irrigation of amniotic cavity in doubtful cases of infection, where membranes are ruptured, carried out by, Obst. 173, 185, 186, 198
——, thyroid tissue in ovarian embryoma, Obst. 217
May, Otto.—Discussion on resection of posterior spinal nerve-roots, Surg. 247, 254
Mayer, case of typhoid carrier, quoted, Epid. 108, 122
——, proportion of typhoid carriers, quoted, Epid. 21, 122
Mayo, mortality percentage after operations for rectal cancer, Surg. 111
——, practice as regards removal of appendix in pelvic operations, Obst. 351
Meachen, G. N.—Case of epithelioma upon lupus vulgaris, Derm. 132
——, case of sycosiform syphilide, Derm. 105
Mead, Richard, cold and tepid applications employed by, Baln. 83
Measles, complicated by inflammatory croup, Epid. 192, 223
—— —— by pneumonia, Epid. 192
—— —— ——, and followed by pulmonary tuberculosis, Epid. 48
——, diagnosis by Koplik's spots, Child. 153, 174, 180
——, disinfection after, Child. 175
——, metastatic ophthalmitis of right eye following, Path. 245, 246, 249
——, more prevalent in Manchester than in St. Marylebone, Epid. 191, 192
——, mortality from, at hospitals of Paris, Child. 174
——, notification of, Child. 173
——, prevalence of, relation to weather, Epid. 190
—— —— at Wellington College, Child. 175
——, transmission from patient to patient by nurse, Child. 180
——, treatment and prevention (R. Milne), Child. 142-154
—— —— by inunction, discussion on, Child. 173-184
—— —— ——, with eucalyptus oil (R. Milne), Child. 142-154
—— —— —— —— ——, Child. 177, 183, 184
——, varying fatality of, Epid. 192
Meat. *Bacillus paratyphosus* B not found in, Epid. 55

Meat (canned), bacteria in, resistant to heat, Epid. 66
—— ——, "blowing" of tins due to presence of *Bacillus cadaveris*, Epid. 67
—— —— toxins cause of food-poisoning following eating of, Epid. 65, 66
——, chewing by nurse before giving to children, old practice of, Child. 119, 120
—— infected with *Bacillus enteritidis* (Gaertner) causing food-poisoning. Epid 74, 102
—— and pancreas, absorption by rectum, Therap. 64
Meat diet delays healing of acute ulcer of stomach, Path. 57, 58
—— —— in low blood-pressure, Med. 74
Meatus and auricle, congenital malformation of (A. H. Cheatle), Otol. 25
——, auditory, foreign bodies in, Otol. 96
—— —— —— ——, injuries from extraction of, Otol. 81, 82
—— —— —— —— —— from introduction of, Otol. 81, 82
—— —— —— —— remaining in without injury to ear, Otol. 82
—— —— —— ——, death from tetanus after failure to extract, Otol. 82
—— —— —— —— (supposed), attempt to extract followed by facial paralysis, Otol. 82
—— ——, fractures of osseous walls of, Otol. 82
—— ——, gunshot injuries to, uncommon, Otol. 82
—— ——, malingering in form of self-injury to walls of, Otol. 82
—— —— (right), invasion of, by erysipelas of head: abscess formed, with extension backwards to sterno-mastoid muscle, and invasion of whole sheath of that muscle (A. H. Cheatle), Otol. 111
—— ——, stab-wounds of, Otol. 82
Mediastinum, gumma of, Electr. 21, 29, 30
——, gummatous, Clin. 52
——, tumours of, pressure from causing œsophageal obstruction, Electr. 21, 22
Medical practitioners of early eighteenth century, superstitious and credulous, Child. 121
Medicine, auto-inoculation in, rôle of (C. Riviere), Therap. 39-50
Medico-Chirurgical Society, Council of (1811), Med. 148
Medico-Chirurgical Transactions, ii (1811), contents of, as illustrating current progress of medicine and surgery (J. Mitchell Bruce), Med. 147-156
Medico-legal aspect of ear lesions, whether traumatic or no, Otol. 104, 105
Medicinal springs, classes of, Baln. 106
—— ——, connexion with volcanic action, Baln. 106
—— ——, discovery of new metals in, Baln. 118
—— ——, diuretic effect of water increased by carbonic acid gas in, Baln. 108
—— ——, gases contained in water of, Baln. 108
—— ——, hydrotherapeutic theory of, Baln. 105
—— ——, hypodermic administration of water from, Baln. 108
—— —— in New Zealand, Baln. 118
—— —— of India, Baln. 117
—— ——, origin of, Baln. 105, 106
—— ——, osmotic pressure or tonicity of water of, Baln. 108
—— ——, powers of, conform with laws of Nature, Baln. 82, 102, 103
—— ——, properties and uses of, Baln. 101
—— ——, radio-activity of water of, Baln. 108
—— ——, water of, in treatment of chronic disease, Baln. 118
—— ——, ——, see also *Spa treatment*
Medicine (clinical), relationship of electrocardiographic method to (T. Lewis), Med. 81, 95-99
——, progress of (1811), illustrated by contents of *Medico-Chirurgical Transactions*, ii, (J. Mitchell Bruce), Med. 147-156
Medicines, local action, Electr. 45
Medin, epidemic of poliomyelitis at Stockholm, reported by, Epid. 200
Meier, on complement fixation, referred to, Path. 251
Meinicke, experimental work on transmissibility of poliomyelitis, Epid. 199
Meisling's colorimeter, Baln. 47, 48

Melæna, after abdominal operations, of grave prognosis, Surg. 62
—— neonatorum, Surg. 49
Melanodermia, associated with pernicious anæmia, case of (J. H. Sequeira), Derm. 26
—— —— —— —— ——, blood-examination in, Derm. 27
Melton Mowbray, epidemic of poliomyelitis around (1910), Epid. 213, 214
Melville, Stanley.—Demonstration of a new tube-stand, Electr. 31
——, unusual case of aneurysm, Electr. 76
Memorial to Sir Felix Semon, K.C.V.O., Laryng. 149
Memory, hysterical loss of, see *Amnesia*
Mena House, health resort for neurasthenics, Baln. 42
Mendelism, illustrated by families of multiple cartilaginous exostoses, Med. 6
——, limitations of, Med. 21
Menière's symptoms—non-infective, in case of labyrinthine vertigo, treated by operation (G. J. Jenkins), Otol. 116
Meningitis (cerebrospinal) epidemics of, connexion with influenza, Epid. 224
——, complicating chronic suppuration in frontal sinus, Laryng. 141
——, fatal, following fracture of petrous bone, Otol. 89
—— (pneumococcal or streptococcal), frequent complication in human trypanosomiasis, Path. 15
—— (tuberculous) in infants following infection by tuberculous midwife, Child. 220
—— ——, proportion of deaths from, Child. 231
Meningococcus intracellularis, relation of, to pseudoglioma (G. Coats and J. Graham Forbes), Path. 242
Meningococcus, micro-organisms resembling, found in eye, in cases of metastatic ophthalmitis of right eye following measles, Path. 245, 246, 249
—— —— —— —— —— —— of associated metastatic ophthalmitis and arthritis of left wrist-joint, Path. 242, 243
Menopause, connexion of Graves's disease with, Clin. 122, 124
——, liability of women to chronic rheumatism at, Baln, 28, 34
——, results as to, after subtotal hysterectomy for fibromyoma uteri, Obst. 124-134, 136, 137
Menorrhagia in chronic endometritis, results of treatment by curetting, Obst. 46
——, treatment by calcium salts, Baln. 121
Menstrual discharge, chemical composition of, and cause of its normal non-coagulability (W. Blair Bell), 234
Menstruation, cessation after total hysterectomy, Obst. 116
——, continuation after subtotal hysterectomy, Obst. 117
—— —— —— —— —— —— for fibromyoma uteri, Obst. 135
——, maintenance of function of, under utriculoplasty, Obst. 274, 276
——, opening-up of vessels during, cause of (James Young), Obst. 291
——, vicarious, Obst. 139, 140
—— and pregnancy, uterine mucosa in (James Young), Obst. 291
Mental deficiency and syphilis, preliminary communication on (E. B. Smith and A. W. G. Woodforde), Child. 166
—— ——, Wassermann reaction in, results, Child. 171
—— depression, accompanying low blood-pressure, Med. 79
—— ——, indication for radical operation in chronic frontal sinus suppuration, Laryng. 132, 144
—— development, high, word-blindness co-existent with, Child. 52
—— phenomena, ordinary, Neur. 2
—— ——, subconscious, Neur. 2
—— process, inhibitory, producing insensibility to pain, Anæsth. 15
—— —— —— —— —— to sound, Anæsth. 16
—— theory of hysteria, Neur. 17
—— work, effect on low blood-pressure, Med. 56, 79, 80
Menthol, eucalyptol and terebene in oil of paraffin, injection of, in chronic frontal sinus suppuration, Laryng. 130

Menthol, spraying with, in frontal sinus suppuration, Laryng. 146
Merck's pneumococcic serum, Med. 102
Mercurial inunction, great improvement of sclerodermia with obstinate callous ulcers on legs under (A. W. Williams), Derm. 13
Mercury in treatment of congenital syphilitic deafness, useless, Otol. 8, 22
—— —— of dysentery, recommended (1811), Med. 148
—— —— of syphilis, administrations by mouth succeeding where injections failed, Clin. 21
Mergari and Palli, blood-changes in congenital pulmonary stenosis, reported by, Clin. 92
Mesenteric glands, tuberculosis of, proportion of deaths from, Child. 231
—— and bronchial glands, susceptibility to tuberculous invasion compared, Child. 230
Mesocolon, pelvic, Surg. 92
Metabolism, free motility of particles of matter essential to, Baln. 107
——, waste in, Therap. 70
——, see also *Nitrogenous metabolism*
Metal, molten, introduction into ear, injuring membrana tympani, Otol. 84
Metallic solutions (colloidal) in thermal waters, Baln. 104, 105
Metals, crystalline character of, Odont. 67
——, finely powdered, or inert salts of metals, application to ulcerated surfaces, Electr. 57, 58
—— (new), discovery of, in medicinal springs, Baln. 118
Metaplasia, see *Tissues*, transformation of
Metastasia (pulmonary) early, with lateral sinus thrombosis (streptococcal), recovery (S. Scott), Otol. 30
Metastatic growth in neck, with endothelioma of palate, treated by radium (J. Gay French), Laryng. 94
—— ophthalmitis of right eye following measles, Path. 245, 246
Methæmoglobinæmia, chronic, secondary polycythæmia not noted in, Clin. 86, 87
Methylene blue, local application in necrosis of jaw, Child. 31
Metritis (chronic fibrotic, fibroid), treatment by "utriculoplasty" (V. Bonney), Obst. 270, 275, 276
—— (hæmorrhagic), treatment by "utriculoplasty" (V. Bonney), Obst. 270, 275, 276
—— (septic), in cows, effect on milk, Epid. 75, 102
Mexico, local prevalence of typhus fever in, Epid. 225, 226
Meyer, paratyphoid carriers, quoted, Epid. 56
Meyer, Professor H., cause of oxalic acid poisoning, quoted, Baln. 124, 125
Mibelli, on angiokeratoma, quoted, Derm. 30
—— parakeratosis, quoted, Derm. 32
Mice, experimental infection with Gaertner bacillus, result fatal, Epid. 68
—— (hairless), specimens of, Path. 292
——, inoculation of centrifugalized extract of mouse cancer in, produces no prophylactic immunization to growth of graft, Path. 85
—— —— with air-dried mouse cancer, Path. 73-77
——, subcutaneous injection of air-dried carcinoma in, produces no prophylactic immunization to growth of subsequent graft, Path. 80
—— —— insertion of air-dried carcinoma in, produces no curative immunization to growth of graft, Path. 83
——, transmission of mouse cancer to, attempted by means of cancer spray, Path. 77
——, virulent effects produced by inoculation of *Streptococcus anginosus* into, Epid. 79, 80
Micrococcus catarrhalis and pneumococcus, mixed vaccines of, in treatment of episcleritis due to oral sepsis, Odont. 34
—— *melitensis*, cows subject to infection with, Epid. 75
Micro-organisms (pathogenic) in cervix, Obst. 193
—— —— in vagina, Obst. 193
—— —— (or saprophytic) on uterine swabbings, determination of nature, Obst. 171, 180, 191, 193, 194, 199, 206, 207
——, see also *Bacteria*

Micturition, nature of, cyclic, Med. 111
Middle age of life, pulmonary tuberculosis in, Epid. 35
Middle Ages, infant-feeding during, Child. 110, 111
Midwife (tuberculous) infecting infants, Child. 220
Miers, H. A., F.R.S., explanation of crystalline elongation, Path. 110, 138, 140
Mignot, time needed for formation of biliary calculus, stated by, Path. 95
Migraine, seaside climate seldom suitable for, Baln. 17
——, treatment by diathermic current indicated in, Electr. 7
Miklucho-Maclays, hypotrichosis in family of Australian aborigines, Path. 303
Miles, W. Ernest, presumed cause of syphilitic stricture of rectum, quoted, Obst. 73
——, radical abdomino-perineal operation for rectal cancer, Surg. 100, 101
——, statistics of cure after operation for rectal cancer, reported by, Surg. 110
Milian, treatment of pityriasis rubra pilaris by tuberculin, quoted, Derm. 24
Milk and powdered casein, absorption in rectal feeding, Therap. 64
—— as cause of human disease through infection from ulcerated teats of cows, discussed, Epid. 82, 83
——, disadvantages and dangers of, Child. 139
——, decomposition in cows affected with gastro-enteritis, Epid. 74
—— —— under Gaertner bacillus infection in cows, Epid. 74
——, diseases of cow (excluding tuberculosis) affecting milk in relationship to human disease (W. G. Savage), Epid. 73-104
—— drawn from healthy quarters of cows' udders partially affected with mastitis not safe for use, Epid. 79
——, effect of septic conditions of animals on, Epid. 75, 102
——, eggs and pancreatic extract, absorption in rectal feeding, Therap. 64
—— (fermented), decalcification of dentine by, Odont. 4, 5, 6, 9
—— from cows with mastitis, ingestion of, causing gastro-enteritis, Epid. 76
—— —— —— —— —— causing septic sore throat, Epid. 76
—— from tuberculous cows, percentage of children infected by, small, Child. 222
——, greatest age-incidence of dental caries coincides with period of greatest consumption of, Odont. 3
——, infected by mastitis in cows, Epid. 76, 99
——, possibility of spread of Malta fever by, Epid. 75
——, recognition as best artificial food for weaned children, Child. 126
——, transmission of foot-and-mouth disease to man through, Path. 75
—— —— of typhoid fever by, through agency of carriers at Aberdeen presumed, Epid. 110-115
—— —— —— —— —— —— —— at Folkestone (1893-1909), Epid. 16, 18
—— —— —— —— —— —— —— —— ——, probability discussed, Epid. 115-118, 137, 138, 145, 146
—— (tuberculous), infection by, Child 220, 221
Milk diet accelerates healing of acute ulcer of stomach, Path. 57, 58
Milk-germs, production of dental caries by (F. J. Bennett), Odont. 3
"Milk of sulphur," Therap. 22
Milkers infected by outbreak of vaccinia in herd of cows, Epid. 75, 99, 103
Milk supply of London in 1850, foul condition of, Child. 134
Miller and Edsall, absorption of proteins in rectal feeding, quoted, Therap. 64
MILLER, A. H.—Case of (?) chronic encephalitis, Child. 197
Miller, W. D., experiments as to fermentation of foodstuffs with regard to production of dental caries, alluded to, Odont. 91
MILLIGAN, W.—Case of chronic otitis media suppurativa; necrosis of external semicircular canal; fistula full of bone granulation tissue; operation; recovery, Otol. 65
——, discussion on case of congenital malformation of auricle and meatus, Otol. 25
—— —— —— of lateral sinus thrombosis, Otol. 33
—— —— —— of retro-cerebellar abscess, Otol. 37
—— —— on deafness due to occlusion of Eustachian tubes by scar-tissue, Otol. 110

MILLIGAN, W.—Discussion on eight cases of mastoid operation (Case I), Otol. 126 (Case II), Otol. 126 (Case VI), Otol. 132
——, discussion on three specimens of chronic middle-ear suppuration, Otol. 113, 114
——, injuries of external and middle ear, Otol. 79
——, suppuration of the labyrinth; facial paralysis; complete "bridge" operation; recovery, Otol. 63
——, two cases of cerebellar abscess, Otol. 61
MILNE, ROBERT.—Measles, its treatment and prevention, Child. 142-154, 180-184
Milner, S. W., case of oral sepsis causing thrombosis of cavernous sinus, reported by, Odont. 117
Mind, hysterical, capacities limited, Neur. 3
——, normal, comprehensiveness of perception, Neur. 3
——, subconscious states of, Neur. 3, 17
Minelli, investigations as to typhoid fever carriers, quoted, Epid. 12, 13, 119, 120
Mineral springs, chemical constitution, Baln. 104
—— —— —— —— does not always explain action, Baln. 104
Mineral water baths (German), re-hydration of skin effected by, Baln. 16
Mineral waters beneficial to gout containing carbonate of lime, Baln. 123
—— —— containing sulphur, action of, Therap. 19
—— —— at Bath, Baln. 15
—— —— at Buxton, Baln. 15
—— —— at Harrogate, Baln. 15
—— —— at Llandrindod Wells, Baln. 15
—— —— at Strathpeffer, Baln. 15
Miners (Welsh), difficulty of anæsthetizing, Anæsth. 5, 11
——, see also *Coal-miners*, *Gold-miners*
Mitral valve, disease of, rarity of pulmonary tuberculosis in subjects of, Child. 226
Mohr, excess of red blood-corpuscles in cases of splenomegalic polycythæmia, quoted, Clin. 86
Molar (permanent), absorption of, due to unerupted premolar (T. Stordy), Odont. 37
Molars (caries), recurrence in cervical cavities of posterior edges, Odont. 2
—— (deciduous), all in place, with premolar erupting internally or externally to arch, Odont. 112
—— (permanent), malocclusion of, cases (F. St. J. Steadman), Odont. 110, 111
—— —— —— ——, ætiology of, developmental defect, Odont. 112
—— —— —— —— —— discussed, Odont. 112, 113
Mole (hairless), specimens of, Path. 296, 297
—— (hydatidiform) association of corpus luteum cysts with, Obst. 220
—— (vesicular), associated with ovarian cyst which involuted, clinical history of case of (R. Drummond Maxwell), Obst. 219
—— upon face, liability to develop epithelioma or rodent ulcer, Electr. 51
Mollereau and Nocard, bacteriology of mastitis in cows first investigated by, Epid. 77
Mollities ossium of forty-five years' duration (H. D. Rolleston and C. H. S. Taylor), Clin. 97
—— —— —— —— ——, X-ray examination, Clin. 99, 104
Molluscum fibrosum, tumours of, nature of, in von Recklinghausen's disease, Clin. 75
MOLONY, J. B. DE W., and ROLLESTON, H. D.—Idiopathic hypertrophic dilatation of the colon, Child. 18
——, and SPRIGGS, E. I.—Angiomata of the left loin and leg, Child. 85
Monilithrix in male child, aged 16 months (S. E. Dore), Derm. 71
Monkeys, callosities in, how differing from those in camel, Path. 309
——, hairless gluteal districts in, Path. 309, 310, 311
——, ischial callosities of, Path. 287
——, spread of typhus fever among, experimentally produced by means of lice, Epid. 225
——, transmissibility of poliomyelitis to, Epid. 199
——, see also *Guinea baboon*, *Macacus*, *Mandrill*
Monnet, "Nouvelle Hydrologie" (1772), Baln. 79

Monochloracetic acid agar medium, colonies of *Bacillus coli* grown on, diversity in size, Path. 98, 99
—— —— —— —— —— —— ——, producing new variant, Path. 100, 101
—— —— —— —— colonies of *Bacillus enteritidis* (Gaertner) grown on, producing new variant, Path. 103
—— —— —— —— —— —— ——, and *Bacillus typhosus*, diversity in size, Path. 99, 100
—— —— —— colonies of bacteria grown on, action of acid discussed, Path. 108
—— —— —— —— —— ——, papillæ formation, Path. 99, 100, 107
—— —— ——, method of preparation, Path. 98
Mont Dore, baths of, pediluvia employed at, Baln. 73
Montgomery, E., practice as regards removal of appendix in pelvic operations, Obst. 351
Monthly nurses, drugging of infants by, Child. 141
Moon (The), genesis of, Path. 144
——, weaning by, superstition regarding, Child. 119
Moore, B., treatment of oxaluria, quoted, Baln. 124
MOORE, F. CRAVEN, and KILROE, LAURENCE.—The use of belladonna in dyspepsia, Med. 23-34
MOORE, IRWIN.—Cases illustrating the treatment of frontal sinus suppuration, Laryng. 124, 125, 145
——, discussion on the treatment of frontal sinus discharge, Laryng. 145
Moos, case of syphilitic disease of internal ear, quoted, Otol. 16
Morat and Dastre, injection of morphine and atropine before chloroform inhalation, quoted, Anæsth. 44, 55
Morbid Growths Committee, Laryngological Section, report on growth from nose (specimen shown by Mr. H. Tilley), Laryng. 18
Morgagni, on tuberculous contagion, quoted, Epid. 161
Morgan, D., association of *Bacillus enteritidis* with summer diarrhœa, quoted, Epid. 61
——, *Bacillus paratyphosus* B not found associated with summer diarrhœa, by, Epid. 55
——, and Savage, W. G., discovery of organism identical with *Bacillus paratyphosus* A in intestinal contents of healthy pigs, Epid. 54
Morgenstern, investigations on pulp-nerves, quoted, Odont. 52
MORISON, A.—Discussion on the electrocardiographic method, Med. 99
Moritz and Johansson, case of chloroma, quoted, Path. 63
Morphine and atropine hypodermic injection before chloroform inhalation, Anæsth. 44, 53, 55
—— —— —— ——, before inhalation anæsthesia, Anæsth. 51, 52
—— —— —— —— —— ——, risks of, Anæsth. 46, 51, 55
—— ——, preliminary use before anæsthesia by ether alternating with nitrous oxide gas and oxygen, Anæsth. 3
——, hypodermic injection before chloroform inhalation, danger of, Anæsth. 43
—— —— —— —— —— —— in parturition, Anæsth. 43, 53
—— —— ——, before ether inhalation, Anæsth. 44
—— —— ——, followed by enema of chloral in successful treatment of status epilepticus, Baln. 8
Morphœa affecting right frontal region and orbit, case of (James Galloway), Clin. 27
—— in a band form (J. L. Bunch), Child. 27
Morris, Sir H., Bt., case of dissemination of unobserved thyroid tumour, quoted, Clin. 125
MORRIS, H. J., and MCWILLIAM, ANDREW.—Comparative elasticity and internal structure of cast and rolled gold plate, Odont. 57
MORRIS, Sir MALCOLM, K.C.V.O.—Case for diagnosis, Derm. 25
——, discussion on cases for diagnosis, Derm. 35, 62, 71, 91
—— —— —— of cheilitis exfoliativa, Derm. 84
—— —— —— of congenital xanthoma (endothelioma), Derm. 77
—— —— —— of erythema ab igne, Derm. 89
—— —— —— of generalized eruption, probably mycosis fungoides, Derm. 21
—— —— —— of syphilitic auto-reinfection, Derm. 130
—— —— —— on herpes zoster, Derm. 88

MORRIS, Sir MALCOLM, K.C.V.O.—Discussion on case of ichthyosis in child, treated by thyroid extract, Derm. 72
——, discussion on case of lichen planus of unusual distribution, Derm. 81
—— —— —— of non-ulcerating tuberculide (?) Boeck's biliary sarcoid, following lupus vulgaris, Derm. 68
—— —— —— of sarcomatosis cutis of the "Perrin" type, Derm. 23
—— —— —— of (?) urticaria pigmentosa in an adult, Derm. 105
—— —— on macular atrophy associated with lupus erythematosus, Derm. 141
—— —— —— —— in syphilitic patient, Derm. 125
—— —— on report on case for diagnosis (sarcoma cutis), Derm. 82
—— —— on series of ringworm cultures, illustrating the eczematoid ringworm of the hands and feet, Derm. 112
—— —— on syphilitic affection of eighth nerve, Derm. 148
—— —— on unusual case of linear nævus, Derm. 39
——, and DORE, S. E.—Acute lupus erythematosus in a girl, aged 21, Derm. 106
—— ——, case for diagnosis, Derm. 107
—— —— —— of summer eruption, Derm. 4
—— —— extensive scarring and pustular eruption following Scinde ulcers, Derm. 107
—— ——, tylosis in a woman, aged 49, Derm. 108
Mortality, Bills, of grouping of children under 2 years of age, possible explanation, Child. 120
MORTON, E. REGINALD.—Discussion on diathermic treatment, Electr. 10
—— discussion on new induction coil, Electr. 82
——, method of applying sinusoidal current, quoted, Electr. 36
——, a sesamoid bone fractured by direct violence, Electr. 96, 98
Morton, R. (1694), on disposition in phthisis, quoted, Epid. 153, 154
—— ——, on tuberculous contagion, quoted, Epid. 161
Moskowicz, case of division of posterior spinal nerve-roots for gastric crises of tabes, Surg. 212
Most, instance of cutaneous scent of axillary glands causing sexual excitation, Path. 287
Mosquitoes, mounting of, by Bentley-Taylor method (A. Powell), Path. 41
—— —— —— —— demonstration of specimens (A. Powell), Path. 41
Motor disturbances, characterized by agitation, Neur. 8
MOTT, F. W., F.R.S.—Comparative neuropathology of trypanosome and spirochæte infections, with a résumé of our knowledge of human trypanosomiasis, Path. 1-40
——, discussion on the problem of disposition in pulmonary tuberculosis, Epid. 180
——, germ-plasm and body-cells, quoted, Path. 307
——, pathology of amaurotic family idiocy, quoted, Path. 171
—— and CARLYLL, H. B.—Seven cases of amaurotic idiocy (Tay-Sachs disease). Part I, by Dr. Carlyll; Part II, by Dr. Mott: Histological and chemical examination of the nervous structure, Path. 147-198
MOUILLOT, F. A. DE T.—Discussion on osteo-arthritic and chronic rheumatism, Baln. 35
Mount Vernon Hospital for Consumption, proportion of staff infected with tuberculosis, Epid. 36
Mountain air in treatment of neurasthenia, Baln. 41, 42
—— streams, freshness of water from, Baln. 72
Mountains, see *Altitudes*, high; *Alps*
Mouse, production of carrier state in, Path. 70
Mouse cancer (air-dried), experiments with (S. G. Shattock and L. S. Dudgeon), Path. 73-86
—— —— ——, lethal effect of drying upon cancer-cell, Path. 73
—— —— —— —— —— —— ——, proved by inoculation into mice, Path. 73-77
—— ——, subcutaneous insertion in mice produces no curative immunization to growth of graft, Path. 80
—— —— —— —— —— ——, produces no prophylactic immunization to growth of subsequent graft, Path. 80
—— —— (centrifugalized extract), inoculation in mice, produces no prophylactic immunization to growth of graft, Path. 85

Mouse cancer, experimental investigation, deductions from, as to rate of growth, Obst. 71
—— ——, transmission to mice attempted by means of cancer spray, Path. 77
Mouse favus (*Achorion Quinckeanum*), cultures of (H. G. Adamson), Derm. 47
Mouth, bacteria of, special, not general forms, factor in production of caries, Odont. 94
——, drugs used in, as local anæsthetics (F. R. Smyth), Therap. 51-58, 60-62
——, sterilization by weak solution of lactic acid, Odont. 9, 10
——, ulceration of, in relation to dental troubles, Odont. 78
——, viscocity of sugar in, shown experimentally, Odont. 96, 104
Mouth-breathing in relation to dental caries, Odont. 102
——, prevention of, Odont. 102
Moxon, W., on albuminuria in adolescence, quoted, Med. 131
Moynihan, B. G. A., close connexion between gall-stones and malignant disease of gall-bladder and bile-ducts, observed by, Path. 93
Much Hadham, epidemic poliomyelitis at, Epid. 209
Muci-carmine staining process for determining spread of cancer of rectum into surrounding tissues, Surg. 102, 121
Mucocele due to intranasal disease, Laryng. 103
Muco-colitis, associated with morbid condition of female reproductive organs, Obst. 328
Mucoid material, see *Colloids*
Mucosa (uterine) in menstruation and pregnancy; action of chorionic cells and function of decidua (James Young), Obst. 291
—— ——, structure of, and cause of opening-up of vessels during menstruation (James Young), Obst. 291
Mucous membrane (normal), penetration by bacteria without lesion being produced, Child. 217
—— —— of mouth, diseases of, the direct result of dental troubles, Odont. 78-80
—— ——, relation between the teeth and certain diseases of the skin and mucous membrane (W. K. Sibley), Odont. 71-84
Mucus, coating of, on teeth, protective action, Odont. 94, 95, 96
Muecke, F. F.—Ventricular voice in woman, aged 28, Laryng. 106
Müller, Reiner, on mutation of micro-organisms, Epid. 124, 125, 146
Müllerian ducts, derivation of adenomatosis vaginæ from, discussed, Obst. 23
Multisinusitis, treatment by Killian's operation, case (I. Moore), Laryng. 124, 145
—— —— by Ogston-Luc operation, case illustrating (I. Moore), Laryng. 125, 126, 145
Mummery, J. Howard.—Discussion on three cases of torus palatinus, Odont. 40
——, short preliminary note on distribution of nerve-fibres of dental pulp, Odont. 51
Mummery, J. P. Lockhart.—Case of fatty tumour in the abdominal wall, Child. 188
——, discussion on case of congenital deformity of palate, Child. 5
—— —— —— of congenital dilatation of colon, Child. 76
—— —— —— ——, obliteration of bile-ducts, Child. 50
—— —— —— of injury to brachial plexus, Child. 9
—— —— —— of old healed tuberculous disease of knee-joint with increase of length of limb, Child. 40
—— —— —— of syphilitic osteitis of femur, Child. 104
—— —— on chronic pyæmia of five years' duration following suppurative epiphysitis of upper end of humerus, Child. 162
—— —— on idiopathic hypertrophic dilatation of colon, Child. 22
—— —— on malformation of femur, Child. 165
—— —— on operative treatment of cancer of rectum, Surg. 125
—— —— on pyloric hypertrophy and spasm, Child. 186
—— —— on rectum in gynæcology, Obst. 336
——, hyperplastic tuberculosis of sigmoid, quoted, Path. 240
——, severe tuberculous peritonitis in a girl, cured by laparotomy, Child. 186
——, tumour of the rectus abdominis in a child, Child. 7
—— and Collier, James.—Case of tabes dorsalis, with early rectal crises, Clin. 117

MUMMERY, STANLEY.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 48
——, discussion on case of pneumococcal stomatitis, Odont. 12
—— —— on destruction of teeth by milk germs, Odont. 9
—— —— on an erosion cavity on the lingual aspect of a lower incisor, Odont. 70
—— —— on infection of cavernous sinus due to oral sepsis, Odont. 118
—— —— on a possible cause of facial asymmetry, Odont. 109
Munich, hydro-therapeutic equipment of University clinic at, Baln. 87
Munk and Rosenstein, absorption of fats in rectal feeding, quoted, Therap. 66
Munro, Dr., suggestion operations, quoted, Anaesth. 22
Murchison, C., contact infection in typhoid fever, quoted, Epid. 130, 139
Murphy, J. B., on appendicitis, quoted, Obst. 347
——, practice as regards removal of appendix in pelvic operations, Obst. 351
MURPHY, Sir SHIRLEY.—Discussion on notification of disease and epidemiology of poliomyelitis, Epid. 222, 223
——, discussion on tuberculosis and its prevention, Epid. 47
Murray, practice as regards removal of appendix in pelvic operations, Obst. 353
Murray, W., "Rough Notes on Remedies," quoted, Baln. 7
Murrell, W., and Ringer, S., action of aconite, quoted, Therap. 157
Muscle strips, contraction of, electric changes accompanying, curve showing, Med. 83
—— —— —— —— —— —— factors producing, Med. 83, 84
—— —— —— —— —— —— indicated by string galvanometer, Med. 82, 83
Muscles, contracture of (progressive), with spondylitis (Wilfred Harris), Neur. 42
—— —— —— —— —— —— (? myositis fibrosa) (Wilfred Harris), Clin. 145
—— facial ? affection of, case of myopathy (F. Langmead), Child. 108
——, ossification of, Surg. 133
—— (palatal), absence of development in case of congenital insufficiency of soft palate, Child. 23
—— (pectoral), absence of (congenital), case of (D. C. L. Fitzwilliams), Child. 79
——, separation of periosteum by, resulting in formation of swelling, Surg. 143
Muscular atrophy (peroneal type) of (Charcot-Marie, Tooth), two cases of, in father and son (L. G. Guthrie), Clin. 126
—— ——, progressive spinal, of infants and young children (F. E. Batten), Neur. 53-84
—— —— —— —— —— —— ——, case of, Neur. 63
—— —— —— —— —— —— ——, cases described, Neur. 76-83
—— —— —— —— —— —— ——, chronic myelitis type, Neur. 65
—— —— —— —— —— —— —— —— —— ——, case of, Neur. 65
—— —— —— —— —— —— —— —— —— ——, clinical and pathological features, Neur. 65
—— —— —— —— —— —— ——, clinical and pathological features, Neur. 63
—— —— —— —— —— —— ——, diagnosis, Neur. 66
—— —— —— —— —— —— —— ——, differential, Neur. 70-74
—— —— —— —— —— —— —— —— from myatonia congenita, Neur. 67
—— —— —— —— —— —— —— —— from spinal caries, Neur. 67
—— —— —— —— —— —— —— —— of one variety from another, Neur. 67
—— —— —— —— —— —— ——, literature as to, Neur. 68
—— —— —— —— —— —— ——, toxic neuritis type, Neur. 63
—— —— —— —— —— —— ——, Werdnig-Hoffmann type, Neur. 54
—— —— —— —— —— —— —— —— ——, cases, Clin. 115 ; Neur. 54, 58
—— —— —— —— —— —— —— —— —— ——, included in, Neur. 68, 70
—— —— —— —— —— —— —— —— —— —— not regarded as belonging to, Neur. 71
—— —— —— —— —— —— —— —— ——, diagnosis from poliomyelitis, Neur. 67
—— —— —— —— —— —— —— —— ——, pathological features, Neur. 55-59, 60-62
Mustard (black), oil from, sulphurous and antiseptic, Therap. 23
Mus nudoplicatus (hairless mouse), Path. 292, 293
Mutation of bacteria, Epid. 124-127, 136, 137, 141, 142, 146

Mutism, case of (G. S. Haynes), Child. 192
Myasthenia gravis, with laryngeal symptoms (S. Hastings), Laryng. 39
Myatonia congenita, diagnosis of progressive spinal muscular atrophy from, Neur. 67
—— ——, or "Amyotonia congenita" (so-called): simple atrophic type of myopathy, case of (F. E. Batten), Child. 100
Mycosis fungoides (?) case of (J. H. Sequeira), Derm. 77
—— ——, probable (generalized eruption), case of (T. Colcott Fox), Derm. 20
Myelitis (chronic), progressive spinal muscular atrophy of infants and young children of type of, Neur. 65
Myeloma of humerus (upper end), Child. 203
Myocarditis (sclerotic progressive), association of delirium cordis with, Med. 96
Myoma of uterus, operations for, preservation of ovaries in, Obst. 117, 118
Myomata of pregnant uterus, successful enucleation possible, Obst. 290
Myomectomy for intramural fibroid in third month of pregnancy, case referred to, Obst. 12
Myopathy, case of (? affection of facial muscles) (F. Langmead), Child. 108
——, atrophic (simple) type of, so-called "myatonia congenita" or "amyotonia congenita" (F. E. Batten), Child. 100
Myositis fibrosa, accompanying scleroma neonatorum (F. S. Langmead), Child. 5
—— —— (spondylitis with progressive muscular contracture) ? case of (Wilfred Harris), Clin. 145
—— ossificans progressiva, multiple cartilaginous exostoses associated with, Med. 3
—— ——, traumatic (R. J. Godlee), Surg. 144-156
—— —— —— (G. H. Makins, C.B.), Surg. 133-142
—— —— ——, ætiology, injury to periosteum as essential factor in, Surg. 154, 156
—— —— —— ——, theories as to, Surg. 138, 140
—— —— ——, cases reported in German Army, Surg. 133
—— —— ——, clinical separation from other ossifications, Surg. 141
—— —— ——, distinct from "rider's bone," Surg. 141
—— —— ——, following fracture of coronoid, Surg. 10
—— —— ——, hæmatoma developing in early stage of, Surg. 133, 140
—— —— —— in connexion with soleus muscle, Surg. 157, 158
—— —— —— in neighbourhood of elbow-joint, Surg. 153, 154
—— —— —— —— of femur, Surg. 144-153
—— —— ——, pathology of, Surg. 136-138
—— —— ——, propriety of term discussed, Surg. 154, 156, 158
—— —— ——, resulting from sprain, Surg. 157, 158
—— —— ——, specimens illustrating, Surg. 147-153
—— —— —— —— ——, comparative rarity, Surg. 152
—— —— ——, structure of bone in, Surg. 138
—— —— ——, treatment, Surg. 142
—— —— —— ——, operative, question of, Surg. 144, 154
Myxœdema (juvenile); infantilism with thyroid inadequacy, case of (H. M. Fletcher), Child. 96
Myxo-sarcoma of uterus, supposed case of, Obst. 112

Nævi of peculiar appearance in girl, aged 12 (E. G. Graham Little), Derm. 74
Nævoid condition (congenital) of left thigh (W. H. Bowen), Child. 193
Nævo-lipomata in an infant (E. G. Graham Little), Derm. 23
Nævus, large cavernous, of foot (Sidney Boyd), Clin. 83
—— (linear), unusual case (J. H. Sequeira), Derm. 39
—— —— ——, pigmentation in, Derm. 39
—— of tongue, removal under application of diathermic current, Electr. 8
—— (pigmentary), unilateral, case of (H. G. Adamson), Derm. 59
—— (pigmented, vascular, sclerosing), large, in girl (H. G. Adamson), Derm. 97

Nævus pigmentosus verrucosus et pilosus (E. G. Graham Little), Derm. 64

—— unius lateris, two cases of (E. G. Graham Little), Derm. 64

—— verrucosus, see *Ichthyosis hystrix*, linearis, syn. nævus verrucosus

Nagel, views on appendiculo-ovarian ligament, Obst. 343

NAGELSCHMIDT, FRANZ.—The thermal effects produced by high-frequency currents and the therapeutical uses of diathermic treatment, Electr. 1-8

Nails, tropho-neurotic (?) separation of, case of (F. Parkes Weber), Derm. 137

Naples, University of, lectures on hydrology and seaside treatment at, Baln. 116

Narceine, hypodermic injection before chloroform inhalation, Anæsth. 44

Nasal duct, blocking of, following nasal antrostomy for antrum suppuration (Dan McKenzie), Laryng. 102

—— method, administration of nitrous oxide gas by, new inhaler for (J. F. Trewby), Anæsth. 12

—— reflex during anaesthesia (J. Blumfeld), Anæsth. 27

—— spine, instrument for more efficient removal in submucous resection of septum (H. Tilley), Laryng. 25

—— ulceration, syphilitic, causing obstruction of Eustachian tube, Otol. 12, 13

Nash, W. G., outbreak of milk-borne sore throat at Bedford, reported by, Epid. 92

Nasmyth's membrane, Odont. 89

Nasopharynx, adenoid tissues of, as portals of tuberculous infection, Child. 223

——, development of, factors affecting, in relation to cleft palate, Surg. 170

——, foreign bodies in, Otol. 105

—— —— ——, injuring mouth of Eustachian tube, Otol. 86

——, infiltrating malignant tumour on lateral wall of, course and symptoms, Otol. 68, 69

——, obliteration (cicatricial) of (hereditary syphilis) (Dan McKenzie), Laryng. 104

——, polypus of, removed from boy (H. J. Davis), Laryng. 32

—— —— (fibrous), case of (W. H. Kelson), Laryng. 60

—— —— ——, method of operating for, Laryng. 61, 62

——, scleroma, causative organism, Laryng. 7

——, see also *Adenoids* (nasopharyngeal)

Nastin, injections of, in leprosy, Laryng. 27

—— ——, in case of leprosy with involvement of external ears, palate, pharynx, &c., Otol. 120

NATHAN, S. H.—Discussion on hypnotism in relation to surgical anæsthesia, Anæsth. 20

National Association for the Prevention of Tuberculosis, Epid. 32

Natural functions, all cyclic, Med. 111

Nauheim bath, aërated (Harrogate), effect on low blood-pressure, Med. 72

—— baths, criticism of value of, Baln. 43

—— ——, nature of, Baln. 96

Naunyn, incidence of gall-stones, quoted, Path. 88

Nausea following operations under anæsthetics, Anæsth. 2

NEAVE, S.—von Jaksch's anæmia, Child. 155

Neck (glands of), tuberculous disease of, production through pediculi of scalp, Child. 223, 224, 232

——, herpes of, followed by internal ear deafness and facial paralysis, Otol. 17

——, right side of, tumour of, involving pharynx, in woman, aged 30 (W. H. Kelson), Laryng. 99

Necrosis and caries of temporal, parietal, and occipital bones following mastoiditis due to scarlet fever in girl, aged 5; operation; recovery (H. J. Davis), Otol. 70

——, extensive, of lower jaw, case of (H. S. Clogg), Child. 30

—— of lower jaw, specimen from case of (H. S. Clogg), Child. 53

—— (painful) of extremities, case of (James Galloway), Clin. 26

—— (spontaneous) of multiple cartilaginous exostoses, Med. 2

—— of tibia, specimens, Child. 202

Needle (Berry's) in operation for cleft palate, Surg. 180

Needle douche (Harrogate), effect on low blood-pressure, Med. 72
—— spray of alternating temperature, application in neurasthenia, Baln. 41
Negro races, susceptible to suggestion, Anæsth. 23
Neisser and Wechsberg, functions of free amboceptors, quoted, Path. 276
Nélaton's sphincter, Surg. 92
Nelson, on errors of diet of young children in eighteenth century, quoted, Child. 122, 123
Nematodes, intestinal invasion of, in birds and animals, associated with *Bacillus coli*, Child. 218
——, invasion of grouse by, Child. 218
Neoplasm, malignant, of larynx, case of (P. Watson-Williams), Laryng. 16
Neoplasms, "rest" theory of, Med. 3
Nephritis (acute) complicating corrosive sublimate poisoning, Clin. 168
—— ——, followed by suppression of urine, treatment by nephrostomy, Clin. 162
—— ——, following scarlet fever, Epid. 194
—— (chronic), treatment by diathermic current, Electr. 8
——, marked intermitting albuminuria not a characteristic of, Med. 112, 113
—— (scarlatinal) with complete suppression, treatment by nephrostomy, Clin. 168
Nephrostomy in acute nephritis followed by suppression of urine, Clin. 168
—— in anuria, Clin. 162
Nerve (eighth), syphilitic affection of, case (J. E. R. McDonagh), Derm. 147
—— (facial), injuries to, during aural operation, Otol. 85
—— —— —— —— —— —— in children, Otol. 85
—— (optic), atrophy on one side associated with hemiplegia on other, Child. 196
—— —— ——, secondary, due to (?) cerebellar tumour (Oliver K. Williamson), Child. 109
Nerve deafness following injury to head, Otol. 92
—— —— (syphilitic), Otol. 16
—— —— —— of thirty years' standing, (A. H. Cheatle), Otol. 26
—— —— with middle-ear disease in congenital syphilis, Otol. 21
Nerve distribution of skin of head and face, Odont. 72, 73
—— ——, effect of sinusitis on, Laryng. 163, 164-166
Nerve-fibres of dental pulp, distribution of (J. Howard Mummery), Odont. 51
—— —— —— —— of, best method of staining to investigate, Odont. 53
Nerve-roots, spinal, posterior, division of, Surg. 200-225
—— —— —— ——, approach to lumbo-sacral roots, Surg. 222
—— —— —— ——, best method of laminectomy for, Surg. 220
—— —— —— ——, degenerations and cell-changes resulting from, Surg. 208
—— —— —— ——, experimental, Surg. 210, 219, 220
—— —— —— —— for gastric crises of tabes, Surg. 210-213
—— —— —— —— —— ——, Guleke's method, Surg. 229
—— —— —— —— —— ——, results, Surg. 227, 228, 253
—— —— —— —— —— —— in ? case of tabes (C. M. Hinds Howell), Neur. 48
—— —— —— —— for spasm, Surg. 213-225
—— —— —— —— ——, cases, 213, 216, 217
—— —— —— —— for spastic paralysis, Surg. 230
—— —— —— —— —— ——, cases, Surg. 231-243, 247, 251
—— —— —— —— —— ——, contra-indications for. Surg. 231, 252
—— —— —— —— —— ——, value discussed, Surg. 230, 231
—— —— —— —— for visceral crises, Surg. 206-213
—— —— —— —— —— ——, cases, 206
—— —— —— ——, historical account of operations for, Surg. 226, 227
—— —— —— —— in relief of athetosis, Surg. 243
—— —— —— —— —— of pain, Surg. 200-206
—— —— —— —— —— ——, cases, Surg. 200, 202, 248
—— —— —— ——, relation of orthopædic operations to, Surg. 222-224
—— —— —— ——, theory upon which operation is based, Surg. 219, 220

Nerves (cranial), ninth, tenth and eleventh, unilateral paralysis of (P. W. Saunders, for James Taylor), Neur. 51
——, fifth and sixth, trunk lesion of (L. McGavin), Clin. 52
——, transplantation of, Surg. 253
—— (trophic), disorder of, causing excess of bony overgrowth in Charcot's disease, Baln. 32
Nervous exhaustion, treatment by subthermal baths, Baln. 97
" Nervousness " not defined, Neur. 8
Nervous system (central), histological changes in, in human trypanosomiasis, Path. 22-28, 35
—— ——, changes in the neural elements, in human trypanosomiasis, Path. 31, 32
—— ——, condition of, in cases of amaurotic idiocy (Tay-Sachs disease), Path. 153-166
—— ——, diseases of, climatic factor in bath treatment of, Baln. 68, 69
—— —— ——, treatment by subthermal baths, Baln. 68
—— ——, disturbances of, atonic dyspepsia with gastric dilatation due to, Electr. 40
—— ——, effects of trypanosome and spirochaete on, compared, Path. 2, 3
—— ——, family disease of, case for diagnosis (C. A. Basker), Child. 198
—— ——, influenza attacking, Epid. 192, 223
—— ——, microscopic examination of nerve-cells and fibres in human trypanosomiasis Path. 32, 33
—— —— (peripheral), action of subthermal baths on, Baln. 96
—— —— (sympathetic), disturbance of, connexion of neurasthenia with, Baln. 44
—— ——, ultimate action of baths on, through nervous end-organs of skin, Baln. 93
Neugebauer, dangers of vaginal pessaries, quoted, Obst. 324
Neuralgia, supra-orbital, following operation for chronic frontal sinus suppuration, Laryng. 139, 143
——, treatment by diathermic current indicated in, Electr. 7
Neurasthenia, auto-intoxication and, Baln. 44
——, becoming chronic after operation, Baln. 38
——, connexion of influenza with, Baln. 44
—— —— with disturbance of sympathetic nervous system, Baln. 44
—— due to anxiety and exhaustion, Baln. 44
—— due to chronic abdominal disease, Baln. 38
——, exact connotation of term needed, Baln. 43, 44
—— from over-strain, marine climate indicated for, Baln. 17
——, gastro-intestinal catarrh in, Baln. 40
——, indication for radical operation in chronic frontal sinus suppuration, Laryng. 132
——, insidious in development, Baln. 37
——, low blood-pressure in subjects of, Med. 73
——, protean nature of, Baln. 43
——, time, important factor in treatment of, Baln. 37
——, tonic effect of chalybeate waters on, Baln. 40
——, treatment by application of hot and cold douches to spine, Baln. 41
—— —— by flushing of colon (Plombières system), Baln. 41
—— —— by high-frequency currents, Baln. 41
—— —— by ionization, Baln. 44
—— —— by massage, Baln. 41
—— ——, balneological (W. R. Gore), Baln. 37
—— —— ——, by wet pack, Baln. 43
—— —— ——, external, Baln. 41
—— ——, climatic (W. R. Gore), Baln. 37-44
—— —— ——, by sea and mountain air, Baln. 41, 42
—— —— ——, by sea-voyages, Baln. 41, 42
—— —— ——, localities for, Baln. 41
—— —— —— ——, in British Isles, Baln. 42

Neurasthenia, treatment, climatic, localities for, in Continental Europe and North Africa, Baln. 42
—— ——, hydrological, Baln. 39
—— —— ——, at Llandrindod Wells, Baln. 39
—— —— ——, by drinking and inhalation of strong radium emanations, Baln. 44
——, see also *Psychasthenia*
Neurasthenics difficult to hypnotize, Surg. 18
Neuritis of ear, Otol. 15
—— (optic, unilateral) and complete ophthalmoplegia externa, resulting from acute sphenoidal sinusitis (H. J. Davis), Laryng. 28
—— (retrobulbar), connexion of, with ethmoidal disease, Laryng. 167, 168
—— (rheumatic), importance in electro-therapy of treating spinal root of nerve, Electr. 104
—— (toxic), diagnosis from poliomyelitis, Neur. 21, 22, 23
—— —— of doubtful nature, case of (F. E. Batten), Neur. 20
—— ——, progressive spinal muscular atrophy of infants and young children of type of, Neur. 63
—— (traumatic) of inner cord (Klumpke-paralysis), prefixed type of plexus (W. Harris), Neur. 31
Neuroglia cell, large, from case of amaurotic idiocy (Tay-Sachs disease) (photomicrograph), Path. 183, 184
—— cells, examination of, in human trypanosomiasis, Path. 24-28
—— fibrils, overgrowth, in amaurotic idiocy (Tay-Sachs disease), Path. 194, 204
Neuromimesis and hysteria, Neur. 2
Neurones, cytoplasm of, fatty degeneration of, in cases of amaurotic idiocy (Tay-Sachs disease), Path. 176, 178, 193
——, nucleus of, auto-katalytic ferment action of, Path. 175, 192, 193
—— —— —— —— —— ——, process of parthogenesis in sea-urchins' eggs compared with, Path. 175, 192, 193
—— ——, failure of, to build up Nissl substance factor in pathogenesis of amaurotic idiocy (Tay-Sachs disease), Path. 174, 175, 192
—— ——, specific, autonomic, inherited energy resident in, Path. 175
Neuropathology, comparative, of trypanosome and spirochæte infections (F. W. Mott, F.R.S.), Path. 1
New growth of unknown nature in tympanum (W. H. Kelson), Otol. 77
New York, hydrotherapy in, Baln. 90
—— ——, see also *Columbia*, University of
New Zealand, thermal springs in, Baln. 118
NEWSHOLME, A.—Discussion on tuberculosis and its prevention, Epid. 48
——, on infection in phthisis, quoted, Epid. 171
——, outbreak of milk-borne sore throat at Brighton, reported by, Epid. 91, 92
Niepratschk, military case of typhoid carrier, Epid. 121, 122
de Niet, Professor, lecturer on hydrotherapy at University of Utrecht, Baln. 116
Nieter and Liefmann, proportion of typhoid carriers in asylums, Epid. 119, 120
Nikolsky's sign present in pemphigus foliaceus of fifteen years' duration, Derm. 15
Nipples, smearing with wormwood or aloes to overcome child's dislike to strange food, Child. 119
Nissl substance, disappearance of, in amaurotic idiocy (Tay-Sachs disease), Path. 176
—— ——, elaboration of, Path. 175
—— —— ——, failure in, on part of nucleus of neurones factor in pathogenesis of amaurotic idiocy (Tay-Sachs disease), Path. 174, 175, 192
—— ——, nature of, Path. 175
Nitrogen, excretion during taking of sulphur water, Baln. 50, 51
—— ——, in subjects of starvation, Therap. 70
—— in medicinal waters, Baln. 108
——, total, in urine estimation by Kjeldahl's method, Baln. 47
—— and oxygen as constituents of air, discovery of, Baln. 3

Nitrogenous equilibrium, Therap. 70
—— metabolism during rectal feeding, Therap. 67
Nitrous oxide gas, administration by nasal method, new inhaler for (J. F. Trewby), Anæsth. 12
—— —— —— and ether, anæsthesia by, Anæsth. 8
—— —— —— and oxygen, anæsthesia by, suitable for short operations, Anæsth. 3
—— —— —— ——, prolonged anæsthesia under ether alternating with (A. L. Flemming), Anæsth. 1
—— —— ——, see also *Ether*, anæsthesia by (prolonged)
De Nobele, discovery of *Bacillus suipestifer* (*Aertryck*), by, Epid. 53
Nocard and Mollereau, bacteriology of mastitis in cows, first investigated by, Epid. 77
Nodes, rheumatic, see *Bruce's nodes*, *Heberden's nodes*
Nodules, tuberculous, disseminated, of skin (E. G. Graham Little), Derm. 48
Noises, loud, damaging organ of hearing, Otol. 93
Non-purin diet, excretion of sulphocyanate during, Baln. 57
NORBURY, L. E. C.—Case of perforated tuberculous ulcer of ileum, Clin. 50
Nordau, Max, asymmetry as stigma of degeneracy, quoted, Odont. 106
Norfolk, alleged prevalence of vesical calculus in, Baln. 125
North American Indians, treatment of fever among (1683), Baln. 89
Nose, accessory sinuses, caries of walls of, indication for radical operation in chronic frontal sinus suppuration, Laryng. 132
—— —— ——, diseases of, in relation to affections of eye and orbit, discussion on, Laryng. 161-168
—— —— —— ——, effect on vision, Laryng. 163, 164-166
——, congenital anomalies of (A. R. Tweedie and A. Keith), Laryng. 47-54
——, disease or operation on, causing epiphora, or lachrymal trouble, Laryng. 168
——, growth from, report on specimen shown by Mr. H. Tilley, Laryng. 18
——, larynx and lungs, chronic tuberculosis of, case of (C. A. Parker), Laryng. 64
——, operations on, nasal reflex during, Anæsth. 28, 30, 32, 33
——, routes of extension of disease from nasal to ophthalmic regions, Laryng. 162
——, syphilitic disease of, treated with "606," case of (G. N. Biggs), Otol. 121
——, tertiary syphilitic ulceration of, accompanying congenital syphilitic deafness, Otol. 8
——, vestibule of, and adjoining skin, primary sore of (N. Patterson), Laryng. 84
Notification of disease, new order of Local Government Board as to, Epid. 185, 196
—— —— —— —— —— —— ——, importance of, and value of results to be expected from, Epid. 186, 196, 197, 222
—— ——, scientific uses (A. Ransome), Epid. 185-197
Notification of Diseases Act (1889), passing of, Epid. 188
—— —— ——, powers of, not fully developed, Epid. 188, 222
Notification of measles, Child. 173
—— of poliomyelitis, Epid. 218, 219
NOURSE, W. J. CHICHELE.—Case of lateral sinus thrombosis following acute otitis media in a boy; operation; recovery, Otol. 56
——, discussion on cases illustrating results of removing nodules from cords by means of galvano-cautery (by indirect method), Laryng. 41
—— —— on syphilis in relation to otology, Otol. 23
—— —— on treatment of frontal sinus discharge, Laryng. 144
Novocaine, action and use of, Therap. 56-58
Nuclein, production of, Path. 175
Nurses, chewing of meat by, before giving to children, old practice of, Child. 119, 120
—— in fever hospitals, contact infection with typhoid fever, Epid. 128, 129, 139
——, see also *Wet-nurses*
Nussbaum, injection of morphine before chloroform inhalation, quoted, Anæsth. 43
Nutrition, disturbances of, allayed by subthermal bath treatment, Baln. 96
Nystagmus, mixed, produced by cooling semicircular canal, Otol. 55

Obeah system in West Indies, Anæsth. 23, 24
Obesity, forms of, benefited by thyroid gland extract, Path. 299
O'Brien, R. A.—Habitat of *Bacillus suipestifer*, Epid. 59
——, and Petrie, G. F.—Experimental production of the carrier state by feeding, Path. 70
—— ——, epizootic in guinea-pigs, Epid. 124, 136
Obsessions, defence neuroses, Neur. 12
——, instances of, Neur. 8, 9
Obstetrical and Gynæcological Section, functions and aims of, Obst. 2
—— —— ——, quinquennial summary of Proceedings suggested, Obst. 1, 2
—— —— ——, work of session 1909-10 reviewed (H. Macnaughton-Jones), Obst. 1-17
—— —— —— ——, 1910-1911 reviewed (H. Macnaughton-Jones), Obst. 337-340
—— —— —— —— —— ——, clinical communications, Obst. 338
—— —— —— —— —— ——, operative communications, Obst. 339
—— —— —— —— —— ——, pathological communications and exhibits, Obst. 337, 338
Obstetrical instruments, sterilization of, Obst. 193
Obstetrics, anæsthesia produced by hypnotism in, Anæsth. 21
Obturator nerve and external iliac vein, large gland lying between, Obst. 75
Occupation, nature of, factor in development of Heberden's nodes, Baln. 32
Oculo-motor type of polio-encephalitis (S. Stephenson), Child. 87-93
Odontoblasts, origin and formation of, Odont. 54, 56
Odontomes, follicular, Odont. 45, 49, 50
Œdema, important in early diagnosis of human trypanosomiasis, Path. 13, 14, 18
——, localized, in relation to dental trouble, Odont. 77
—— of conjunctiva from acute frontal sinusitis (A. J. M. Wright), Laryng. 160
—— of glottis and urgent dyspnœa, due to hæmorrhage into thyroid (Arthur Evans), Clin. 25
——, treatment by diathermic current, Electr. 7
Oerum, Dr., colorimetric adaptation of Bouma's red indigo method, Baln. 47
——, percentage of oxygen capacity and blood per 100 grm. body-weight, obtained by, Clin. 89
Œsophago-gastroscope, improved, combining methods of direct and indirect vision (W. Hill), ·Laryng. 58
——, new type of, used in demonstration on living subject of the Hill-Herschell method of combined direct and indirect gastroscopy (W. Hill), Laryng. 69
Œsophago-gastroscopy on living subject, demonstration (W. Hill and G. Herschell), Laryng. 151, 152
Œsophagoscope, expanding, new pattern (W. Hill), Laryng. 58
Œsophagoscopy and bronchoscopy, demonstrations, Laryng. 151-153
——, hypodermic injection of scopolamorphine before inhalation anæsthesia for, Anæsth. 52
Œsophagus and air-passages, removal of foreign bodies from, specimens illustrating (H. Tilley), Clin. 152
—— and larynx, cancer (party-wall) of (L. H. Pegler), Laryng. 101
—— and posterior margin of larynx, extension of cancer of deep pharynx to (W. Hill), Laryng. 44
——, cancer of, associated with aneurysm, Electr. 23, 30
—— ——, diathermic current indicated in, Electr. 9, 11
—— ——, dysphagia treated by new form of intubation apparatus (W. Hill), Laryng. 9
—— —— (fungating) of, disappearance of fungation and ulceration after treatment by radium (W. Hill), Laryng. 8
—— ——, treatment by radium, cases reported, Electr. 64-66
—— —— —— —— ? temporary cure (W. Hill), Laryng. 113
——, carcinoma of (W. Hill), Laryng. 43
——, denture removed from (N. Patterson), Laryng. 54
——, deviation of, due to thoracic aortic aneurysm and dilatation of aortic arch, Electr. 23 26

Œsophagus, examination under X-rays (A. C. Jordan), Electr. 13-30
—— —— —— ——, by means of bismuth meals, Electr. 14-23, 27
—— —— —— ——, method, Electr. 13, 14
—— —— —— ——, when normal, Electr. 14
—— —— —— ——, when obstructed, Electr. 15-26
——, foreign bodies in, skiagraphy of, Electr. 91
——, malignant stricture of, case of (P. Watson-Williams), Laryng. 83
—— —— ——, œsophagoscopy and application of radium (P. Watson-Williams, C. Hope, N. S. Finzi), Laryng. 153
——, obstruction of, cicatricial, Electr. 15, 16
—— ——, due to foreign bodies, Electr. 16
—— ——, due to new growths, Electr. 17, 30
—— —— —— —— at lower end, Electr. 17
—— —— —— —— at middle portion, Electr. 19
—— —— —— —— at upper end, Electr. 18
—— ——, due to piles, Electr. 17
—— ——, due to pressure from tumours of mediastinum, lungs, and pleura, Electr. 21, 22
—— ——, spasmodic, Electr. 15, 16, 28, 30
——, peristalsis of, Electr. 14, 28, 29
—— ——, reversed, Electr. 27
——, stricture of, careful probing important, Laryng. 9
—— —— of lower end (J. Gay French), Laryng. 36
—— —— ——, spasmodic origin discussed, Laryng. 36-39
—— ——, simple and malignant, differential diagnosis, Electr. 27, 29
—— ——, spasmodic, co-existent with great dilatation, Electr. 30
——, upper end, larynx, pharynx, lower part, excision for malignant disease (squamous-celled carcinoma), subsequent report on case (A. Evans), Clin. 142
Oesterlen, sceptical ideas as to therapeutics, Baln. 84
Ogston-Luc operation in chronic frontal sinus suppuration, Laryng. 134, 144, 146, 147, 148
—— —— in treatment of multisinusitis, case illustrating (I. Moore), Laryng. 125, 126, 145
—— —— —— of septic ethmoiditis, cases illustrating (W. Stuart-Low), Laryng. 126
Oil, rubbing with, in anorexia nervosa, Child. 80
Okehampton, health resort suitable for foreign invalids, Baln. 21
Old age, pulmonary tuberculosis in, Epid. 35
Olecranon, fractures of, and below, treatment, Surg. 10
OLIVER, GEORGE.—Combination of the auscultatory and tactile methods of reading the arterial blood-pressure, quoted, Med. 151
——, two new methods (auditory and visual) of reading arterial blood-pressure, Clin. 8
Oliver's hæmodynamometer, Med. 76
Olshausen, primary mortality from vaginal hysterectomy, recorded by, Obst. 101
Omentum and small intestine, suppurating ovarian tumour perforated into, successfully removed with affected bowel, omentum and appendix, Obst. 157
Omnopon, see *Pantopon*
Onychia, syphilitic, rare form of, case of (J. E. R. McDonagh), Derm. 55
Open-air galleries, substitution for winter gardens at British spas, recommended, Baln. 15
Operation, new, for radical treatment of flat pelvis (E. W. H. Groves), Obst. 264-270
Operations (abdominal) followed by gastric or intestinal hæmorrhages (probably of septic origin) (J. Hutchinson), Surg. 58-64
——, dilatation of stomach following, Electr. 41
—— (short), anæsthesia by nitrous oxide and oxygen suitable for, Anæsth. 3
—— under anæsthetics, after-effects, Anæsth. 2
—— (urgent), hypnotic anæsthesia not yet suitable for, Anæsth. 23
Ophthalmitis (metastatic), association with arthritis of left wrist-joint, Path. 242, 243
—— —— and arthritis of left knee-joint, simultaneous onset without constitutional symptoms, Path. 242

Ophthalmitis (metastatic), see also *Pseudoglioma*
Ophthalmoplegia externa, complete, with unilateral optic neuritis resulting from acute sphenoidal sinusitis (H. J. Davis), Laryng. 28
Opitz, extraperitoneal Cæsarean section, quoted, Obst. 165
Opium, effect of, on ecbolic action of ergot, Therap. 130-132
Optic atrophy, see *Nerve* (optic), atrophy
Orang, peculiar callosities in, example of, Path. 312
Orbit and eye, affections of, in relation to diseases of nose and accessory sinuses (opened by StC. Thomson), Laryng. 161-168
——, each, invaded by lymphoma causing double proptosis (L. V. Cargill), Clin. 53
—— and right frontal region affected by morphœa (James Galloway), Clin. 27
Ord, W. M., "Influence of colloids on crystalline form," quoted, Path. 136
——, and Shattock, S. G., microscopic structure of urinary calculi composed of calcium oxalate, quoted, Path. 111
Organs, deeply seated, association of surface areas of body with, Baln. 92, 93
——, disused or mutilated, non-transmission of negative results ensuing from, Path. 308
Oriental races, susceptible to suggestion. Anæsth. 23
ORMEROD, J. A.—Discussion on case of syringo-bulbia, Neur. 27
——, discussion on case of syringomyelia, with symptoms precipitated by trauma, Neur. 39
—— —— —— of toxic neuritis of doubtful nature, Neur. 21
—— —— on cases of spondylose rhizomélique and on spondylitis, with progressive muscular contracture, Neur. 45
—— —— on traumatic neuritis of inner cord (Klumpke-paralysis), prefixed type of plexus, Neur. 31
——, two theories of hysteria, Presidential Address, Neurological Section, Neur. 1-18
Ornaments dependent from lobule of ear, effects of wearing, Otol. 81
Ornithorhynchus, cochlea of, in relation to that of other mammals, Otol. 115
Oro-œsophago-gastric tube, permanent styletted, in situ for thirteen weeks (W. Hill), Laryng. 113
Orth, inhalation theory in phthisis, Child. 228
Orthopædic operations, relations of, to that of nerve-root division, Surg. 222-224
Orthopnœa, accompanying pericarditis, explanation, Med. 45
ORTON, G. HARRISON.—Discussion on demonstration of new tube-stand, Electr. 32
——, discussion on Röntgen ray examination of œsophagus, Elect. 27
—— —— on a sesamoid bone fractured by direct violence, Electr. 98
Os calcis, exostoses or osteomata of, as cause of painful heel, Med. 3
Osgood, experimental work on transmissibility of poliomyelitis, Epid. 199
Osler. Professor Sir W., Bt., F.R.S., local prevalence of primary cancer of lung among workers in cobalt mines at Schneeberg, quoted, Path. 78
Osmotic growths (W. D. Butcher), Electr. 93-96
—— pressure of medicinal waters, Baln. 108
"Ossific diathesis," of Virchow, Surg. 137
Ossification in brachialis anticus, Surg. 136, 139
—— of muscles, Surg. 133
——, re-absorption of bone after, Surg. 138
Osteitis deformans, case of (E. M. Corner), Clin. 160
—— (post-typhoidal), recurrent attacks of (H. D. Rolleston), Clin. 156
—— (syphilitic) of femur, case of (P. M. Heath), Child. 103
Osteo-arthritis, abandonment of term suggested, Baln. 34
—— and the arthropathies, essential difference between, Baln. 24
—— and hæmophilia, Baln. 24
——, arterio-sclerosis as cause of, Baln. 28
——, bone-changes in, compensatory, Baln. 29
——, cases illustrating development from different causes, but similarity in appearance, Baln. 30, 31

Osteo-arthritis, changes originate in cartilage, Baln. 24
——, conditions found at different stages of, Baln. 24
——, definition of, Baln. 23
——, diagnosis by "scrunch" heard in stiff joints by auscultation, Med. 38
—— distinct from rheumatoid arthritis, Med. 38
—— due to osteo-myelitis, Baln. 24
—— (gouty), Baln. 25
—— ——, cases of, Baln. 25, 26, 27
——, identity of Charcot's disease with, insisted upon by Virchow, Baln. 24
——, lowered resistance to, in joints, conditions tending to, Baln. 28
—— of hips and knee following tuberculous disease of some joints, Baln. 24
—— of infective origin, Baln. 24
—— of knee-joint (incipient), abnormal sounds heard under auscultation, Med. 37
—— of spine, Clin. 149
——, relation to arthropathies, Baln. 24
——, see also *Fingers*; *Hip-joints*; *Knee-joints*; osteo-arthritis of
Osteogenesis, imperfect, case of (F. J. Poynton), Clin. 105
——, imperfect? case for diagnosis (F. Kidd), Clin. 106
Osteoma, cancellous, callus assuming form of, Surg. 138
Osteomalacic pelvis (S. J. Cameron), Obst. 151
Osteomata, or exostosis of os calcis as cause of painful heel, Med. 3
Osteomyelitis complicating radical operation for frontal sinus suppuration, Laryng. 134, 135, 139, 145, 146, 147
—— following operation on maxillary antra, Laryng. 143
——, osteo-arthritis following, Baln. 24
Osteophytes, development of, in osteo-arthritis of knees and hips, Baln. 29, 33
Osteophytic spicules on inner surface of parietal bone from case of atypical chloroma, Path. 62
Osteotomy, supracondylar, when permissible, Surg. 10
Otalgia, with history of syphilis, examination of post-nasal space in, Otol. 23
Othæmatoma, "spontaneous," Otol. 106
——, syphilis and insanity, relations between, Otol. 104, 106
——, traumatic, Otol. 80, 81
Otic element of Arctic whale, eight specimens of (F. H. Westmacott), Otol. 124
Otitis media (acute) followed by lateral sinus thrombosis; operation; recovery (W. J. Chichele Nourse), Otol. 56
—— —— ——, following condylomata of external auditory meatus, Otol. 11
—— ——, suppurative, chronic; necrosis of external semicircular canal; fistula full of bone granulation tissue; operation; recovery (W. Milligan), Otol. 65
—— ——, chronic suppurative, followed by cerebellar abscess (W. Milligan), Otol. 61
—— ——, suppurative, following injury to tympanic membrane, Otol. 83
——, syphilis in relation to (A. H. Cheatle), Otol. 1-24
Otosclerosis not due to syphilis, Otol. 3, 21
de Ott, Professor Dmitri, practice as regards removal of appendix in pelvic operations, Obst. 352
——, primary mortality from vaginal hysterectomy, Obst. 101
Ovarian extract in exophthalmic goitre, Clin. 124
Ovaries (both), malignant papillomatous cysts of, removal followed six years later by carcinoma of rectum (M. A. Scharlieb), Obst. 358
——, diseases of, communications summarized (1909-10), Obst. 15
——, preservation in operations for uterine myomata, Obst. 117, 118, 129, 134, 144, 145
——, transplantation of, experimental work, Obst. 116
——, and uterus, adeno-carcinoma of, case (M. A. Scharlieb), Obst. 359
Ovariotomy during labour, cases recorded, Obst. 256-259

Ovariotomy (abdominal) during labour for removal of dermoid tumour of right ovary (G. Blacker), Obst. 250
Ovary and appendix, paths of infection between, Obst. 343
—— and peritoneum, cysts of, with hydrosalpinx (H. Macnaughton-Jones), Obst. 221
——, carcinoma (columnar-celled) of (H. P. Dimmock), Obst. 62
—— —— ——, report of Pathological Committee on, Obst. 63
——, connexion of rheumatoid arthritis with, presumed, Baln. 36
——, cyst of, involution of, associated with vesicular mole, clinical history of case (R. D. Maxwell), Obst. 219
—— ——, suppurating, in girl, aged 10, probably from hairpins impacted in vagina (H. J. Paterson), Obst. 259
—— cysts of, in children (not including dermoids) (H. J. Paterson), Obst. 262
——, fibroma of, diffuse, with central degeneration, with extravasation of blood (R. H. Paramore), Obst. 248
—— ——, with twisted pedicle (M. A. Scharlieb), Obst. 35
——, follicle cells of, origin of, demonstration on (A. Louise McIlroy), Obst. 226
——, pathological specimens, exhibited (1909-10), summarized, Obst. 13
—— (right), dermoid tumour of, removed by abdominal ovariotomy during labour (G. Blacker), Obst. 250
——, sarcoma of, diagnosis aided by rectal examination, Obst. 330
——, teratoma of (J. E. Gemmell), Obst. 285
——, tumours of, malignancy in, communications on (1909-10), summarized, Obst. 10
—— ——, obstructing delivery and fixed by suction in pelvis (J. D. Malcolm), Obst. 253
—— —— (suppurating), perforated into small intestine and omentum, successfully removed with affected bowel, omentum, and appendix (H. R. Spencer), Obst. 157
Ovum, early human, microscopical appearances, Obst. 299, 300
—— (tubal, very young), development of amnion, Obst. 321
—— —— ——, specimen of, age discussed, Obst. 319
—— —— —— ——, demonstration of (Miss F. Ivens), Obst. 313
Oxalate of lime calculi, prevention of formation, Baln. 124
Oxalic acid poisoning, cause of, Baln. 124
Oxaluria, effects of Vittel waters on subjects of, Baln. 124
——, treatment of, Baln. 124
Oxidizing effect of sulphur water on tissues, Baln. 62
Oxygen and nitrogen as constituents of air, discovery of, Baln. 3
—— as constituent of air, discovery of, Baln. 3
Oxygen capacity, total, estimation in case of congenital pulmonary stenosis, Clin. 88, 89
—— ——, hæmoglobin of, Clin. 86

Pachydermia laryngis, inequality of planes of vocal cords in, Laryng. 63
Paddington Dispensary for the Prevention of Consumption, results of examination of contacts, Epid. 37, 46
Page, C. M., and Cassidy, M. A.—Method for determining the absolute pressure of the cerebro-spinal fluid, Clin. 56
Paget, Sir James, view regarding hysteria, quoted, Neur. 2
Pain caused by condylomata, Otol. 20, 21, 23
—— (chronic abdominal), in chronic endometritis, results of treatment by curetting, Obst. 45,
——, consciousness to, voluntary loss of, Anæsth. 19
—— —— —— —— of among Christian Scientists, Anæsth. 19
——, insensibility to, resulting from inhibitory mental processes, Anæsth. 15
—— in visceral and gastric crises, simulating that of organic disease, Surg. 227
—— (local), fixed idea of, hysterical symptom, Neur. 8, 9
——, relief of, by division of posterior spinal nerve-roots, Surg. 199, 248
Paisley, outbreak of milk-borne sore throat at, Epid. 95

Palate (cleft), associated with harelip, Surg. 180, 181
—— ——, complications in non-operated cases, Surg. 193, 194
—— ——, factors affecting development of nasopharynx in relation to, Surg. 170
—— ——, mortality of non-operated cases, Surg. 170, 189, 190
—— ——, treatment, operative, age for, Surg. 170, 174, 184, 189, 191-194
—— —— —— —— by Arbuthnot Lane's (flap) method, Surg. 171, 173, 175, 176, 180, 181, 184, 189-197
—— —— —— —— by Brophy's method, Surg. 173, 175, 177, 180, 185, 197
—— —— —— —— by Langenbeck's method, Surg. 173, 175, 176, 177, 180, 181, 184, 189, 190
—— —— —— —— by prothesis, Surg. 179
—— —— —— —— care of patients previous to, Surg. 191
—— —— —— —— deferred, Surg. 170, 182, 183, 190, 192-194
—— —— —— ——, demonstration of cases, Surg. 169
—— —— —— —— in infancy, Surg. 170-172, 181-183, 190, 192-195
—— —— —— —— ——, mortality, Surg. 195, 197
—— —— —— —— instruments for, Surg. 171, 174, 176, 180, 191
—— —— —— —— —— for compression of jaws in, Surg. 178
—— —— —— —— results, Surg. 178, 179, 181, 195
—— —— —— —— —— as regards speech, Surg. 186-188, 190, 194, 195
—— —— —— —— ——, report on fifty-six cases (J. Ulrich), Surg. 173-180
—— —— —— ——, speech training after, Surg. 185, 186
—— —— —— ——, State support for, in Denmark, Surg. 174
—— —— —— ——, statistics, Surg. 182
——, congenital, anomalies of (A. R. Tweedie and A. Keith), Laryng. 47-54
——, deformity of, congenital (D. Wood), Child. 5
——, endothelioma of, with metastatic growth in neck, treated by radium (J. Gay French) Laryng. 94
—— (hard, cleft), Surg. 190
——, pharynx, external ear, &c., involved in case of leprosy (G. N. Biggs), Otol. 120
—— (soft), adhesion to pharyngeal wall causing pain in ears, Otol. 13
—— ——, (cleft), Surg. 190
—— ——, congenital insufficiency of (L. G. Guthrie), Child. 23
—— ——, perforation of, with unusual features (W. J. Horne), Laryng. 116
—— ——, pharynx, and larynx, tuberculosis of, case of (W. G. Howarth), Laryng. 66
—— ——, rhythmical elevations accompanied by rhythmical clickings in Eustachian tube and tympanum on left side in syphilitic, Otol. 13
—— ——, tumour of, removal under application of diathermic current, Electr. 8
—— —— —— small (G. W. Badgerow), Laryng. 13
—— ——, ulceration of (H. FitzGerald Powell and G. W. Badgerow), Laryng. 11
Palato-glossal fold, right, epithelioma on, in man, aged 48 (W. Stuart-Low), Laryng. 60
Palleri and Mergani, blood-changes in congenital pulmonary stenosis, Clin. 91
Palmer, A., case of pseudo-hemaphroditism, quoted, Obst. 62
Palms and soles, hyperkeratosis of (H. G. Adamson), Derm. 18
Panhysterectomy, see *Hysterectomy*, total
Panada as article of diet for young children in eighteenth century, Child. 122, 123
Pancreas and meat, absorption by rectum, Therap. 64
Pancreatic extract, milk and eggs, absorption in rectal feeding, Therap. 64
Panophthalmitis due to oral sepsis, fatal case, Odont. 31, 32
—— following septicæmia, Odont. 28
—— from streptococcal infection, Path. 247, 248
Pansinusitis, treatment, operative, risks of, Laryng. 143
—— (unilateral), treatment by Killian's operation, cases illustrating (StC. Thomson), Laryng. 123, 124
Pantopon, hypodermic injection before inhalation anæsthesia, Anæsth. 44, 45, 48
Pap-boat and pap-spoon for feeding infants, Child. 132

Papillæ formation in colonies of bacteria grown on monochloracetic acid medium, Path. 99, 100, 107

—— —— —— —— —— —— ——, significance of. Path. 107

Papilloma of larynx in a child (D. Mackenzie), Laryng. 80

—— of pharynx, case of (G. C. Cathcart), Laryng. 62

——, see also *Cysts* (papillomatous, malignant)

Parakeratosis, Derm. 32

—— variegata (?), case of (A. Whitfield), Derm. 30

—— ——, synonym for, Derm. 31

Paralysis (abductor), unilateral, in man, corresponds to "roaring" in horses, Laryng. 90

——, area of, in oculo-motor type of polio-encephalitis, Child. 90, 93

—— (epidemic), see *Poliomyelitis*

—— (Erb's), cause of, in infants, Child. 9

—— (facial) due to injuries and lesions of middle and internal ear, Otol. 93

—— ——, due to suppuration of labyrinth; complete bridge operation; recovery (W. Milligan), Otol. 63

—— ——, following attempt to extract supposed foreign body from ear, Otol. 82

—— ——, following gunshot injury to ear, Otol. 99, 100

—— —— in case of internal ear deafness, Otol. 17

—— ——, treatment, Otol. 103

—— and hemiplegia (post-diphtheritic), case of, Child. 195

—— (general, of insane), early case, with deafness and tinnitus, Otol. 18

—— (hysterical), due to idea, Neur. 27

—— ——, affecting bodily function, Neur. 7, 8

—— (Klumpke's), traumatic neuritis of inner cord (W. Harris), Neur. 31

—— (motor, hysterical), Neur. 27

—— of right third nerve following ethmoidal operation (A. J. M. Wright), Laryng. 159

—— (peripheral, incipient and reflex), treatment by subthermal baths, Baln. 96

—— (pseudo-bulbar), case of doubtful origin (T. Grainger Stewart), Neur. 23

—— —— of doubtful origin, probably disseminated sclerosis (T. Grainger Stewart), Neur. 46

—— (spastic), treatment by division of posterior spinal nerve-roots, Surg. 229

—— —— —— —— —— —— ——, after-treatment of cases, Surg. 244

—— —— —— —— —— —— ——, cases, Surg. 231-243, 247-251

—— (spastic, infantile), case of (G. S. Haynes, for C. Searle), Child. 192

—— (unilateral) of ninth, tenth, and eleventh cranial nerves (P. W. Saunders, for James Taylor), Neur. 51

Paramé, Baln. 18

PARAMORE, R. H.—Diffuse fibroma of the ovary, which has undergone central degeneration with extravasation of blood, Obst. 248

Paraplegia (hysterical), Neur. 7

Paratyphoid, typhoid and allied organisms, strains of, complement fixation with, studies in (H. R. Dean), Path. 251-278

—— bacillus, carriers of, experimental production (G. F. Petrie and R. A. O'Brien), Path. 70-72

—— fever, ætiology of (F. A. Bainbridge), Epid. 51

—— —— and Malta fever, mixed infections of, Epid. 118

—— —— and typhoid fever, mixed infections of, Epid. 118

—— ——, bacteriology of, Epid. 51, 52

—— ——, clinically indistinguishable from typhoid fever, Epid. 63

—— ——, epidemiology (F. A. Bainbridge), Epid. 53

—— —— (A), geographical distribution, Epid. 53, 54

—— —— (B), carriers of, Epid. 56, 57, 64

—— —— —— ——, greater frequency among women, Epid. 56

—— —— —— ——, infection from, Epid. 56, 57, 64

—— —— ——, clinical characteristics, Epid. 67, 68

Paré, Ambroise, advocacy of cold water dressings in surgery, Baln. 87

Parental constituents of somatic cells, Med. 6

Parhon and Goldstein, first case of amaurotic idiocy (Tay-Sachs disease) observed in Roumania, reported by, Path. 169
Parietal bone, osteophytic spicules on inner surface of, in case of atypical chloroma, Path. 62
Paris, hospitals of, mortality from measles at, Child. 174
——, paratyphoid fever (A) in, Epid. 54
——, teaching of medical hydrology in, Baln. 113, 114
—— —— —— ——, considered deficient, Baln. 114
Paris Foundling Hospital, infant mortality at (1771-1877 and 1820-1822), Child. 130
Park, investigations as to typhoid carriers, quoted, Epid. 12, 13
PARKER, C. A.—Case of chronic tuberculosis of the nose, larynx and lungs, Laryng. 64
——, discussion on case for diagnosis, Laryng. 15
——, specimen of tonsillolith, Laryng. 33
——, tonsillolith in left supra-tonsillar fossa, Laryng. 12
Parker, G., epidemic of poliomyelitis at Bristol (1909), reported by, Epid. 212
PARKER, RUSHTON.—Discussion on fractures in neighbourhood of joints, Surg. 23
Parkes, effect of hard water on horses, quoted, Baln. 122
PARKINSON, J. PORTER.—Case of congenital dilatation of the colon, Child. 75
——, discussion on case of enlargement of liver, Child. 55
—— —— —— of rheumatoid arthritis, Child. 37
—— —— —— of sclerema neonatorum, Child. 6
—— —— —— of splenomegaly with recurrent jaundice, Child. 69
—— —— on specimen of congenital malformation of heart, Child. 4
—— —— on two cases of infantilism, Child. 60
—— —— on ulcerative stomatitis in children, Child. 73
Parotid gland, facial, cystic adenoma of (W. Stuart-Low), Laryng. 59
Parrel and Castex, syphilitic showing rhythmical elevations of soft palate accompanied by rhythmical clickings in Eustachian tube and tympanum on left side, Otol. 13
Parry, adoption of inguinal method of radical cure of femoral hernia, quoted, Surg. 166
PARSONS, F. G.—Discussion on diseases of cow affecting milk in relation to human disease, Epid. 101
Parsons, J. H., pathology of amaurotic family idiocy, quoted, Path. 171
Partick, food-poisoning at, following eating of canned meat, Epid. 66
Parturition, hypodermic injection of morphine before chloroform inhalation in, Anæsth. 43
——, stricture of rectum due to, Obst. 336
Passive movements combined with massage in early stage of fractures about elbow-joint injurious, Surg. 7
—— —— in treatment of fractures about joints, Surg. 22, 23, 27, 32, 36, 37, 40, 43, 46
Passler, cause of heart-failure in acute disease, quoted, Baln. 93
Passow, case of rupture of tympanic membrane, quoted, Otol. 95
——, fatal attempts to extract foreign body from ear, Otol. 97
Pasteur, L., experimental production of anthrax by, Child. 218
——, infective virus of lowered intensity regaining virulent power, quoted, Epid. 195
Pasteur, W., absorption of salt and water in rectal feeding, quoted, Therap. 67
——, Bradshaw Lecture on massive collapse of lung, quoted, Med. 40
——, epidemic poliomyelitis at Much Hadham, reported by, Epid. 209
Pastille doses, Electr. 61
Patella, fracture of, advantages of non-operative treatment, Surg. 33, 34
—— ——, liability to re-fracture after non-operative treatment, Surg. 42
—— ——, treatment of, Surg. 40, 41
—— —— ——, operative, Surg. 26, 30
—— —— (transverse), difficulties in treatment, Surg. 78
—— —— ——, fibrous union unsatisfactory, Surg. 80, 81
—— —— ——, likelihood of, not excluded by walking of patient, Surg. 78, 79
—— —— ——, without separation of the fragments (A. E. Johnson), Surg. 79-82, 84
—— —— —— —— —— ——, danger of failure to diagnose, Surg. 79, 80

Patella, fracture of (transverse), without separation of the fragments, importance of X-ray examination, Surg. 79, 80-82
——, "tear fracture" of, Surg. 75, 79
Patent foods for children, origin and history of, Child. 135, 136
Paterson, Professor, drawing of dissection showing appendiculo-ovarian ligament, Obst. 345
PATERSON, D. R.—Deafness due to occlusion of Eustachian tubes by scar-tissue, Otol. 109
——, discussion on case of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 103
—— —— on case of removal of rabbit-bone from right bronchus, Laryng. 98
—— —— on report on fatal termination of case of deafness, Otol. 68
—— —— on specimen of pedunculated growth on an elongated uvula, Laryng. 107
—— —— on treatment of frontal sinus discharge, Laryng. 143
—— —— on tumour of right side of neck involving pharynx, Laryng. 100
PATERSON, H. J.—Discussion on fractures in neighbourhood of joints, Surg. 31-34
——, suppurating ovarian cyst in a girl, aged 10, probably infected from hairpins impacted in the vagina, Obst. 259
PATERSON, W. B.—Discussion on inflammatory eye conditions due to oral sepsis, Odont. 33
Pathology (chemical), advance in (1811), Med. 151
Pathology Committee (Obstetrical and Gynæcological Section), reports: columnar-celled carcinoma of ovary (Lieut.-Col. Dimmock), Obst. 63
—— —— —— —— —— ——, melanotic sarcoma of vulva (C. White), Obst. 68
PATTERSON, NORMAN.—Denture removed from œsophagus, Laryng. 54
——, epithelioma of the epiglottis, case of, Laryng. 83
——, primary sore of the vestibule of the nose and adjoining skin, Laryng. 84
Pau, suitable health resort for neurasthenics, Baln. 42
Paukul, experimental work on auricular ventricular bundle, Therap. 98
Pausanias, description of spa treatment in days of Hippocrates, quoted, Baln. 9
Pavlov, acidity of native gastric juice, quoted, Med. 26
——, quasi-independence of acidogenic and pepsinogenic functions of gastric glands, Med. 27
Pawlow, see above, *Pavlov*
PAYNE, J. LEWIN.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 49
——, discussion on distribution of nerve-fibres of dental pulp, Odont. 55
Peachey, John, "General Treatise of the Diseases of Infants and Children" (1697), Child. 118
——, on suckling and weaning, quoted, Child. 118, 119, 120
PEACOCK, C. N.—Discussion on comparative elasticity and internal structure of cast and rolled gold plate, Odont. 66
Pearl Life Assurance Company, mortality records of tuberculosis in relation to family predisposition, Epid. 37
Pearson, Karl, F.R.S., on heredity in phthisis, Epid. 173, 174
Peckham House Asylum, percentage incidence of gall-stones based upon post-mortem records of, Path. 88
Pediculi of scalp causing infection of cervical glands with tuberculous disease, Child, 223, 224, 232
Pediluvia, hot, value in defective cutaneous circulation, Baln. 73
PEGLER, L. H.—Case of party-wall cancer of larynx and œsophagus, Laryng. 101
—— case of tuberculosis of the vocal cords in a young man, Laryng. 76
——, discussion on case of aphonia, Laryng. 42
—— —— —— of tuberculosis of nose and cheek, Laryng. 85
—— —— on cases illustrating results of removing nodules from cords by means of galvano-cautery (by indirect method), Laryng. 41
——, extensive malignant ulceration of pharynx and tonsils, Laryng. 78
——, functional aphonia in a boy, aged 15, Laryng. 115, 116
——, tuberculous ulceration of the larynx, Laryng. 78
——. ulceration of pharynx, due to pyorrhœa, Laryng. 100

Pels-Leusden, ætiology of multiple cartilaginous exostoses, quoted, Med. 3
Pelvic operations, hypodermic injection of scopolamine and atropine before chloroform inhalation in, Anæsth. 50
—— organs, operation on, question of removal of appendix in, Obst. 350-353
Pelvis, asymmetry of (Naegele), with partial congenital suppression of left lateral wing of the sacrum and scoliosis (A. H. Tubby), Child. 163
—— (contracted), operative procedures in, communications on (1909-10), summarized, Obst. 5
—— ——, with protracted labour, bacteriological examination of cases, Obst. 169
—— —— —— ——, treatment by Cæsarean section and alternative operations, Obst. 161, 172
—— —— —— —— —— —— —— —— —— in various types of suspected septic cases, Obst. 172
——, dissections of, in case of cancer of uterus showing distribution of lymphatic glands, Obst. 74, 75
—— (flat), new operation for radical treatment of (E. W. H. Groves), Obst. 264-270
——, lymphatic vessels of, Obst. 72
—— (osteomalacic) (S. J. Cameron), Obst. 151
——, ovarian tumour fixed by suction in, and obstructing delivery (J. D. Malcolm), Obst. 253
——, see also *Bones* (pelvic)
Pembrey, M. S., supposed primary abdominal pregnancy in rabbit, quoted, Obst. 233
Pemphigus following ptomaine poisoning, Med. 32
—— foliaceus, dermatitis herpetiformis mistaken for, Derm. 17
—— —— of fifteen years' duration, Nikolsky's sign present in, Derm. 15
—— —— of sixteen years' duration (H. G. Adamson), Derm. 15
—— of pharynx and conjunctiva (P. Watson-Williams), Laryng. 158
Pen-infection in relation to pulmonary tuberculosis, Child. 224
PENFOLD, W. J.—Fermentation of lactose by *Bacillus typhosus*, Epid. 70
——, discussion on typhoid carriers and contact infection, Epid. 142
——, variability in the gas-forming powers of intestinal bacteria, Path. 97-109
Penn, William, on treatment of fever among North-American Indians (1683), Baln. 89
Penny, on intermittent albuminuria, quoted, Med. 113, 115
Penzance, Baln. 17
Pepsinogenic and acidogenic functions of gastric glands, Med. 27
Peptic values, see *Stomach contents*, peptic values of
Péraire and Terrier, injection of morphine and atropine before chloroform inhalation, Anæsth. 44
Perception, comprehensiveness of, Neur. 3
Percussion, date of introduction, Med. 150
Pereira, action of aconite, quoted, Therap. 157
Pericardium, adherent, and chronic cyanosis, case of, blood-changes in, Clin. 92
—— ——, cardiolysis for, case of (G. A. Sutherland), Child. 41
Pericarditis complicated by peritonitis, Med. 49
—— (mediastinal), case of, blood-changes in, Clin. 91
——, peritonitis mistaken for, Med. 48
—— (rheumatic) complicated by pleurisy, Med. 49, 50
—— ——, cyanosis, dyspnœa, and upright position of patient accompanying, explanation of, Med. 45
—— ——, high inspiratory position of diaphragm in, Med. 49
—— ——, indicated by absence of abdominal respiratory movement (W. E. Wynter), Med. 40
—— —— —— —— —— —— ——, illustrative cases, Med. 40-44, 46, 47
—— —— —— —— —— ——, more marked in fibrinous than effusion form, Med. 45
—— ——, position of friction sounds in, Med. 48
—— —— —— of præcordial impulse in, Med. 49
—— ——, presence of abdominal respiratory movement in, Med. 47
——, with effusion, differential diagnosis from dilatation of heart, Med. 44, 48

Perimeter test for implication of optic nerve in diseases of the sinuses, Laryng. 163, 164-166
Periosteum, injury to, essential factor in causation of traumatic myositis ossificans, Surg. 154, 156
—— separated by injury, formation of bone in (Sir A. A. Bowlby), Surg. 142
—— —— by muscles, resulting in formation of swelling, Surg. 143
Periotic bones in fossil Cetacea (W. Jobson Horne), Otol. 114
Peripheral field, anomalies of, not reflex in origin, Laryng. 165
—— —— contraction, occurrence of, in sinusitis, Laryng. 163, 164, 166
—— —— ——, value of, in diagnosis of sinusitis, Laryng. 166, 167
Peripheral resistance, increased, at high altitudes, factor in causation of rise in low blood-pressure, Med. 59
Peristalsis of œsophagus, Electr. 14, 28, 29
—— ——, reversed, Electr. 27
Peritoneum and ovary, cysts of, with hydrosalpinx (H. Macnaughton-Jones), Obst. 221
——, infection of, how to avoid, in Cæsarean section in suspected septic cases, Obst. 191
—— on pelvic floor, treatment in abdominal panhysterectomy, Obst. 179
Peritonitis complicating pericarditis, Med. 49
—— (acute), fatal, following Cæsarean section, with septic infection, Obst. 186, 215
——, fatal, resulting from liquor amnii escaping into abdominal cavity, Obst. 196, 209
—— mistaken for pericarditis, Med. 48
—— (tuberculous), enlargement of Fallopian tubes in, Child. 226
—— ——, proportion of deaths from, Child. 231
—— ——, severe, case of, cured by laparotomy (P. Lockhart Mummery), Child. 186
Perkins and Hektoen, case of sporotrichosis, quoted, Derm. 114
PERNET, G.—A coloured drawing of nodular lupus erythematosus, Derm. 108
——, discussion on calcareous (hard) drinking water, Baln. 127
—— —— on cases for diagnosis, Derm. 43, 53, 63, 71, 102
—— —— —— of acute lupus erythematosus, Derm. 51, 93, 107
—— —— —— of auto-reinfection, Derm. 131
—— —— —— of cicatrizing sarcoma, Derm. 128
—— —— —— of congenital nævoid condition of left thigh, Child. 194
—— —— —— of disseminated tuberculous nodules of skin, Derm. 50
—— —— —— of elephantiasis of lip, Derm. 58
—— —— —— of epithelioma of back of hand, Derm. 142
—— —— —— of (?) granuloma inguinale, Derm. 136
—— —— —— of ichthyosis in child treated by thyroid extract, Derm. 73
—— —— —— of leucoderma syphiliticum, Derm. 103
—— —— —— of (?) mycosis fungoides, Derm. 78
—— —— on macular atrophy in syphilitic patients, Derm. 125
—— —— —— showing keloids forming on cicatrices resulting from ulcerating syphilitic lesions, Derm. 75
—— —— on *Trichophyton roseum* and *ectothrix*, Derm. 94
Peroneal type of muscular atrophy (Charcot-Marie, Tooth), two cases of, in father and son, (L. G. Guthrie), Clin. 126
"Perrin" type of sarcomatosis cutis (E. G. Graham Little), Derm. 22
Personality, human, dissociation of, Neur. 3
—— ——, origin, Neur. 3
Pessaries (vaginal), dangers of, Obst. 324
Pessary, see *Ring Pessary*
Pestalozza, Cæsarean section and its alternatives, quoted, Obst. 165
PETERS, E. A.—Cases illustrating results of removing nodules from the cords by means of galvano-cautery (by the indirect method), Laryng. 40
——, discussion on case of bilateral tuberculous disease of tonsils, Laryng. 68
—— —— on new type of œsophago-gastroscope, Laryng. 73
——, primary carcinoma of tonsil, case of, Laryng. 79

PETRIE, G. F., and O'BRIEN, R. A.—Epizootic in guinea-pigs, quoted, Epid. 124, 136
—— ——, the experimental production of the carrier state by feeding, Path. 70
Petrous bone, fractures of, aural complications, Otol. 88, 89
—— —— ——, symptoms, Otol. 90
—— —— ——, treatment, Otol. 101
Pharmacology, development of the new school of, Therap. 3
—— studied apart from practice of medicine, Therap. 2, 3
Pharyngitis (syphilitic), Eustachian deafness rare in, Otol. 19
Pharyngoscope (Hay's), improved form of (H. Tilley), Laryng. 108
Pharynx, adhesion of soft palate to wall of, causing pain in ears, Otol. 13
—— and conjunctiva, pemphigus of (P. Watson-Williams), Laryng. 158
—— and tonsils, extensive malignant ulceration of, case of (L. H. Pegler), Laryng. 78
——, cancer of deep portion, with extension to gullet and posterior margin of larynx (W. Hill), Laryng. 44
—— —— (party-wall), case of (C. Graham), Laryng. 55
—— ——, treatment by radium, cases reported, Electr. 64, 66
——, involvement of, by tumour of right side of neck, in woman, aged 30 (W. H. Kelson), Laryng. 99
——, larynx, and soft palate, tuberculosis of, case of (W. G. Howarth), Laryng. 66
——, lower part, larynx, and upper end of œsophagus, excision for malignant disease (squamous-celled carcinoma), subsequent report on case (A. Evans), Clin. 142
——, malignant disease of, two cases (P. R. W. de Santi), Laryng. 113, 114
—— —— ——, under treatment by radium (R. H. Scanes Spicer), Laryng. 64
——, palate, external ears, &c., involved in case of leprosy (G. N. Biggs), Otol. 120
——, papilloma of, case of (G. C. Cathcart), Laryng. 62
——, tertiary specific ulcer of (P. Watson-Williams), Laryng. 158
——, ulceration of, due to pyorrhœa (L. H. Pegler), Laryng. 100
Phenacetin in acute frontal sinusitis, Laryng. 127
Phenazone in acute frontal sinusitis, Laryng. 127
Phillips, Professor L., "The treatment of tetanus by the intraspinal injections of solution of magnesium sulphate," Med. 153
Phlebitis (double) following hysterectomy (H. Macnaughton-Jones), Obst. 223
Phlegmatic type, individuals of, good subjects for hypnotism, Anæsth. 18
"Phobias," defence neuroses, Neur. 12
Phosphates, estimation by acetate and uranium method, Baln. 47
——, excretion of, before and after taking sulphur water, Baln. 53, 54
—— not harmful to teeth, Odont. 97
Phosphaturia, low blood-pressure in subjects of, Med. 74
Phosphorescent organs, blind fish provided with, Path. 308
Phosphorus in brain-tissue, Path. 196
—— —— in amaurotic idiocy (Tay-Sachs disease), diminution of lipoid and increase of extractive forms, Path. 198
Phthisis, see *Tuberculosis* (pulmonary)
Physical therapy, lectures on, at Bologna, Baln. 116
—— —— ——, at Zürich, Baln. 116
Pickling, *Bacillus enteritidis* (Gaertner) resistant to, Epid. 69
Picqué, case of gunshot injury to ear, Otol. 99
Pierce, P. W. C., outbreak of milk-borne sore throat at Guildford, reported by, Epid. 93
Pigmentation in unusual case of linear nævus, Derm. 39
—— present in case for diagnosis (E. G. Graham Little), Derm. 145
Pigs, fæces of, *Bacillus paratyphosus* B not found in, Epid. 55
——, healthy, organism identical with *Bacillus paratyphosus* A found in intestines of, Epid. 54
—— ——, presence of *Bacillus suipestifer* in alimentary canal of, Epid. 58, 59
Piles (œsophageal), causing obstruction of œsophagus, Electr. 17

Piles, see also *Hæmorrhoids*
Pilocarpine, hypodermic injections of, in treatment of congenital syphilitic deafness, Otol. 9
Pinna, dermatitis (chronic) of, due to infection by *Staphylococcus aureus*, under treatment by vaccine (C. E. West), Otol. 38
Pirie, A. H.—Discussion on diathermic treatment, Electr. 8
——, discussion on new induction coil, Electr. 83
—— —— on Röntgen ray examination of œsophagus, Electr. 28
—— —— on a sesamoid bone fractured by direct violence, Electr. 98
—— —— on success or failure in electrotherapy, Electr. 114
Pirie's stereoscope, Electr. 92
Pitcairn, cold and tepid applications employed by, Baln. 83
Pitt, investigation of *Bacillus nodulifaciens bovis*, Path. 253
Pitts, A. T.—Discussion on a dentigerous cyst with cellular contents, Odont. 89
Pituitary body, anterior, disease of, associated with acromegaly, Odont. 26
—— ——, ectopia of, with other congenital anomalies of nose, palate, and upper lip (A. R. Tweedie and A. Keith), Laryng. 47-52
—— extract, effect on low blood-pressure, Med. 68
Pituitrin, reaction of arteries to, Therap. 85
Pityriasis, acute, of scalp (T. Colcott Fox), Derm. 140
—— following X-ray treatment for ringworm, Derm. 141
—— rubra pilaris and follicular psoriasis, diagnosis between, Derm. 98, 100
—— —— ——, cases of (E. G. Graham Little), Derm. 23
—— —— —— (H. G. Adamson), Derm. 98
Placenta prævia, Cæsarean section in, communications on (1909-10) (summarized), Obst. 7
Plague (bubonic), seasonal prevalence in Punjab, Epid. 225
—— ——, transmission of, method, Epid. 224, 225
——, see also *Athens*, plague of
Plants, changes of habit due to climatic conditions, Path. 308
Plate-holder, adjunct of tube-stand, Electr. 86, 92
——, methods of using, Electr. 87, 88, 92
Plesch, percentage of oxygen capacity and blood per 100 grm. body-weight, recorded by, Clin. 89
Pleura, disease of, cause of unilateral inhibition of action of diaphragm, Med. 40
——, tumours of, pressure from, causing œsophageal obstruction, Electr. 21, 22
Pleurisy complicating rheumatic pericarditis, Med. 49, 50
—— followed by pulmonary tuberculosis, Epid. 48
——, "painful affections of side from tumid spleen" (1811), probably case of, Med. 154
——, treatment by diathermic current indicated in, Electr. 7
Plexus, prefixed type of, in traumatic neuritis of inner cord (W. Harris), Neur. 31
Plica transversalis recti, Surg. 91
Pliny, "Natural history," quoted, Child. 119
Plombières, radio-activity of waters at, Baln. 108
Plombières system, see *Colon*, flushing of
Plugging middle meatus with cocaine and hemisine solution in acute frontal sinusitis, Laryng. 142, 146
—— with adrenalin and cocaine solutions in acute frontal sinusitis, Laryng. 147
Pneumococcal infections leading to bony formation in and around joints, Baln. 24
Pneumococcic infection in human trypanosomiasis, Path. 15
—— serum, see *Serum* (pneumococcic)
Pneumococcus and *Micrococcus catarrhalis*, mixed vaccines of, in treatment of episcleritis due to oral sepsis, Odont. 34
——, see also *Stomatitis* (pneumococcal)
Pneumonokoniosis predisposes to primary cancer of lung, Path. 78
Pneumonia, associated with typhoid fever or measles, followed by pulmonary tuberculosis, Epid. 48
——, bronchitis complicating sign of pulmonary failure, Med. 107

Pneumonia complicating bronchitis, not necessarily fatal, Med. 107
—— —— influenza, Epid. 192, 223
—— —— measles, Epid. 192, 223
——, prevalence of, in relation to weather, Epid. 189
——, treatment by diathermic current indicated in, Electr. 7
Pocock, R. I., function of cutaneous glands in antelope, quoted, Path. 286
Poirier, lymphatic tract (middle) of, Obst. 73
"Poker-back," Clin. 148
Polio-encephalitis, oculo-motor type of (S. Stephenson), Child. 87-93
—— —— ——, area of paralysis in, Child. 90, 93
—— —— ——, diagnosis, Child. 81
—— —— ——, experimental method of determining nature, Child. 94
—— —— ——, frequency in children under 1 year, Child. 89, 92
Poliomyelitis, clinical aspects of, Epid. 198
——, definition of term, Epid. 198
——, diagnosis of progressive spinal muscular atrophy (Werdnig-Hoffmann type) from, Neur. 67
—— —— of toxic neuritis from, Neur. 21, 22, 23
——, distribution of cases in London (1904), Epid. 217, 218, 219
——, epidemics of, age-incidence factor common to all, Epid. 204
—— —— around Melton Mowbray (1910), Epid. 213, 214
—— —— at Barrow-in-Furness (1910), Epid. 212-215
—— —— at Bristol (1909), Epid. 212
—— —— at Carlisle (1910), Epid. 212, 213
—— —— at Cerne Abbas (1910), Epid. 216, 226
—— —— at Harvieston (near Tillicoultry, Clackmannanshire), Epid. 217
—— —— at Irthlingborough (1910), Epid. 214, 215
—— —— at Maryport (1910), Epid. 212
—— —— at Much Hadham, Epid. 209
—— —— at Upminster (1908), Epid. 210
—— —— at Workington (1910), Epid. 212
—— ——, connexion with influenza, Epid. 224
—— ——, history of, Epid. 200
—— —— in Australia, Epid. 201, 203
—— —— ——, map showing, Epid. 203
—— —— in England (1909-10), Epid. 212-216
—— —— —— ——, map showing, Epid. 211
—— —— in Europe, Epid. 200, 201
—— —— ——, map showing, Epid. 201
—— —— in Great Britain, Epid. 209-218
—— —— in Massachusetts (1907-8-9), Epid. 206
—— —— in Steiermark (1909), Epid. 208
—— —— in Steyr (1908), Epid. 209
—— —— in Sweden (1899), Epid. 200
—— —— —— (1905), Epid. 204
—— —— in United States, Epid. 200-203
—— —— —— ——, map showing, Epid. 202
—— —— in Westphalia (1908), Epid. 207
—— —— on west coast of Cumberland, Epid. 212, 214, 226
—— ——, list of, with localities and statistics, Epid. 220, 221
—— ——, mortality factor common to all, Epid. 204
—— ——, seasonal relationship, factor common to all, Epid. 204
—— in colts, Epid. 206, 207
——, infectivity of, Epid. 218, 219, 222, 223
——, notification of, Epid. 219

Poliomyelitis, pathological lesion in, similar to that of rabies, Epid. 200
——, seasonal prevalence, resemblance of that of typhus to, Epid. 225
—— transmissible from man to monkeys by inoculation, Epid. 199
——, transmission of, Epid. 210
—— ——, by animals, possible, Epid. 206
—— —— by bathing in sewage-contaminated water possible, Epid. 206
—— —— by foreign flour, Epid. 216
—— —— by school infection, Epid. 204-206, 208, 209
——, virus of, nature, Epid. 199
—— ——, similarity to that of rabies, Epid. 199, 200
Polish woman, scleroma of nasopharynx in (StC. Thomson), Laryng. 7
Politzer, A., fatal meningitis following fracture of petrous bone, quoted, Otol. 89
——, term "anæmia" of labyrinth due to, Otol. 123
Polk, practice as regards removal of appendix in pelvic operations, Obst. 351
Pollock, G. D., case of hypertrophy of gums and alveolar process, quoted, Odont. 22
Pollosson, primary mortality from Wertheim's operation, given by, Obst. 95
Polycythæmia accompanying congenital malformation of heart, Child. 3, 4
—— —— —— cyanosis, Child. 4
—— (secondary), Clin. 96
—— ——, in congenital heart disease, Clin. 93
—— —— —— —— ——, association with erythroblastic reaction in bone-marrow, Clin. 93
—— —— —— —— ——, cause of, Clin. 90, 91, 92, 94, 95
—— ——, not noted in chronic methæmoglobinæmia and sulphæmoglobinæmia, Clin. 86, 87
—— (splenomegalic), albumin in blood and blood-serum in, Clin. 92
—— ——, excess of red blood-corpuscles in, Clin. 86, 91
Polygraph, string galvanometer confirms facts already made known by, Med. 97
Polyphyodontism, theory of, Odont. 46
Polypi, multiple, of rectum, Surg. 105, 106
Polypus (fibromyomatous) of uterus in girl, aged 17 (G. Blacker), Obst. 63
—— (naso-antral), Laryng. 98, 99
—— (nasopharyngeal), case of (W. H. Kelson), Laryng. 60
—— ——, removed from boy (H. J. Davis), Laryng. 32
—— (tuberculous, large), removed from middle ear of infant (H. J. Marriage), Otol. 41
—— (uterine), undergoing malignant degeneration (Professor Schottländer, shown by H. Macnaughton-Jones), Obst. 31
Polysinusitis, effect of, on vision, Laryng. 165, 166
Polyuria and chronic renal disease associated with infantilism, case of (H. M. Fletcher), Child. 95
Poncet, danger of morphine injection before chloroform inhalation, stated by, Anæsth. 43
Poorer classes, homes of, chief centre of tuberculous infection, Epid. 35
Popper, experimental work on transmissibility of poliomyelitis, Epid. 199
Population (general), incidence of gall-stones among, greater than among hospital patients, Path. 90
Pork pies, eating of, followed by food-poisoning at Derby, Epid. 65
—— —— —— —— —— at St. Anne's-on-the-Sea, Epid. 64
Porro's operation, see *Cæsarean section* (Porro's)
Porter, T. C., elongation of crystals, quoted, Path. 140
Posner, experiments as to differentiation of typhoid and paratyphoid bacillus group, Path. 252
Post-allantoic gut, Surg. 93, 94, 95, 97
Post-hypnotic influence, Anæsth. 17
—— ——, unreliability of, Anæsth. 17
Post-mortem examinations, percentage incidence of gall-stones in, Path. 88
Post-nasal space, examination in otalgia with history of syphilis, Otol. 23
—— ——, tumour of (H. FitzGerald Powell and G. W. Badgerow), Laryng. 12

Post-pharyngeal tumour and enlarged glands obstructing œsophagus, Electr. 22
Posture as cause of adolescent albuminuria, Med. 124, 125, 138-141, 144
——, change from recumbent to sitting, effects in anæsthesia, Anæsth. 29
—— in relation to adolescent albuminuria, Med. 121, 124, 125
Potassia sulphurata not suited for internal use, Therap. 18
Potassium, bichromate of, electrical introduction of, in treatment of gastric ulcer, Electr. 42
——, chlorate of, in ulcerative stomatitis in children, Child. 72
——, permanganate of, as mouth-wash in ulcerative stomatitis in children, Child. 73
—— ——, in hot water, as mouth-wash during rectal feeding, Therap. 72
—— ——, use in gastric lavage, Electr. 37
——, iodide of action, Electr. 45
—— ——, effect on high blood-pressure, Med. 77, 78
—— ——, in treatment of congenital syphilitic deafness, Otol. 8, 22
Potpeschnigg, epidemic of poliomyelitis in Steiermark (1909), reported by, Epid. 208
Pott's disease, case of, successfully treated by resection of posterior spinal nerve-roots, Surg. 242
—— fracture, cause of bad results, Surg. 33
—— —— complicated by split fracture of lower end of tibia, Surg. 18
—— ——, danger of erroneous deflection of body-weight in walking, Surg. 20
—— ——, importance of recognizing backward displacement of foot, Surg. 20
—— —— —— of sufficient reduction, Surg. 20, 21
—— ——, operation for malunited cases, Surg. 22
—— ——, reasons for operative treatment, Surg. 34
—— ——, Thomas's device for reducing lateral deviation of foot, Surg. 21
Potters, prevalence of phthisis among, how explained, Child, 218
Poupart's ligament, Surg. 166
Pourville, sequestered French marine health resort, Baln. 18
Powell, A.—The Bentley-Taylor method of mounting mosquitoes, with demonstration of specimens, Path. 41
Powell, H. FitzGerald.—Discussion on bacteriology of chronic post-nasal catarrh, Otol. 52
——, discussion on case of acute inflammation of mastoid, treated by Bier's method, Otol. 59
—— —— —— of epithelioma on right palato-glossal fold, Laryng. 60
—— —— —— of leprosy, Laryng. 27
—— —— —— of lingual thyroid, Laryng. 82
—— —— —— of nasopharyngeal fibrous polypus, Laryng. 61
—— —— —— of papilloma of pharynx, Laryng. 62
—— —— —— of swelling at junction of right vocal cord and apex of vocal process, Laryng. 80
—— —— on cases illustrating results of removing nodules from cords by means of galvano-cautery (by indirect method), Laryng. 41
—— —— on eight cases of mastoid operation (Case II), Otol. 127
—— —— on functional aphonia in a boy, aged 15, Laryng. 115
—— —— on injuries of ear, Otol. 104
—— —— on stricture of lower end of œsophagus, Laryng. 38
—— —— on tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 111
——, large growth in the larynx, Laryng. 84
——, and Badgerow, G. W.—Tumour of post-nasal space, Laryng. 12
—— ——, ulceration of soft palate, Laryng. 11
Power, Henry, "On the relation between dental lesions and diseases of the eye," quoted, Odont. 27-36
Poynton F. J.—Case of imperfect osteogenesis, Clin. 105
——, discussion on case of actinomycosis of abdominal wall, Clin. 81
—— —— of pulmonary stenosis (congenital?), Clin. 83
—— —— on cases of familial pigmentary dermo-fibromatosis, Clin. 76
—— —— on rigidity of the spine, Clin. 149
—— —— on Stokes-Adams symptoms in a lad, Clin. 42

Poynton, F. J.—Discussion on treatment of acute suppression of urine by incision and drainage of kidneys, Clin. 168
——, pathology of amaurotic family idiocy, quoted, Path. 171
Pozzi, practice as regards removal of appendix in pelvic operations, Obst. 353
Præcordial impulse, position in acute pericarditis, Med. 49
Pre-cancerous growths, removal by non-operative measures, Electr. 51
Pregnancy, abdominal (primary), in rabbit (W. Blair Bell), Obst. 228
—— and menstruation, uterine mucosa in, action of chorionic cells and functions of decidua, (James Young), Obst. 291
——, bacteriology of vagina during, Obst. 169
—— (normal), mucosa changes in, cause of (James Young), Obst. 301
—— complicated by fibroid tumour of uterus, after-history of case (A. H. N. Lewers), Obst. 307
—— (tubal) absent in case of hæmatosalpinx with torsion of tubes (Miss A. L. McIlroy), Obst. 112
—— ——, bimanual examination for, by rectal passage, Obst. 328
—— ——, communications on (1909-10) (summarized), Obst. 16
—— ——, ruptured, communication on (summarized), Obst. 11
—— ——, twin (S. J. Cameron), Obst. 149
Premolar, eruption internally or externally to arch with deciduous molars in place, Odont. 112
——, unerupted, causing absorption of permanent molar (T. Stordy), Odont. 37
Premolars, development in anthropoid apes, Odont. 112
Prentice, H. R.—Case of syringo-bulbia, Neur. 25
von Preuschen, glands of vagina, quoted, Obst. 22
Preysing, case of gunshot injury to ear, quoted, Otol. 99
Price, F. W.—An investigation of the action of aconite on the pulse-rate, Therap. 151-191
——, discussion on drugs used as local anæsthetics in mouth, Therap. 60
—— —— on low blood-pressure, Med. 78
Prideaux, W. de C.—Skull from a barrow showing somewhat symmetrical mandibular exostoses, Odont. 39
——, three cases of torus palatinus, Odont. 39
Priessnitz, Vincent, establishment of water-cure by, Baln. 84
Priestley, Joseph, discovery of oxygen in air by, Baln. 3
Prigge, period of excretion of typhoid bacillus by carriers, stated by, Epid. 4, 5
——, typhoid carriers among those not attacked by typhoid fever, quoted, Epid. 5
—— and Sachs-Müke, paratyphoid carriers, quoted, Epid. 56
Pring, C. H., and Evans, Arthur.—Case of Graves's disease: its treatment, first by medical methods, and later by excision of one of the lobes and the isthmus of the thyroid gland, Clin. 122
Pringle, J. J.—Discussion on cases for diagnosis, Derm. 25, 35, 42, 53, 70, 102
——, discussion on case of acute lupus erythematosus, Derm. 51
—— —— —— of angiokeratoma, Derm. 45
—— —— —— of elephantiasis of lip, Derm. 57
—— —— —— of generalized eruption, probably mycosis fungoides, Derm. 21
—— —— —— of granuloma annulare, Derm. 25
—— —— —— of granuloma inguinale, Derm. 136
—— —— —— of hyperkeratosis of palms and soles, Derm. 18
—— —— —— of ichthyosis in child treated by thyroid extract, Derm. 72
—— —— —— of imperfect hair-growth in scalp of child, aged 18 months, Derm. 54
—— —— —— of keratosis of finger, Derm. 32
—— —— —— of lupus erythematosus associated with ulceration, Derm. 54
—— —— —— of macular atrophy associated with lupus erythematous, Derm. 143
—— —— —— —— —— in syphilitic patient, Derm. 125
—— —— —— of melanodermia, associated with pernicious anæmia, Derm. 28
—— —— —— of multiple idiopathic pigmentary sarcoma (so-called), Derm. 10

Pringle, J. J.—Discussion on case of (?) mycosis fungoides, Derm. 78
——, discussion on case of parakeratosis variegata (?), Derm. 31
—— —— —— of pemphigus foliaceus, Derm. 17
—— —— —— of rare form of syphilitic onychia, Derm. 56
—— —— —— of summer eruption, Derm. 4
—— —— —— of sycosiform syphilide, Derm. 106
—— —— —— of symmetrical angiomatous swellings in a tuberculous subject, Derm. 30
—— —— —— of syphilitic auto-reinfection, Derm. 131
—— —— on convenient method of giving "606" intravenously, Derm. 38
—— —— on nodular lupus erythematosus, Derm. 109
—— —— on small-spored ringworm of scalp, Derm. 67
—— —— on two cases of nævus unius lateris, Derm 66
Pringle type of adenoma sebaceum, case of (J. H. Sequeira), Derm. 58
Pritchard, Eric.—Case of achondroplasia (shown by W. Hampson), Child. 1
——, discussion on two cases of infantilism, Child. 60
——, idioglossia, Child. 52
——, intermittent word-blindness (congenital), Child. 51
Pritchard, Urban.—Discussion on case of acute vertigo with repeated falls, always to left, Otol. 73
——, discussion on case of caries and necrosis following mastoiditis due to scarlet fever, Otol. 70
—— —— on syphilis in relation to otology, Otol. 19
Probyn-Williams, R. J.—Discussion on anæsthesia in post-pharyngeal abscess, Anæsth. 37
—— —— on nasal reflex during anæsthesia, Anæsth. 29
Proctodeum, Surg. 94, 95, 97
Proctoscope, uses of, Obst. 331
Prognathism, see *Gums* and alveolar processes of jaw, excessive hypertrophy
Proptosis (double) due to growth (lymphoma, hæmangio-lymphadenoma) invading each orbit (L. V. Cargill), Clin. 53
——, visceral, how induced, Med. 112
Protargol, weak solutions of, in intranasal treatment of chronic frontal sinus suppuration, Laryng. 129
Protein disintegration, amino-acids ultimate products of, Therap. 70
Proteins, absorption in rectal feeding, Therap. 64
—— —— —— —— ——, fallacy regarding, Therap. 64
—— —— —— —— ——, incomplete, Therap. 65
—— —— —— —— ——, no relation to amount injected, Therap. 65
Prothesis in cleft palate, Surg. 179
Proud, P. J.—Model of a maxilla with exostoses, Odont. 41
Pruritus, treatment by calcium salts, Baln. 121
Pseudo-diphtheria bacillus in cervix, Obst. 193
Pseudoglioma as clinical and pathological entity, Path. 247
——, relation of *Meningococcus intracellularis* to (G. Coats and J. G. Forbes), Path. 242
——, retina usual primary seat of metastasis in, Path. 247
——, symptoms of, Path. 248
Pseudo-hermaphroditism, case of (P. P. Cole), Obst. 53-61
Pseudo-typhoid, organism from case of, fermentation results of cultures on sugars, Path. 43
Psoriasis (follicular) and pityriasis rubra pilaris, diagnosis between, Derm. 98, 100
—— imitated by lichen scrofulosorum (F. P. Weber), Derm. 93
Psychasthenia (or neurasthenia), distinction from hysteria, Neur. 9
Psycho-analysis, treatment of hysteria by, method described, Neur. 14, 15
Ptomaine poisoning producing pemphigus and other bullous dermatoses, Med. 32
Pubiotomy, Obst. 162, 190, 208
——, advantages of, Obst. 183
——, risks of, Obst. 162
Puerperal infection, micro-organisms present in, Obst. 166
—— —— —— ——, anaerobic, Obst. 167

Puerperal infection, micro-organisms present in, gonococcal, Obst. 169
—— —— —— ——, pathogenic, Obst. 166, 167, 170
—— —— —— —— —— and saprophytic, dividing line between, not always clear, Obst. 170
—— —— —— ——, putrefactive, Obst. 166, 167
—— —— —— ——, saprophytic, Obst. 167, 170
——, sepsis, severe, lesser prevalence in London, Obst. 189
—— ——, *Staphylococcus albus* associated with, Obst. 194
—— ——, suspected, definition needed, Obst. 187, 189
—— ——, treatment by vaccines, Obst. 194, 200
—— septicæmia, diagnosis from sapræmia, Obst. 194
—— —— in cows, effect on milk, Epid. 75, 102
Puerperium, complications of, communication on (summarized), Obst. 11
Pulsation, absence of, under screen examination, indicating pericardial effusion, Med. 44, 48
——, presence of, under screen examination, indicating dilatation of heart, Med. 44, 48
Pulse, irregular, due to intermittences, Med. 87, 88
Pulse-rate, action of aconite on, Therap. 151-191
——, effect of atropine on, in cases of heart disease under influence of digitalis (M. D. Silberberg), Therap. 192-210
——, records of low blood-pressure compared with, Med. 53
Punjab, seasonal prevalence of bubonic plague in, Epid. 225
——, see also *Kangra* district
Purin, pure, see *Hypoxanthin*
Purkinje cells, changes in amaurotic idiocy (Tay-Sachs disease), Path. 201-203
—— ——, degenerated, from case of amaurotic idiocy (Tay-Sachs disease) (photomicrograph), Path. 182, 183; (plate) Path. 191
—— fibres, Therap. 97
Purpura, case of (L. F. Knuthsen, shown by G. W. Dawson), Derm. 60
—— (Henoch's), two cases (W. Essex Wynter), Clin. 48
——, term for hæmorrhagic diseases, Surg. 49
——, treatment by calcium salts, Baln. 121
PURSLOW, C. E.—Retroflexion of gravid uterus with superimposed fibroid, Obst. 288
Putnam, Professor J. J., labours on behalf of hydrotherapy, Baln. 90
Pyæmia (chronic) of five years' duration following suppurative epiphysitis of upper end of humerus (D. Drew), Child. 162
Pyknosis of cell-nuclei in case of atypical chloroma, Path. 64
Pylorus, hypertrophy of, and spasm (E. Cautley), Child. 185
Pyorrhœa alveolaris and consecutive arthritis, relationship between, Clin. 141
—— —— causing ulceration of pharynx (L. H. Pegler), Laryng. 100
—— —— complicating membranous colitis, Clin. 140
—— ——, preparation of vaccine for, Odont. 81
—— ——, skin diseases caused by, Odont. 80
—— ——, treatment by diathermic current indicated in, Electr. 7
Pyosalpinx, bimanual examination for, by rectal passage, Obst. 328
Pyramidal cells from case of amaurotic idiocy (Tay-Sachs disease) (plate), 190
Pyrexia, toxic condition from pathogenic invasion, Baln. 93

Quadriceps extensor femoris, ossification of, frequency, Surg. 138
Quadrilateral cartilage (nasal septum), two specimens of, showing fenestræ; removed by submucous section (Dan McKenzie), Laryng. 5
Qualimeter (Bauer's) for determining hardness of X-ray tube (W. Deane Butcher), Electr. 71
Queen Charlotte's Hospital, cases of obstructed labour at, how dealt with, Obst. 208
—— —— ——, saving of fœtal life at, how effected, Obst. 216
Quinine in acute lupus erythematosus, Derm. 51
—— in lupus erythematosus, Derm. 92
Quiserne, Dr., on waters of Bagnoles d'Orne, Baln. 72

Rabbit, antiserum derived from, after intravenous injection of paratyphoid A, Path. 262
—— injected with *Bacillus paratyphosus* B, serum from, titrated with the homologous extract, Path. 263, 265
—— —— with paratyphoid A serum results from, of use, Path. 263, 264
——, primary abdominal pregnancy in (W. Blair Bell), Obst. 228
Rabbit-bone, portion of, removed from right bronchus (H. Tilley), Laryng. 96
Rabbits, crossing of breeds, results as affecting Mendelian theory, Med. 21, 22
——, uterine tumours in (A. E. Boycott), Path. 225, 232
Rabies, pathological lesion in poliomyelitis, similar to that of, Epid. 200
——, virus of, similarity of that of poliomyelitis to, Epid. 199, 200
Rabinowitch, infection experiments with tubercle bacilli by the alimentary tract, Child. 221
Radial form, distribution of, Path. 142
Radiator tubes (Cossor), Electr. 63
—— —— (Gundelach), Electr. 63
Radio-activity of medicinal waters, Baln. 108
Radiography, bony connexion between vertical column and inner border of scapula in Sprengel's deformity detected by, Electr. 69
Radium, demonstrations of application of, to growth in œsophagus, Laryng. 151, 153
—— emanations, dissolved in water, treatment by, of inoperable cancer, Electr. 66
—— in treatment of cancer (J. Hall-Edwards), Electr. 47-69
—— —— ——, dosage, Electr. 62
—— —— ——, selection of cases, Electr. 62
—— —— —— of œsophagus, cases reported, Electr. 64-66
—— —— —— —— ? temporary cure (W. Hill), Laryng. 43
—— —— —— of pharynx, cases reported, Electr. 64-66
—— —— of endothelioma of palate, with metastatic growth in neck (J. Gay French), Laryng. 94
—— —— of epithelioma, Electr. 59
—— —— of fungating carcinoma of œsophagus (W. Hill), Laryng. 8
—— —— of malignant disease, Electr. 52
—— —— —— —— of pharynx (R. H. Scanes Spicer), Laryng. 64
—— —— of rodent ulcer, effect of, Electr. 61
——, "selective action" of, criticized, Electr. 52, 68
—— —— —— discussed, Electr. 61, 62
——, β-rays of, Electr. 62
—— ——, secondary rays derived from silver possess some penetrative power as, Electr. 67
——, γ-rays of, Electr. 61, 62, 65
Radius, absent on both sides, in case of double congenital club-hand of the radio-palmar variety (A. H. Tubby), Child. 164
——, dislocation forwards and inwards, complicating fractures about elbow-joint, treatment by extension, supination and acute flexion, Surg. 8
——, fracture of head of, Surg. 9
Railway accident, terror caused by, resulting in hypnoid state, Neur. 13
Rainfall of Margate and Borkum compared, Baln. 19
Ramsay, Sir W., F.R.S., discovery of argon as constituent of atmosphere, by, Baln. 3
RANSOME, ARTHUR, F.R.S.—The scientific uses of the notification of disease, Epid. 185-197
Raschkow, plexus of, Odont. 52, 53, 55
Ratke's pouch, Laryng. 53
Rat virus organisms, Path. 254
—— viruses containing *Bacillus enteritidis*, danger of, Epid. 61, 69
Rats, experimental infection with Gaertner bacillus, result fatal, Epid. 68
—— (hairless), specimens of, Path. 293
—— ——, thyroid gland of, Path. 301
——, sporotrichosis infection from, Derm. 115
Ravenel, feeding experiments with tubercle bacillus, Child. 217

Raw, N.—Discussion on absence of abdominal respiratory movement in pericarditis, Med. 47
—— —— on low blood-pressure, Med. 76
—— —— on use of belladonna in dyspepsia, Med. 31
Rayleigh, Lord, F.R.S., discovery of argon as constituent of atmosphere, by, Baln. 3
Raynaud's disease, Derm. 101
Reaction, laws of, in hydrotherapy, Baln. 92, 110
——, powers of, possessed by living tissues, Baln. 92, 110
—— —— —— —— ——, disordered in chronic disease, Baln. 110
Recamier, combination of suggestion with chloroform anæsthesia, quoted, Anæsth. 22
v. Recklinghausen's disease, cardinal signs of, Clin. 75, 76
—— ——, case of (C. Rowntree), Clin. 36
—— ——, relation of familial pigmentary dermo-fibromatoses to, Clin. 74, 75
Rectal aliment, predigestion by liquor pancreaticus, Therap. 63
—— crises, early, associated with tabes dorsalis, case of (J. P. Lockhart Mummery and James Collier), Clin. 118
—— feeding, absorption of carbohydrates in, Therap. 65
—— —— —— ——, fallacy regarding, Therap. 64
—— —— —— of fats in, Therap. 66
—— —— —— ——, absent in case of chyluria, Therap. 66, 67
—— —— —— ——, in case of fistula of thoracic duct, Therap. 66
—— —— —— of proteins in, Med. 64, Therap. 65
—— —— —— ——, no relation to amount injected, Therap. 65
—— ——, amino-acids in, Therap. 73
—— ——, complicated by vomiting, Therap. 72
—— ——, death from air-hunger during, Therap. 74
—— —— —— —— ——, presumed cause of, Therap. 74, 75
—— ——, dextrose in, Therap. 73
—— ——, disadvantages of, Therap. 72
—— —— injurious in gastric ulcer, Therap. 72, 73
—— ——, mouth-washes during, Therap. 72
—— ——, nitrogenous metabolism during, Therap. 67
—— ——, value of, critical inquiry into (W. Langdon Brown), Therap. 63-76
Rectifier, high-tension, Electr. 82
Rectum, cancer, diagnosis by means of sigmoidoscope, Surg. 126, 128
—— —— —— facilitated by early examination, Surg. 121, 122, 126
—— ——, examination to determine whether growth fixed or mobile, Surg. 123
—— ——, in patient from whom malignant papillomatous cysts of both ovaries had been removed six years previously (M. A. Scharlieb), Obst. 358
—— ——, inoperable, statistics, Surg. 108, 125
—— —— —— treatment, palliative by colostomy, Surg. 125
—— ——, involvement of lymphatics in, Surg. 107, 121
—— ——, pathology, Surg. 105, 121
—— ——, recurrence, site of, Surg. 107
—— ——, spread into surrounding tissues as determined by muci-carmine staining process, Surg. 102, 121
—— ——, treatment, operative, Surg. 99-132
—— —— —— ——, administration of anæsthetic during, Surg. 117
—— —— —— ——, aseptic healing after, Surg. 127
—— —— —— ——, attempt to preserve natural anus after, Surg. 103, 108, 119, 127, 130
—— —— —— ——, avoidance of shock during, Surg. 130, 131
—— —— —— ——, control of hæmorrhage during, Surg. 111, 117
—— —— —— ——, feasibility to be determined by examination under anæsthesia, Surg. 106, 107
—— —— —— ——, limitations to, Surg. 101, 102, 106
—— —— —— ——, methods abdomino-anal, Surg. 123, 124, 129

Rectum, cancer, treatment, operative, methods, abdomino-perineal, Surg. 100, 113, 114, 117. 127-129, 131
—— —— —— —— —— ——, mortality, Surg. 111, 114
—— —— —— —— —— ——, radical, Surg. 100, 101, 104, 111, 132
—— —— —— —— —— ——, small amount of shock after, Surg. 131
—— —— —— —— —— ——, with permanent colostomy, Surg. 111, 118, 121, 131
—— —— —— —— ——, by extended operation, Surg. 108, 120, 124
—— —— —— —— ——, choice of, Surg. 107, 108
—— —— —— —— ——, excision, partial, Surg. 103
—— —— —— —— ——, joining ends of gut by secondary operation, Surg. 108
—— —— —— —— ——, sacro-anal, Surg. 124
—— —— —— —— ——, sacro-coccygeal, Surg. 103
—— —— —— —— ——, trans-sacral (Kraske's), Surg. 99, 108, 110, 112, 113, 116
—— —— —— —— —— ——, modified, Surg. 104
—— —— —— ——, preceded by preliminary colotomy, Surg. 103
—— —— —— ——, prophylactic injections of vaccine or serum before, Surg. 130, 131
—— —— —— ——, recurrence after, Surg. 103, 131
—— —— —— ——, severe case, successful, no recurrence, Surg. 109, 110
—— —— —— ——, statistics, Surg. 103, 109, 110, 114
—— —— —— —— —— of permanent cure, Surg. 110
——, delimitation of (F. W. Jones), Surg. 85-98
—— ——, difficulties in, Surg. 98
—— ——, uncertainty of various teachers' views regarding, Surg. 88
——, examination of, aided by Allingham's dilator (modification), Obst. 330, 331, 336
—— —— ——, by proctoscope, Obst. 331
—— ——, under bimanual method, aiding diagnosis of hæmorrhoids from other diseased conditions, Obst. 331, 322
—— —— —— —— —— ——, of sarcomatous ovary, Obst. 330
—— —— —— —— —— in diagnosis of pelvic abscess, Obst. 329, 330
—— —— —— —— —— —— of ureteral calculus, Obst. 329
——, excision, total, for cancer, advantages, Surg. 132
——, functions of, interfered with by diseases of female genital organs, Obst. 327
——, imperforate, Surg. 90, 94
——, importance of, in gynæcology (E. Stanmore Bishop), Obst. 327-336
——, involvement, in cancer of cervix, Obst. 78
——, loose subcutaneous layer of coat of, influence on development of hæmorrhoids, Obst. 332, 333
—— of fœtus, Surg. 89-98
—— (pelvic), Surg. 92
—— ——, how marked off from pelvic colon, Surg. 92, 93
—— (perineal), Surg. 93
—— ——, development, Surg. 93
——, polypi of, multiple, Surg. 105, 106
—— —— ——, relationship with cancer, Surg. 105, 106
——, prolapse of, treatment, Obst. 336
——, stricture of, due to parturition, Obst. 336
——, "syphilitic stricture of" (so-called), presumed cause of, Obst. 73
——, use of, for bimanual examination, Obst. 328
—— ——, under bimanual method in diagnosis of pyosalpinx or tubal pregnancy, Obst. 328
Rectus abdominus muscle, tumour of, in child (J. P. Lockhart Mummery), Child. 7
—— muscle, external, principally alone involved in oculo-motor type of polio-encephalitis, Child. 90, 93
Red indigo method (Bouma's), colorimetric adaptation in estimation of indican, Baln. 47
Rees, W. T.—Discussion on balneological treatment of neurasthenia. Baln. 44
Reflex, nasal, during anæsthesia (J. Blumfeld), Anæsth. 27

Registrar-General's mortality returns, causes of death frequently undiscoverable from, Epid. 188

Registration of disease, system of, established by Manchester and Salford Sanitary Association, Epid. 186, 187

—— ——, work done by, with regard to, by British Medical Association and Lancashire and Cheshire Branch, Epid. 187

Regnier, danger of morphine injection before chloroform inhalation, quoted, Anæsth. 43

Reibmayr and Ballner, differentiation of *Bacillus typhosus* and *Bacillus paratyphosus*, quoted, Path. 252

Reid, Professor, on genito-mesenteric fold, Obst. 346

——, on appendiculo-ovarian ligament, Obst. 344

Reid, Archibald D.—Demonstration of lantern slides of three cases, Electr. 73

——, discussion on a sesamoid bone fractured by direct violence, Electr. 97

——, the technique of skiagraphy, Electr. 84-92

Reinecke, sex-incidence of multiple cartilaginous exostoses, Med. 3

Remittent fevers, treatment of (1811), Med. 148

Respiration (artificial), Shutly's method of, as cause of Erb's paralysis in newly born infants, Child. 9

——, "Cheyne-Stokes," during anæsthesia, case of (Cecil Hughes), Anæsth. 37

Respiratory failure in asphyxia under chloroform, Path. 217-222

—— movement (abdominal), absence of, accounting for cyanosis, dyspnœa and upright position of patient in pericarditis, Med. 45

—— —— ——, absence of, indicating presence of pericarditis, Med. 40

—— —— —— —— —— —— ——, illustrative cases, Med. 40-44, 46, 47

—— —— —— —— —— —— ——, more marked in fibrinous than in effusion form, Med. 45

—— —— ——, present in pericarditis, Med. 47

—— tract, untoward post-operative symptoms and lowered vitality, causal relationship between, Anæsth. 6

"Rest" theory of neoplasms, Med. 3

Retina, central artery of, embolism of, in a girl (S. Stephenson), Child. 56

——, examination and appearances in case of amaurotic idiocy (Tay-Sachs disease) (photomicrographs), Path. 188, 189

——, usual primary seat of metastasis in pseudoglioma, Path. 247

Retinitis, metastatic, Path. 243, 245, 246

Retrobulbar neuritis, see *Neuritis*, retrobulbar

Retrocerebellar abscess, case of; autopsy (S. Scott), Otol. 34

—— ——, operation for (S. Scott), Otol. 35

Reubsaël, multiple cartilaginous exostoses and myositis ossificans progressiva, quoted, Med. 3

Revolving seat for examination under X-rays, Electr. 13, 26

Revue d'Hydrologie, commencement of (1880), Baln. 79

Reynolds, Sir J. Russell, view regarding hysteria quoted, Neur. 2

Rheumatism, no immunity to, from drinking soft water, Baln. 124

——, seaside climate seldom suitable for, Baln. 17

——, treatment by subthermal baths, Baln. 97

—— —— by sulphur, Therap. 15, 16

—— (acute), pericarditis following, see *Pericarditis* (rheumatic)

—— (articular, chronic), name applied to chronic villous arthritis, Baln. 34

—— (chronic), Baln. 26

—— —— and thyroid gland, Baln. 27

—— ——, liability of women to, at menopause, Baln. 28, 34

—— (muscular), fibrositis essential feature of, Baln. 29, 34

—— —— prevalent in damp districts and climates, Baln. 28

——, see also *Heart disease*, rheumatic

Rhythmical elevations in soft palate accompanied by rhythmical clickings in Eustachian tube and tympani of right ear in syphilitic, Otol. 13
Ribbert, inhalation theory in phthisis, Child. 228
Ribs, malformations of, in Sprengel's deformity, Electr. 70
Rice as food for children, early mention, Child. 126
Richards, H. Meredith, case of typhoid carrier, quoted, Epid. 18, 108
Richardson, Sir B. Ward, hard water in relation to calculus, quoted, Baln. 124
Rickets, achondroplasia and, Child. 108
——, first record of, as appearing in West of England in seventeenth century, Child. 119
——, late, in connexion with adolescent albuminuria, Med. 132
—— multiple cartilaginous exostoses and, Med. 3
——, relation of von Jaksch's anæmia to, Child. 157
——, seaside climate scarcely a specific indication for, Baln. 17
——, treatment by calcium salts, Baln. 121
Rider's bone, traumatic myositis ossificans distinct from, Surg. 141
Riedel, percentage of primary carcinoma of gall-bladder and biliary passages in cholelithiasis, recorded by, Path. 94
Riedel's operation in chronic frontal sinus suppuration, Laryng. 134, 143
Rieder and Delépine, "Atlas of urinary sediments," quoted, Path. 132, 133, 134, 135
Riegel, action of atropine on gastric secretion, quoted, Med. 23
Rimbaud and Vallet, intermediate forms between *Bacillus coli* and *Bacillus typhosus*, quoted, Epid. 118
Ring pessaries, black, chemical composition, Obst. 324-326
—— ——, celluloid, light transparent, advantages of, Obst. 326
—— ——, red, chemical composition, Obst. 324-326
—— ——, pink rubber, chemical effect of, probably producing gangrene of vagina (A. E. Johnson), Obst. 323
Ringer, S., F.R.S., on action of aconite, quoted, Therap. 157
——, "The relative amount of sugar and urea in the urine in diabetes mellitus," quoted, Med. 152, 153
Ringworm ("eczematoid") of fungus associated with tineæ barbæ, cultures of violet endothrix trichophyton from case of (H. G. Adamson), Derm. 126
—— of toe-nails, case of (A. Shillitoe), Derm. 151
——, small-spored, of scalp (J. M. H. MacLeod), Derm. 67
——, treatment by X-rays followed by acute pityriasis of scalp. Derm. 141
Ringworm cultures, illustrating eczematoid ringworm of hands and feet, series of (A. Whitfield), Derm. 111
von Ritter, cases of progressive spinal muscular atrophy of infants and young children, Neur. 69
Riverside Baths (New York), treatment at, Baln. 90
Riviera, Italian, suitable for climatic treatment of neurasthenia, Baln. 42
RIVIERE, C.—Congenital aortic stenosis, Child. 85
——, the rôle of auto-inoculation in medicine, a plea for its rational extension, Therap. 39-50
"Roaring" and "whistling" in horses, ætiology of, Laryng. 89, 90, 91
—— —— ——, unilateral abductor paralysis in man corresponds to, Laryng. 90
—— —— ——, results of ventricle-stripping operation for (F. Hobday), Laryng. 87
Robb, epidemic of poliomyelitis at Irthlingborough (1910), reported by. Epid. 214, 215
Robb, A., outbreak of milk-borne sore throat at Paisley, reported by, Epid. 95
ROBBINS, C.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 47
ROBERTS, C. HUBERT.—Discussion on chronic endometritis, Obst. 50
——, discussion on indications for, and technique of, Cæsarean section and its alternatives. Obst. 207
—— —— on an ovarian tumour obstructing delivery, Obst. 257
Robertson and Boyd, percentage of absorption of carbohydrates in rectal feeding, Therap. 65

Robertson, W., mixed outbreak of milk-borne diphtheria and septic sore throat at Leith, reported by, Epid. 96
Robin, classification of functional dyspepsias, Electr. 34
Robin, A., ignorance of medical hydrology among Paris medical graduates, Baln. 114
Robinson, Professor, on appendiculo-ovarian ligament, Obst. 344
Robinson, G. Drummond.—Cervical glands of the uterus in fœtus at term, Obst. 282
——, some observations on vaginal secretions in infants, Obst. 26
Robinson, H. Betham.—Discussion on case of epithelioma of epiglottis, Laryng. 84
——, discussion on case of large growth in larynx, Laryng. 85
—— —— —— of lingual thyroid, Laryng. 82
—— —— —— of ulceration on left tonsil, Laryng. 75
—— —— on empyema of maxillary antrum in boy, Laryng. 31
—— —— on tumour of right side of neck involving pharynx, Laryng. 100
Robinson, M. K., outbreak of milk-borne sore throat at Craigmore, reported by, Epid. 89
Robson, A. W. Mayo, cancer of gall-bladder rare as primary affection, quoted, Path. 94
——, explanation of less incidence of gall-stones among hospital patients, quoted, Path. 90
Rodent, hairless (*Heterocephalus glaber*, *H. phillipsi*), Path. 298-304
Rodent ulcer, liability of warts and moles upon face to develop, Electr. 51
—— ——, treatment by radium, X-rays and carbon dioxide snow compared, Electr. 61
Roderick, H. B.—Demonstration of pathological specimens, Child. 202
Roentgen rays, see *X-rays*
Rolleston, H. D.—Discussion on case for diagnosis, family nervous disease, Child. 199
——, discussion on case for diagnosis: imperfect osteogenesis, Clin. 108
—— —— —— of actinomycosis of abdominal wall, Clin. 81
—— —— —— of congenital heart disease, Child. 192
—— —— —— ——, obliteration of bile-ducts, Child. 49
—— —— —— of rheumatoid arthritis, Child. 37
—— —— —— of splenic enlargement, Child. 29
—— —— on cases of familial pigmentary dermo-fibromatosis, Clin. 75
—— —— on portals of entry in phthisis, Child. 225
—— —— on treatment of splenic anæmia by splenectomy, Clin. 69
——, percentage incidence of gall-stones in post-mortem examinations, given by, Path. 88
——, presence of gall-stones in great majority of cases of primary malignant disease of gall-bladder, quoted, Path. 94
——, recurrent attacks of post-typhoidal osteitis, Clin. 156
——, symmetrical lipomatosis, Clin. 1
——, and Humphry, G. P.—Rigidity of the spine, Clin. 146
——, Molony, J. B.—Idiopathic hypertrophic dilatation of the colon, Child. 18
——, Taylor, C. H. S.—Mollities ossium of forty-five years' duration, Clin. 97
Rolleston, J. D.—Concurrent scarlet fever and chicken-pox, Child. 14
——, discussion on case of extensive necrosis of lower jaw, Child. 31
—— —— on Dr. Milne's paper, "Measles: its treatment and prevention," Child. 173
—— —— on oculo-motor type of polio-encephalitis, Child. 94
—— —— on preliminary communication on syphilis and mental deficiency, Child. 171
—— —— on two cases of infantilism, Child. 60
——, gangrene of leg following diphtheria, Child. 10
——, inherited syphilis and "blue sclerotics," case of, Child. 96
——, and Macnaughtan, N. S.—Familial pigmentary dermo-fibromatosis, Clin. 71
—— ——, report on tumour of the upper lip, Clin. 114
Romanes, G., internal heat of earth, quoted, Path. 143
Romans (ancient), use of baths among, Baln. 3
Romberg, cause of heart-failure in acute disease, quoted, Baln. 93
Rome, University of, lectures on medical hydrology in, Baln. 115
Römer, investigations on pulp-nerves, quoted, Odont. 52
Rommeler's series of carriers of typhoid fever, Epid. 107

Ronda, suitable health resort for neurasthenics, Baln. 42
ROOD, F. S.—Discussion on use of drugs before inhalation anæsthesia, Anæsth. 49
Roscoff, Baln. 18
ROSE, FRANK.—Case of tuberculous tonsillitis, Laryng. 67
——, discussion on case of nasopharyngeal fibrous polypus, Laryng. 61
ROSE, S. F.—Ulcerative stomatitis in children, Child. 70
Rosenau, investigations as to typhoid fever carriers, quoted, Epid. 12, 13
Rosenberg, supernumerary teeth, quoted, Odont. 47
Rosenstein and Munk, absorption of fats in rectal feeding, Therap. 66
Rosenthal, case of typhoid carrier, Epid. 123
——, search for typhoid carriers, Epid. 121, 122
Roseola, fading, simulating ichthyosis in case of secondary syphilis (H. G. Adamson), Derm. 33
Rostock, University of, course of lectures on medical hydrology at, Baln. 114
Roth, case of oral sepsis causing thrombosis of cavernous sinus, quoted, Odont. 117
ROTH, P. B.—Discussion on large congenital diverticulum of bladder, Surg. 165
Rotter, method of performing operation for rectal cancer, Surg. 111
——, mortality percentage after operations for rectal cancer, Surg. 111
Roumania, first case of amaurotic idiocy (Tay-Sachs disease) observed in, Path. 169
ROUTH, AMAND.—Discussion on carcinoma of body of uterus, Obst. 311
——, discussion on case of sarcoma of uterus, Obst. 112
—— —— on chronic endometritis, Obst. 49
—— —— on hysterectomy followed by double phlebitis, Obst. 224
—— —— on indications for, and technique of, Cæsarean section and its alternatives, Obst. 161; (reply), 215
—— —— on new operation for radical treatment of flat pelvis, Obst. 269
—— —— on rupture of gravid uterus, Obst. 153
—— —— on an ovarian tumour obstructing delivery, Obst. 257
—— —— on primary abdominal pregnancy in rabbit, Obst. 233
Rowlands, R. P., description of Lothiessen's inguinal method of radical cure of femoral hernia, quoted, Surg. 167
ROWNTREE, C.—Carcinomatous degeneration in a chronic dermatosis, Clin. 159
——, case of von Recklinghausen's disease, Clin. 36
Royal Society of Medicine appeal for donations and subscriptions to building fund, Med. 155
—— —— ——, Sections of, closer union among, advocated, Med. 155
Rugby, outbreak of milk-borne sore throat at, Epid. 89
RUSHTON, W. T.—A possible cause of facial asymmetry, Odont. 105-109
——, discussion on an erosion cavity on lingual aspect of a lower incisor, Odont. 70
RUSSELL, A. W.—Discussion on indications for and technique of Cæsarean section and its alternatives, Obst. 204
Russell, J. S. Risien, and Kingdon, E. C., infantile cerebral degeneration with symmetrical changes at the macula, quoted, Path. 169
Russia, hydrotherapy in, Baln. 89
Rutherford, cysts of vagina, quoted, Obst. 22

Saccharomyces albicans present in ulcerative stomatitis in children, Child. 73
Saccharose fermentation by *Bacillus typhosus* not possible, Path. 51, 52
—— —— —— by culture from pseudo-typhoid organism, Path. 43
Sachs, name of "amaurotic family idiocy" first employed by, Path. 167
Sachs-Müke and Prigge, paratyphoid carriers, quoted, Epid. 56
Sacquepée, groups of strains of micro-organisms separated from cases of food-poisoning, by, Path. 253
—— and Bellot, epidemic of paratyphoid fever due to carriers, reported by, Epid. 57
Sacro-uterine ligaments, involvement of, in cancer of uterus, exaggerated, Obst. 72, 73
Sacrum, congenital absence of, fœtus with (C. White), Obst. 279

Sacrum, partial congenital suppression of left wing of, in case of asymmetry of pelvis; scoliosis (A. H. Tubby), Child. 163
Sainsbury, H.—Discussion on therapeutic use of digitalis group, Therap. 35
St. Anne's-on-Sea, outbreak of food-poisoning at, after eating pork pies, Epid. 64, 65
St. Jean de Luz, suitable health resort for neurasthenics, Baln. 42
St. Marylebone, diseases of air-passages more common in Manchester than in, Epid. 192, 193
——, measles more prevalent in Manchester than in, Epid. 191, 192
St. Marylebone General Dispensary, examination of "contacts" with tuberculous patients at, Epid. 30, 31, 38
St. Pol-sur-Mer, Baln. 18
Salicin in acute lupus erythematosus, Derm. 51
Saline, physiological, Therap. 53
—— solutions, weak, taken in medicinal waters, beneficial, Baln. 118
—— waters of Llandrindod wells, action, Baln. 39, 40
Salines, rectal, absorption of, Therap. 67
—— ——, effect of addition of dextrose to, Therap. 69, 70
—— ——, nitrogenous metabolism following administration of, Therap. 68
Saliva, free access of, importance in preventing caries, Odont. 97
——, mixture with food in relation to fermentation, Odont. 91, 92
—— prevents action of weak acids on teeth, Odont. 95
——, secretion of, effect of administration of atropine on, Med. 31
——, sulphocyanate in, before and during taking of sulphur water, Baln. 57, 58
—— —— of, estimation with ferric chloride in Meisling's colorimeter, Baln. 48
Salmon (1716) on disposition in phthisis, quoted, Epid. 154
Salpingitis (tuberculous), mode of infection in, discussed, Child. 225, 226, 232
Salter, S. J. A., hypertrophy of gums and alveolar processes, quoted, Odont. 23
Salts, dissociation of, Baln. 107
Salvarsan in treatment of case of syphilitic disease of nose (G. N. Biggs), Otol. 121
—— ("606"), intravenous injection of, instruments for, demonstration of (W. A. Lieven), Laryng. 45
Samuel Hyde Memorial Lectures: "Outlines of medical hydrology" (R. Fortescue Fox), Baln. 63-118
—— —— —— ——, see also *Hyde, Samuel*
Sanatoria, delay of consumptives in entering, Epid. 31
—— in treatment of pulmonary tuberculosis, Epid. 29
—— —— —— ——, effective only when cases are admitted early, Epid. 30
Sandalwood oil, local action possessed by, Electr. 45
de Santi, P. R. W.—Discussion on case of party-wall cancer of larynx and œsophagus, Laryng. 101
—— discussion on complete stenosis of the larynx from epithelioma in a man, aged 23, Laryng. 121
—— —— on endothelioma of palate, with metastatic growth in neck, treated by radium, Laryng. 96
—— —— on tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 110
—— —— on tumour of right side of neck, involving pharynx, Laryng. 100
——, two cases of malignant disease of tonsil, pharynx, and tongue, Laryng. 113, 114
Sapræmia, diagnosis from septicæmia, Obst. 194
Saprophytes, pathogenic micro-organisms present in puerperal infection acting as, Obst. 167, 170
Sarcoid, benign, Derm. 25
Sarcoma (cicatrizing), case of (K. Digby, for C. J. Symonds), Derm. 126
—— cutis, report on case for diagnosis (F. Ashton Warner and Wilfrid Fox), Derm. 81
——, empyema of antrum developing into (H. J. Davis), Laryng. 30
—— of femur, case of (O. L. Addison), Child. 40
—— (melanotic) of vulva (Clifford White), Obst. 66
——, multiple idiopathic pigmentary (so-called), case of (J. H. Sequeira), Derm. 8

Sarcoma of ovary, Obst. 330
——, periosteal, swelling formed as result of separation of periosteum by muscles, diagnosed as, Surg. 143
—— of skin, idiopathic multiple hæmorrhagic, Derm. 42, 43
—— of small intestine, specimen, Child. 203
—— of uterus in patient, aged 25 (D. Drew), Obst. 111
—— of vulva (A. H. N. Lewers), Obst. 64
—— ——, microscopical examination, Obst. 66
Sarcomatosis cutis of the "Perrin" type (E. G. Graham Little), Derm. 22
—— —— —— ——, treatment suggested, Derm. 23
Sarcomatous growth of both tonsils, with multiple sarcomata in and beneath skin (W. Hill), Laryng. 57
Sauerbruch and Bruno, case of division of posterior spinal nerve-roots for gastric crises of tabes, reported by, Surg. 211
SAUNDBY, R.—Discussion on after-history of albuminuria in adolescence, Med. 120-123
SAUNDERS, P. W.—Unilateral paralysis of ninth, tenth, and eleventh cranial nerves (shown for Dr. James Taylor), Neur. 51
SAVAGE, W. G.—Association of *Bacillus enteritidis* with typhoid fever, quoted, Epid. 61
——, diseases of the cow (excluding tuberculosis) affecting the milk in their relationship to human disease, Epid. 73-104
——, multiplication of bacilli of Gaertner group, quoted, Epid. 65
——, negative search for *Bacillus suipestifer*, quoted, Epid. 58
——, outbreak of milk-borne sore throat at Colchester, quoted, Epid. 95, 96
——, search for *Bacillus paratyphosus* B, quoted, Epid. 55
—— and Morgan, D., discovery of organism identical with *Bacillus paratyphosus* A in intestinal contents of healthy pigs, quoted, Epid. 54
Savage tribes, mutilating operations on ear among, Otol. 103
SAVILL, Mrs.—Discussion on success or failure in electro-therapy, Electr. 113
Savill, T. D., view regarding hysteria, quoted, Neur. 2
SAYER, Miss E.—Discussion on success or failure in electro-therapy, Electr. 114
Scalds of middle and internal ear, Otol. 95, 96
Scalp, acute pityriasis of (T. Colcott Fox), Derm. 140
——, ringworm (small-spored) of (J. M. H. MacLeod), Derm. 67
——, see also *Pediculi* of scalp
Scandinavia, hydrotherapy in, Baln. 89
Scaphoid, fracture of, Surg. 3
—— ——, reduction, method of, Surg. 11
—— ——, treatment by hyper-extension of wrist, Surg. 3
Scapula, inner border of, and vertebral column, bony connexion between, in Sprengel's deformity, detected by radiography, Electr. 69
Scarlet fever, action of aconite on pulse-rate in cases of, Therap. 164, 184-189
—— —— (bovine), existence of, as constitutional disease, arguments for, Epid. 87
—— —— —— —— —— —— against, Epid. 84, 102
—— —— and chicken-pox, concurrent (J. D. Rolleston), Child. 14
—— —— causing mastoiditis followed by caries and necrosis of temporal, parietal, and occipital bones, in girl, aged 5; operation; recovery (H. J. Davis), Otol. 70
—— —— followed by acute nephritis, Epid. 194
—— —— infectivity, variation in, in different localities, and in same localities at different times, Child. 179
—— —— in man reproduced in modified form in cow, Epid. 102
—— ——, malignant sinusitis due to, Laryng. 32
—— ——, outbreak at Dr. Barnardo's village homes, cause of, Child. 182
—— —— —— of, milk-borne, Epid. 86, 87, 100, 101, 104
—— —— —— ——, associated with teat ulceration or mastitis in cows, Epid. 88
—— —— —— ——, concurrent with septic sore throat and diphtheria from same source, Epid. 101

Scarlet fever, treatment by inunction with eucalyptus oil, Child. 177
—— —— —— —— —— —— (R. Milne), Child. 142-148, 152, 153
—— ——, varying fatality of, Epid. 192
Scarring (extensive) and pustular eruption following Scinde ulcers (Sir M. Morris and S. E. Dore), Derm. 107
Scar-tissue, large formation of, trophic ulceration over, of (E. M. Corner), Clin. 160
Scepticism in therapeutics in middle of nineteenth century, Baln. 84
Schäfer, E. A., F.R.S., injection of morphine and atropine before chloroform inhalation, Anæsth. 44
Schaffer, pathology of amaurotic idiocy (Tay-Sachs disease), advocacy of, quoted, Path. 167, 170
SCHARLIEB, Mrs.—Case of adeno-carcinoma of the uterus and ovaries, Obst. 359
—— case of carcinoma of the rectum occurring in a patient from whom malignant papillomatous cysts of both ovaries had been removed six years previously, Obst. 358
——, discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 200
—— , fibroma of ovary with twisted pedicle, Obst. 35
Schauta, Professor, practice as regards removal of appendix in pelvic operations, Obst. 352
Schede, congenital hairless children described by, Path. 303
Scheele, discovery of oxygen and nitrogen as constituents of atmosphere, Baln. 3
Scheib, ultimate results of Wertheim's operation, Obst. 98
Scheller, search for typhoid carriers by, Epid. 121
SCHELLING, C.—Discussion on case of pneumococcal stomatitis, Odont. 12
Schenck, case of sporotrichosis, quoted, Derm. 114
Schiff, action of atropine in reducing acid-values of stomach contents, quoted, Med. 24
Schittenhelm and Brugsch, administration of hypoxanthin in gouty patients, Baln. 59
Schmidt, separation of scopolamine from *Scopolia japonica*, Anæsth. 44
Schmidt, C., priority of A. Marcet over, in regarding cerebro-spinal fluid as secretion, Med. 153
SCHMIDT, D.—A universal safety and protective tube-stand for all kinds of fluoroscopy and radioscopy, Electr. 71
Schmiegelow, death from tetanus after attempts to extract foreign body from ear, reported by, Otol. 82
Schneeberg cobalt mines, local prevalence of primary cancer of lung among workers in, Path. 78
School-children, instruction in hygiene, Epid. 41
——, medical inspection of, agency in prevention of tuberculosis, Epid. 40, 41
School infection, outbreaks of poliomyelitis due to, Epid. 204-206, 208, 209
SCHOTTLÄNDER, Professor, practice as regards removal of appendix in pelvic operations, Obst. 35
—— (shown by H. MACNAUGHTON-JONES).—Cases of uterine polypi undergoing malignant degeneration, Obst. 31
Schottmüller, gastro-enteritis due to *Bacillus paratyphosus* B, Epid. 56, 57
Schroeder, percentage incidence of gall-stones in post-mortem examinations at Strasburg, given by, Path. 88, 89
——, percentage of primary carcinoma of gall-bladder and biliary passages in cholelithiasis, given by, Path. 94
Schultz, Professor, occurrence of traumatic myositis ossificans in German Army, Surg. 133
——, practice as regards removal of appendix in pelvic operations, Obst. 352
Schumacher, bacteriological examination of excreta of typhoid carriers, quoted, Epid. 118
——, proportion of typhoid carriers, quoted, Epid. 21, 119
Schuster, amaurotic idiocy (Tay-Sachs disease), quoted, Path. 169, 170
Schwartze, rupture of tympanic membrane, quoted, Otol. 93
Sciatica, treatment by diathermic current indicated in, Electr. 7
Scinde ulcers followed by extensive scarring and pustular eruption (Sir Malcolm Morris and S. E. Dore), Derm. 107

Sclerema neonatorum, with myositis fibrosa (F. S. Langmead), Child. 5
—— —— —— —— resembling sclerodermia of adult type, Child. 6, 7
Sclerodermia, hemiatrophy from, case of (P. C. Knapp), Neur. 28
—— of adult type, sclerema neonatorum with myositis fibrosa resembling, Child. 6, 7
—— with obstinate callous ulcers on legs, greatly improving under mercurial inunction (A. W. Williams), Derm. 13
Scleroma of nasopharynx in Polish woman (StC. Thomson), Laryng. 7
Sclerosis (cerebral) and idiocy (E. Cautley), Child. 184
——, see also *Spinal cord* (sclerosis)
Scoliosis in case of asymmetry of the pelvis (Naegele) (A. H. Tubby), Child. 163
Scopolamine and morphine, see also *Scopolamorphine* (below)
——, dangers of, Anæsth. 44
——, hypodermic injection before inhalation anæsthesia, operations for which suitable, Anæsth. 52
——, separation from *Scopolia japonica*, Anæsth. 41
Scopolamorphine, difference between German and English preparations, Anæsth. 54
——, hypnotic action of, Anæsth. 44
——, hypodermic injection before inhalation anæsthesia, Anæsth. 46, 53
—— —— —— ——, after-care of patients, Anæsth. 48
—— —— —— ——, dangers of, Anæsth. 46
—— —— —— ——, disadvantages alleged, Anæsth. 53
—— —— —— ——, dosage, Anæsth. 47
—— —— —— ——, methods of, Anæsth. 46, 47
—— —— —— ——, reduction of shock under, Anæsth. 52, 53, 54
—— —— —— ——, value during performance of endoscopic methods, Anæsth. 52
—— and atropine, hypodermic injection before chloroform inhalation, Anæsth. 50
—— —— —— —— ——, in abdominal and pelvic operations, Anæsth. 50
—— —— —— —— before inhalation anæsthesia, Anæsth. 46, 47
—— —— —— —— ——, dosage, Anæsth. 49, 56
Scopolia japonica, separation of scopolamine from, Anæsth. 41
Scotland, first advocacy of cold-bath treatment in, Baln. 83
Scotoma, occurrence of, in sinusitis, Laryng. 165, 166
SCOTT, S. GILBERT.—Demonstrations of skiagrams, Electr. 75
——, discussion on Bauer's qualimeter, Electr. 72
—— —— on demonstration of new tube-stand, Electr. 32
—— —— on lantern slides of three cases, Electr. 75
—— —— on sesamoid bone fractured by direct violence, Electr. 97
SCOTT, SYDNEY R.—Case of retrocerebellar abscess; autopsy, Otol. 34, 36
——, discussion on bacteriology of chronic post-nasal catarrh, Otol. 51
—— —— on case of acute vertigo, with repeated falls always to left, Otol. 73
—— —— —— of chronic dermatitis of pinna, Otol. 39
—— —— —— —— otitis media suppurativa, Otol. 66
—— —— —— of persistence of disease in large sinus tympani, Otol. 55
—— —— on deafness due to occlusion of Eustachian tubes by scar-tissue, Otol. 110
—— —— on labyrinthine vertigo (Menière's symptoms, non-infective) treated by operation, Otol. 119
—— —— on specimen of temporal bone from case of thrombosis of cavernous sinus, Otol. 28
—— —— on syphilis in relation to otology, Otol. 23
—— —— on three specimens of chronic middle-ear suppuration, Otol. 113
—— —— on two cases of cerebellar abscess, Otol. 63
——, fistula of the ampulla of the superior semicircular canal of the left side; bilateral radical mastoid operation: instrumental exploration of the left vestibule; total vestibular ablation reactions (left side); cochlea unaffected, Otol. 73
——, lateral sinus thrombosis (streptococcal), with early pulmonary metastasis; recovery, Otol. 30

Scoutetten, Professor H., report upon hydrotherapeutic institutions of Germany, Baln. 87

Screen examination, pulsation under, absence indicating pericardial effusion, Med. 44, 48

—— —— —— ——, presence indicating dilated heart, Med. 44, 48

——, fluorescent, lead-glass lined, for examination under X-rays, Electr. 13, 26

Scroff, M., action of aconite, quoted, Therap. 156

Scrofulous diathesis, marine climate indicated for, Baln. 17

"Scrunch" heard in stiff joints by auscultation, Med. 38

—— —— —— —— ——, diagnostic of osteo-arthritic changes, Med. 38

Scurvy (infantile), femur and tibia from case of, Child. 204

Sea air in treatment of neurasthenia, Baln. 41, 42

Sea-bathing, re-hydration of skin affected by, Baln. 16, 17

SEARLE, C.—Infantile spastic paraplegia (shown by Dr. G. S. Haynes), Child. 192

"Seaside biliousness," Baln. 126

Seaside treatment, lectures on, at Naples, Baln. 116

Sea-urchins' eggs, parthogenesis in, process compared with auto-katalytic ferment action of nucleus of neurones, Path. 175, 192, 193

Sea-voyages, question of suitability for neurasthenics, Baln. 41, 42

Sea-water, mixture of "primitive" water with water of infiltration, Baln. 108

Seborrhœa in relation to dental trouble, Odont. 74

Secondary invaders in typhoid fever, Epid. 117, 137

Seegen, Dr. J., first recorded lecturer upon balneology and climatology in Europe, Baln. 115

SEGURA, Dr.—Discussion on case of leprosy, Laryng. 26

Seifert, excess of red blood-corpuscles in cases of splenomegalic polycythæmia, quoted, Clin. 86

Seiffert, negative search for *Bacillus typhosus* B in human fæces, Epid. 55

——, search for *Bacillus suipestifer*, Epid. 58

Seligmann, C. G., and Shattock, S. G., thyroid gland of dachshund, Path. 299, 300

Sellheim, advocacy of embryotomy, Obst. 165

—— —— of formation of utero-abdominal fistula, Obst. 163, 165

——, indications for embryotomy, Obst. 214

Semicircular canal, external, injury to, in operations, Otol. 85

—— —— —— necrosis of, in case of chronic otitis media suppurativa (W. Milligan), Otol. 65

—— ——, posterior, caloric tests applied to exposed inferior crus of, in case in which disease in large sinus tympani caused persistence of discharge after radical operation (A. H. Cheatle and G. J. Jenkins), Otol. 54

—— —— (superior) fistula of ampulla of, on left side, bilateral radical mastoid operation (Sydney Scott), Otol. 73

Semolina as food for children, first mention, Child. 126

SEMON, Sir FELIX, K.C.V.O.—Discussion on ectopia of pituitary, Laryng. 53

——, discussion on equine larynx and "ventricle-stripping," Laryng. 89

—— —— on stricture of lower end of œsophagus, Laryng. 37

——, memorial to, Laryng. 149

Semon Lectureship in Laryngology, Laryng. 149, 150

Semple, Lieut.-Col. Sir D., examination of carriers, quoted, Epid. 135

——, isolation of *Bacillus typhosus* A in India, recorded by, Epid. 53

—— and Greig, proportion of typhoid carriers, given by, Epid. 4, 15, 119, 120, 121

Senator, cases not regarded as belonging to Werdnig-Hoffmann type of progressive spinal muscular atrophy, Neur. 73

——, percentage of albumin in blood and blood-serum in erythræmia, given by, Clin. 92

Senile changes, moderate, allayed by subthermal bath treatment, Baln. 97

Sepsis (oral), causing arthritis, Odont. 35

—— —— —— choroiditis, Odont. 31

—— —— —— episcleritis, Odont. 34

—— —— —— iridocyclitis, Odont. 29

—— —— —— keratitis, Odont. 34

Sepsis (oral), causing panophthalmitis, case ending fatally, Odont. 31, 32
—— —— —— thrombosis of cavernous sinus (G. Taylor), Odont. 113-118
—— —— —— —— —— ——, rarity, Odont. 113-118
—— ——, inflammatory diseases of eye due to (C. Goulden), Odont. 27-36
—— —— —— —— ——. method of infection. Odont. 27
—— —— —— —— ——, question of direct infection, Odont. 32, 35, 36
—— (uterine), causing lymphangitis, Obst. 73
Septic conditions in cows, Epid. 75
—— irritation, dental cysts following, Odont. 15-17
Septicæmia followed by panophthalmitis, Odont. 28
—— hæmorrhagica in animals, Surg. 48, 49
—— —— (probable) following abdominal operations, Surg. 58-64
—— ——, severe capillary and parenchymatous hæmorrhages of septic origin (W. G. Spencer), Surg. 48-57
——, see also *Puerperal septicæmia*
Septum (nasal). quadrilateral cartilage of, two specimens removed by submucous resection. showing fenestræ (Dan MacKenzie) Laryng. 5
—— ——, submucous resection, instrument for more efficient removal of nasal spine in (H. Tilley), Laryng. 25
—— ——, ulcer (chronic) of (Cecil Graham), Laryng. 56
Sequeira, J. H.—Acute lupus erythematosus, Derm. 91
—— alopecia areata in a syphilitic subject, Derm. 150
—— ——, of acquired syphilis in boy, aged 7, Derm. 79
——, an unusual case of linear nævus, Derm. 39
——, case of adenoma sebaceum (Pringle type), Derm. 58
—— ——, of elephantiasis of the lip. Derm. 56
—— —— of inherited syphilis treated by Ehrlich-Hata's "606," Derm. 10
—— —— of multiple idiopathic pigmentary sarcoma (so-called), Derm. 8.
—— —— of (?) mycosis fungoides, Derm. 77
——, discussion on cases for diagnosis, Derm. 91, 146
—— —— —— of acute lupus erythematosus, Derm. 51
—— —— —— of generalized eruption, probably mycosis fungoides, Derm. 21
—— —— —— of (?) granuloma inguinale, Derm. 136
—— —— —— of herpes zoster, Derm. 88
—— —— —— of hyperkeratosis of palms and soles, Derm. 18
—— —— —— of ichthyosis in child, treated by thyroid extract, Derm. 73
—— —— —— of lupus erythematosus associated with ulceration, Derm. 54
—— —— —— of pityriasis rubra pilaris, Derm. 24
—— —— —— of syphilitic auto-reinfection, Derm. 130
—— —— on chronic (artificial ?) skin eruption in a patient whose case has been followed for fourteen years, Derm. 44
—— —— on convenient method of giving "606" intravenously, Derm. 38
—— —— lichen planus of unusual distribution, Derm. 80
—— ——, on macular atrophy associated with lupus erythematosus, Derm. 143
—— —— on syphilitic affection of eighth nerve, Derm. 148
——, melanodermia associated with pernicious anæmia, Derm. 26
——, non-ulcerating tuberculide (?) Boeck's miliary sarcoid, following lupus vulgaris, Derm. 67
——, symmetrical angiomatous swellings in a tuberculous subject, Derm. 28
——, two cases of leprosy, Derm. 109
Serum from rabbit injected with *Bacillus paratyphosus* B, titration with the homologous extract. Path. 263, 265
—— —— —— injected with paratyphoid A, results from use of, Path. 263, 264
—— (antistreptococcic), injection before operating for rectal cancer, Surg. 131
—— ——, polyvalent, prophylactic injection at time of Cæsarean section, Obst. 195

Serum (antityphoid) and extracts from strains of *Bacillus typhosus* and *paratyphosus* and strain of *Bacillus Aertryck*, reactions between, Path. 260-262

—— ——, titration with extract made from another strain of *Bacillus typhosus*, Path. 257-259

—— —— —— with typhoid extract, Path. 274, 276

—— (pneumococcic, Merck's) administration in case of pneumococcal bronchitis, Med. 102

Sesamoid bone fractured by direct violence (R. Morton), Electr. 96

Severn, W. D., and Grey-Edwards, C., outbreak of milk-borne sore throat in Anglesey, Epid. 91

Sewage, bathing in water contaminated by, possible source of infection of poliomyelitis, Epid. 206

Sex-limited heredity, Med. 20

Sexual characters, secondary, Path. 304, 305

—— excitation caused by cutaneous scent of axillary glands, Path. 287

—— intercourse and urinary tuberculosis, Child. 224

—— memories in relation to origin of hysteria, Neur. 15, 16, 18

—— precocity in chick, Path. 305

Sharkey, S. J., absorption of milk and powdered casein in rectal feeding, quoted, Therap. 64

—— —— of salt and water in rectal feeding, quoted, Therap. 67

Sharp, J. G.—Discussion on ergot: a clinical experimental study, Therap. 150

——, ergot: a clinical experimental study, Therap. 114-150

Shattock, S. G.—Lamarckism and callosities, Path. 279-312

——, on the microscopic structure of uric acid calculi, Path. 110-146

——, review of Virchow's article on "Transformation and descent," quoted, Path. 306

—— and Battle, W. H., case of diffuse cancellous osteoma of femur following fracture, quoted, Surg. 138

—— and Dudgeon, L. S.—Experiments with air-dried mouse cancer: (1) the lethal effect of drying upon mouse cancer; and (II) the inability of air-dried mouse-cancer, or of a centrifugalized extract, to produce prophylactic or curative immunization, Path. 73-86

——, and Ord, W. M., microscopic structure of urinary calculi composed of calcium oxalate, quoted, Path. 114

—— and Seligmann, C. G., thyroid gland of dachshund, described by, Path. 299, 300

Shaw, Ernest H.—Discussion on indications for and technique of Cæsarean section and its alternatives, Obst. 193

Shaw, H. Batty.—Cases of Hastings Gilford type of symptomatic infantilism, Clin. 33

——, syphilis, chyliform ascites, dropsy and albuminuria, case of, Clin. 112

——, two cases showing unusual ulceration of the skin, Clin. 110

Shaw, W. Fletcher, and Donald, Archibald.—The symptoms and treatment of chronic endometritis, with special reference to the results of curetting, Obst. 37-49

Sheep, nematode infection accompanied by presence of *Bacillus coli*, Child. 218

Sherwin, case of poisoning by aconite, quoted (1837), Therap. 152

Shield-holder, used in skiagraphy, Electr. 86, 87

Shillitoe, A.—Case of corymbose syphilide, Derm. 152

——, case of (?) granuloma inguinale, Derm. 133

—— —— ——, pathological report, Derm. 134

—— ——, ringworm of the toe-nails, Derm. 151

Shock, avoidance of, during operations for rectal cancer, Surg. 130, 131

——, dilation of stomach following, Electr. 41

—— during or following abdominal panhysterectomy (Wertheim's method), Obst. 91

—— (reactionary) following operations under anæsthetics, Anæsth. 2

—— ——, follows ether more frequently than chloroform anæsthesia, Anæsth. 2

——, reduction of, under hypodermic injection of scopolamorphine before inhalation anæsthesia, Anæsth. 52, 53, 54

——, small amount, after combined operation for rectal cancer, Surg. 131

—— (surgical), method of avoidance under ether anæsthesia followed by nitrous oxide and oxygen, Anæsth. 3

Shore, C. S., on poliomyelitis in colts, quoted, Epid. 206, 207

Shoreditch, outbreak of typhoid fever in, Epid. 131

Short, A. R., note on the degenerations and cell changes resulting from section of the posterior nerve-roots in man, Surg. 208

Shoulder, congenital elevation of (Sprengel's deformity), Electr. 69

—— —— —— —— ——, bony connexion between vertebral column and inner border of scapula, detected by radiography, Elect. 69

—— —— —— —— ——, malformations of ribs in, Elect. 70

—— —— —— —— ——, mistaken for birth palsy, Electr. 69

Shoulder-joint, auscultation of, Med. 36

——, fractures about, treatment, Surg. 29

Shrubsall, F. C.—Discussion on the problem of disposition in pulmonary tuberculosis, Epid. 179

Shutly's method of artificial respiration in newly born infants as cause of Erb's paralysis, Child. 9

Sibley, W. K.—The relation between the teeth and certain diseases of the skin and mucous membrane, Odont. 71-84

Sidis, hypnoidal state of, Anæsth. 23

Sidmouth, health resort suitable for foreign invalids, Baln. 20

Sigmoid, tuberculosis of, hyperplastic, Path. 240

Sigmoidoscope in diagnosis of rectal cancer, Surg. 126, 128

——, use of, Obst. 336

Sigmoid sinus, injury to, during operation, Otol. 86

Silberberg, M. D.—The effect of atropine on the pulse-rate in cases under the influence of digitalis, Therap. 192-210

Silk, J. F. W.—Discussion on anæsthesia by ether and gas alternately, Anæsth. 8, 9

——, discussion on use of drugs before inhalation anaesthesia, Anaesth. 53

Silver, nitrate of, solutions in intranasal treatment of chronic frontal sinus suppuration, Laryng. 129

——, secondary rays derived from, Electr. 67

—— —— —— ——, penetrative power same as β-rays of radium, Electr. 67

—— —— —— ——, therapeutic effects, Electr. 67

Simmons, G. **Allpress**.—Discussion on lantern slides of three cases, Electr. 74

——, discussion on rigidity of the spine, Clin. 150

——, skiagrams illustrating position of bismuth after bismuth meal in case of idiopathic hypertrophic dilatation of the colon, Child. 20-22

——, X-ray examination in case of mollities ossium of forty-five years' duration, Clin. 99, 104

Simnitzky, experiments as to differentiation of typhoid and paratyphoid bacillus group, Path. 252

Simon, Sir John, prevalence of stone in bladder in Norfolk, quoted, Baln. 125

Simpson, Sir J., hermaphroditism, quoted, Obst. 111

Simpson, W., "Hydrological Essayes," Baln. 79

Singer's nodules, methods of removing, discussed, Laryng. 40-42

Sinus tympani (large), disease in, causing persistence of discharge after radical operation (A. H. Cheatle and G. J. Jenkins), Otol. 54

Sinusitis, diagnosis of, value of peripheral field contractions in, Laryng. 166, 167

—— (malignant), due to scarlet fever, Laryng. 32

——, perimeter test for implication of optic nerve in disease of the sinuses, Laryng. 163, 164-166

——, see also *Sphenoidal sinusitis*

Sinusoidal current, application by Abrams's method in treatment of hyperchlorhydria, Electr. 45

—— —— —— —— in severe functional dyspepsia, Electr. 36

—— —— ——, intra-gastric application, Electr. 37

Sitting attitude of monkeys, Path. 312
Skelding, H.—Discussion on case of congenital dilatation of colon, Child. 76
Skiagrams, demonstration of (R. Knox), Electr. 76
—— —— (S. Gilbert Scott), Electr. 75
——, method of viewing stereoscopically, Electr. 92
Skiagraphy, chair for screening chest in, Electr. 88
——, couch for use of patients under, Electr. 88
—— of foreign bodies, Electr. 91
——, technique of (A. D. Reid), Electr. 84-92
—— —— for various regions of body, Electr. 87-91
—— —— —— —— ——, presenting difficulties, Electr. 89-91
Skin as portal of entry of bacteria into body, Child. 207, 208
——, atrophy of, congenital, Path. 302
——, diseases of, connected with dental trouble, Odont. 74-78
—— ——, thyroid extract in, Derm. 72
—— —— treated by vaccine of pyorrhœa alveolaris, Odont. 82
——, effect of calcium salts on, Baln. 122
—— of head and face, nerve-distribution of, Odont. 72, 73
——, re-hydration of, effected by mineral water baths, Baln. 16
—— —— —— by sea-bathing, Baln. 16, 17
——, relation between the teeth and certain diseases of the skin and mucous membrane (W. K. Sibley), Odont. 71-84
——, sarcoma of, idiopathic multiple hæmorrhagic, Derm. 42, 4
——, ulceration of, unusual, two cases showing (H. Batty Shaw), Clin. 110
——, see also *Glands*, cutaneous
Skin-eruption (chronic artificial?) in patient whose case has been followed for fourteen years (F. Parkes Weber), Derm. 43.
—— eruptions prevalent in spring, Therap. 14. 15
Skull, base of, fractured, treatment, Otol. 100, 101
—— —— ——, followed by labyrinthine suppuration twenty-one months later, proving fatal, Otol. 89
—— —— —— complicating middle and internal ear, Otol. 87
—— from barrow, showing somewhat symmetrical mandibular exostoses (W. de C. Prideaux), Odont. 39
——, pulsating tumour of, resembling thyroid tissue, Clin. 125
Slack bandaging in fractures, importance of, Surg. 26
Slaughter-houses (private), agency in spreading food-poisoning, Epid. 69
Sleep, F.—A successful surgical and prosthetic treatment of a case of excessive hypertrophy of the gums and alveolar processes of the jaws, Odont. 18-26
Sleeping sickness, see *Trypanosomiasis* (human)
Sloan, S., method of applying induced current, quoted, Electr. 36
——, success or failure in electro-therapy; a consideration of some of the causes, Electr. 99-113, 114
Sloane, Sir Hans, on mortality of dry-nursed compared with that of suckled infants, quoted, Child. 129
Smellie, on action of ergot in floodings and abortions, quoted, Therap. 142
Smith, E. Bellingham.—Case of spasmus nutans, Child. 75
——, discussion on case of von Jaksch's anæmia, Child. 157
—— —— on a preliminary communication on syphilis and mental deficiency, Child. 171
—— —— on the oculo-motor type of polio-encephalitis, Child. 93
——, and Woodforde, A. W. G.—A preliminary communication on syphilis and mental deficiency, Child. 166
Smith, Eustace, on artificial feeding of infants, quoted, Child. 137
——, "Wasting diseases of children," Child. 137
Smith, F. J.—Discussion on case of syringomyelia, with symptoms precipitated by trauma, Neur. 38

Smith, G. Bellingham, supposed primary abdominal pregnancy in rabbit, recorded by Obst. 233

SMITH, HEYWOOD.—Discussion on carcinoma of body of uterus, Derm. 111

——, discussion on case of gangrene of vagina, probably due to chemical effect of pink rubber ring pessary, Obst. 327

Smith, J. Lorrain, and Haldane, J. S., carbon monoxide method of, Clin. 89

——, and McKisack, H. L., blood-changes in case of adherent pericardium and chronic cyanosis, quoted, Clin. 92

Smith, Professor Lapthorne, practice as regards removal of appendix in pelvic operations, Obst. 351

SMYTH, F. R.—Drugs used as local anæsthetics in mouth, Therap. 51-62

Snow-water, melted, drinking of, producing goitre, Baln. 122

Soamin, possible action of, on lecithin of protozoa, Path. 2

Sobernheim, negative search for *Bacillus typhosus* B in fæces and urine, Epid. 55

——, search for *Bacillus enteritidis*, by, Epid. 61

Soda, glyco-cholate of, in exophthalmic goitre, Clin. 124

Sodium biurate, solubility of, Baln. 122, 123

—— ——, solvent action of distilled water on, how lessened, Baln. 123

——, bromide of, injection in anorexia nervosa, Child. 80

—— nitrate, reaction of arteries to, Therap. 89

—— sulphate, medical action and use of, Therap. 21

—— ——, reaction of arteries to, Therap. 90

——, urate of, trace of, in uric acid calculus, cancellous variety, Path. 118

Solar system, nebular origin of, Path. 143

Soleus muscle, traumatic myositis ossificans in connexion with, Surg. 157, 158

Soles, see *Palms and soles*

Sömme, Dr., outbreak of milk-borne sore throat at Christiania, reported by, Epid. 97

Somnambulism, active, induction of hypnotism to state of, Anæsth. 16

—— (artificial), identical with hysteria, Neur. 10

——, fit of hysteria comparable to, Neur. 5

Sondermann's suction apparatus in acute frontal sinusitis, Laryng. 127, 146

Soper, case of typhoid carrier, quoted, Epid. 17

Sore throat, caused by invasion of *Streptococcus anginosus*, Epid. 79

—— —— —— —— of *Streptococcus pyogenes*, Epid. 79

—— ——, outbreak, milk-borne, due to infection from ulcerated teats of cows, Epid. 82

—— —— —— ——, in British Isles, Epid. 89-97, 103, 104

—— —— (septic), caused by ingestion of milk from cows with mastitis, Epid. 76

—— —— ——, outbreak of concurrent, with scarlet fever, and derived from same milk-borne source, Epid. 101

Sound, insensibility to, resulting from inhibitory mental process, Anæsth. 16

Soupault and Jouaust, dysmenorrhœa and appendicitis, quoted, Obst. 356

Southwark, outbreak of typhoid fever in, Epid. 130

Spa physicians, duties of, Baln. 80

Spa treatment, common factors in, Baln. 72, 73

—— —— concerned only with chronic disease, Baln. 109, 112

—— —— founded on humoral doctrine, Baln. 109, 110

—— —— in days of Hippocrates, Baln. 9

—— ——, principles of, simple, Baln. 110

Spaeth, ovarian tumour obstructing delivery, quoted, Obst. 256

Spark-coil of new induction coil apparatus, Electr. 79, 80

Sparteine, reaction of arteries to, Therap. 90

Spasm and pyloric hypertrophy (E. Cautley) Child, 185

——, division of posterior spinal nerve-roots for, Surg. 213

—— of colon, inhibited by belladonna, Med. 31

—— of gastric musculature inhibited by belladonna, Med. 29, 30, 33

Spasmus nutans, case of (E. Bellingham Smith), Child, 75
Spät, experiments as to differentiation of typhoid and paratyphoid bacillus group, Path. 253
Specialism, professional, dangers of excess in, how to avoid, Med, 156
Spedale Santa Maria Nuova (Florence) baths at, Baln. 89
Speech, results of operation for cleft palate on, Surg. 186-188, 190, 194, 195
Speech training after operations for cleft palate, Surg. 185
SPENCER, H. R.—Carcinoma of the body of the uterus in a patient, aged 29. Obst. 308-312
——, case of ovarian tumour obstructing delivery, quoted, Obst. 256
——, cervical glands in the uterus of the fœtus at term, Obst. 281
——, discussion on indications for and technique of Cæsarean section and its alternatives, Obst. 189
—— —— on operation for cancer of uterus, Obst. 101
—— —— on rupture of gravid uterus, Obst. 153
—— —— on subtotal hysterectomy for fibromyoma uteri, Obst. 143
—— —— on thyroid tissue in ovarian embryoma, Obst. 218
—— —— on vaginal secretion in infants, Obst. 31
—— —— method of ligation of cervix, preliminary to abdominal hysterectomy, Obst. 178
——, recommendation of subtotal hysterectomy in septic cases, by, Obst. 177, 187
——, suppurating ovarian tumour, perforated into small intestine and omentum, successfully removed with affected bowel, omentum, and appendix, Obst. 157
SPENCER, W. G.—Discussion on case for diagnosis, Laryng. 15
——, discussion on case of ectopia vesicæ in a female child, Clin. 158
—— —— —— of recurrent attacks of post-typhoidal osteitis, Clin. 157
—— —— —— on treatment of cleft palate, Surg. 197
——, lingual thyroid ; thyroid gland in neck not palpable. Laryng. 13
——, septicæmia hæmorrhagica, severe capillary and parenchymatous hæmorrhages of septic origin, Surg. 48-57, 67
Spender, J. Kent. association between rheumatoid arthritis and Graves's disease, quoted, Child, 36
Sphenoidal cells, effect of disease of, on vision, Laryng. 163, 164-166
—— sinusitis (acute) followed by unilateral optic neuritis and complete ophthalmoplegia (H. J. Davis), Laryng. 28
—— ——, untreated, deaths following, Laryng, 140
Sphincter, Nelaton's, Surg. 92
Sphygmomanometer, records of blood-pressure enumerating maximum figure obtained from, only partial, Med. 75, 76
SPICER, R. H. SCANES.—Discussion on case of carcinoma of larynx, Laryng. 93
——, discussion on case of endothelioma of palate, with metastatic growth in neck, treated by radium, Laryng. 95
—— —— on case of party-wall cancer of larynx and œsophagus, Laryng. 101
—— —— on complete stenosis of larynx from epithelioma in a man, aged 23, Laryng. 120
—— —— on ectopia of pituitary, Laryng. 53
—— —— on equine larynx and "ventricle-stripping." Laryng. 91
——, malignant disease of pharynx under treatment by radium, Laryng. 64
——, origin of cancer in larynx, Laryng. 19
Spielmeyer, late case of amaurotic idiocy (Tay-Sachs disease), recorded by, Path. 169
Spiess, advocacy of intranasal operations in chronic frontal sinus suppuration, Laryng. 130
Spiller, late case of amaurotic idiocy (Tay-Sachs disease), recorded by, Path. 169
Spina bifida, analysis of fluid from case of (1811), Med. 153
Spinal column, rigidity of, through ossification of ligaments, protective and compensatory process in spondylitis, Baln. 29, 32
—— cord, actinomycosis in neighbourhood of, Clin. 81
—— ——, cells from anterior horn of cervical and lumbar regions from cases of progressive spinal muscular atrophy (toxic neuritis type), Neur. 64
—— —— —— —— —— —— —— —— —— —— —— —— —— (Werdnig-Hoffmann type), Neur. 59-61

Spinal cord, cells of anterior cornu of, from case of amaurotic idiocy (Tay-Sachs disease), Path. 190
—— ——, changes in central canal of, in human trypanosomiasis, Path. 30
—— ——, grey matter from case of progressive spinal muscular atrophy (Werdnig-Hoffmann type), Neur. 58
—— —— (inner), traumatic neuritis of (Klumpke-paralysis) (Wilfred Harris), Neur. 31
—— —— sclerosis (disseminated), probable in case of pseudo-bulbar paralysis of doubtful origin (T. Grainger Stewart), Neur. 46
—— ——, sections through, from cases of progressive spinal muscular atrophy (Werdnig-Hoffmann type), Neur. 55
Spine, application of hot and cold douches to, in neurasthenia, Baln. 41
——, caries of, see *Caries* (spinal)
—— (cervical), deformity, case of (P. Maynard Heath), Child. 105
——, osteo-arthritis of, Clin. 149
——, rigidity of, case of (H. D. Rolleston and G. P. Humphry), Clin. 146
—— ——, see also *Poker-back*
—— of tibia, fracture of, Surg. 17, 44
Spirochæta dentium present in ulcerative stomatitis in children, Child. 73
Spirochæte and trypanosome infections, comparative neuropathology of (F. W. Mott, F.R.S.), Path. 1, 2, 3
Spirochætes linked by characters to protozoa rather than to bacteria, Path. 1
Spleen, enlargement, case of (E. Cautley), Child, 28
—— microscopical appearances at necropsy of case of congenital obliteration of bile-ducts, Child. 45
——, "tumid," painful affections of side from (reported 1811), Med. 149
—— —— —— —— —— —— ——, probably case of pleurisy, Med. 154
Splenectomy in splenomegaly, Clin. 132
——, in treatment of splenic anæmia (G. A. Sutherland and F. F. Burghard), Clin. 58
—— —— —— ——, method of operation, Clin. 65
Splenomegaly, chronic acholuric jaundice with, Clin. 112
—— (? congenital family cholæmia), case of (R. Hutchison), Clin. 129
——, "familial," Clin. 68
——, jaundice with, see *Jaundice* (splenomegalic)
—— of inherited syphilis in children (F. P. Weber), Child. 65
—— —— —— —— and Banti's disease, Child. 69
—— —— —— ——, symptoms, Child. 68
—— —— —— ——, treatment, Child. 68
—— —— —— —— ——, caution in, Child. 68
——, treatment by splenectomy, Clin. 132
—— with recurrent jaundice, ending in hepatic cirrhosis and ascites (F. P. Weber), Child. 62
——, see also *Anæmia* (splenomegalic)
Splints in fractures, method of application more important than kind of, Surg. 4
——, twisted metal, for fracture of humerus and Colles's fracture, Surg. 25
SPOKES, SIDNEY.—Case of pneumococcal stomatitis, Odont. 11
——, discussion on cyst of jaw, Odont. 17
—— —— on destruction of teeth by milk-germs, Odont. 9
—— —— on distribution of nerve-fibres of dental pulp, Odont. 53
Spondylitis, ossification of ligaments, compensatory change, Baln. 29, 32
——, with progressive muscular contracture (W. Harris), Neur. 42
—— —— —— —— (? myositis fibrosa), case of (W. Harris), Clin. 145
—— deformans, case of (A. E. Garrod), Clin. 29
Spondylose rhizomélique, case of (F. E. Batten), Neur. 40
—— —— —— (E. F. Buzzard), Neur. 42
Sporothrix Schenkii, Derm. 114
Sporotrichosis, areas involved in, and complications of, Derm. 114

Sporotrichosis, case of (H. G. Adamson), Derm. 101
——, case of, cultures from, Derm. 117
—— ——, description, Derm. 115
—— ——, fungus from, Derm. 117
—— ——, pathological anatomy, Derm. 118
——, cultivation media for, Derm. 115
——, geographical distribution, Derm. 113, 114
——, sources of infection, Derm. 115
—— —— ——, animal, Derm. 115
—— —— ——, vegetables, Derm. 115
——, and syphilis, Derm. 114
——, and tuberculosis, Derm. 114
Sprain as cause of myositis ossificans, Surg. 157, 158
Sprengel's deformity, see *Shoulder*, congenital elevation of
SPRIGGS, E. I.—Discussion on case of rheumatoid arthritis, Child. 38
——, discussion on the therapeutic use of the digitalis group, Therap. 35
——, and MOLONY, J. B. DE W.—Angiomata of the left loin and leg, Child. 85
"Spring rashes," Therap. 14, 15
Sputum, tubercle bacillus in, indication of infection, Epid. 46, 47
Squill, dosage of, Therap. 28
——, therapeutical value tested by accurate clinical records, Therap. 7-12
Squint, see *Strabismus*
SQUIRE, J. EDWARD, C.B.—Some points in relation to tuberculosis and its prevention, Epid. 27-50
Stab wounds of auditory meatus, Otol. 82
Staining, methods of, employed for sections from specimens of amaurotic idiocy (Tay-Sachs disease), Path. 177-194
Standard bread, Odont. 97
—— ——, supposed advantages of, Odont. 99, 100
Stanley's family of multiple cartilaginous exostoses, Med. 4, 5
Staphylococci, percentage of cases of mastitis in cows due to, Epid. 78
Staphylococcus causing puerperal infection, Obst. 188
Staphylococcus albus associated with infective endocarditis, Obst. 194
—— —— —— with "puerperal fever," Obst. 194
—— —— in cervical canal, Obst. 193
—— *aureus*, infection by, causing chronic dermatitis of pinna, case under treatment by vaccine (C. E. West), Otol. 38
—— ——, presence in cavernous sinus thrombosis, Odont. 117
—— *pyogenes aureus* causing puerperal infection, Obst. 167
—— —— —— from cultures from teeth in case of oral sepsis causing infection of cavernous sinus, Odont. 116
Staphylococcus and streptococcus infection compared, in frontal sinus suppuration, Laryng. 132
Starvation, excretion of nitrogen in subjects of, Therap. 70
Status epilepticus, condition of, method of successful treatment, Baln. 8
—— —— —— ——, produced by ministrations of Christian Scientist, Baln. 8
Statuvolism, see *Consciousness*, voluntary loss of
STEADMAN, F. ST. J.—Cases of malocclusion of permanent molars, Odont. 110-113
Steiermark, epidemic of poliomyelitis in (1909), Epid. 208
Stellate reticulum, Odont. 89
Stenger, labyrinthine concussion, quoted, Otol. 103
Stenosis, complete, of larynx from epithelioma (StC. Thomson), Laryng. 119
——, pulmonary (congenital) (?), case of (J. Galloway), Clin. 82
—— —— —— ——, with special consideration of nature of secondary blood-changes (F. Parkes Weber and G. Dorner), Clin. 85-94

Stenosis, subglottic, after tracheotomy, case of (H. Barwell), Laryng. 6

——, tracheo-laryngostomy for, following tracheo-laryngotomy (W. Hill), Laryng. 109

Stephenson, S.—Case of night-blindness with peculiar conjunctival changes, Child. 188

——, discussion on ulcerative stomatitis in children, Child. 73

—— —— on case of inherited syphilis and "blue sclerotics," Child. 99-100

——, embolism of the central artery of the retina in a girl, aged 11¾ years, Child. 56

——, the oculo-motor type of polio-encephalitis, Child. 87-93

Stereoscope (Pirie's), Electr. 92

Stereoscopic method of viewing skiagrams, Electr. 92

Sterility in chronic endometritis, effect of curetting on, Obst. 48

Sternberg, classification of chloromas, by, Path. 69

Sterno-mastoid muscle, extension backwards to. of abscess in right auditory meatus, invaded by erysipelas of head; invasion of whole sheath of that muscle (A. H. Cheatle), Otol. 111

Stethoscope, date of introduction, Med. 150

——, with chest-piece, in auscultation of joints, Med. 35, 36

Stevens, T. G.—Discussion on chronic endometritis, Obst. 50

——, microscopical report on case of sarcoma of uterus, Obst. 112

Steward, F. J.—Discussion on the treatment of frontal sinus discharge, Laryng. 141

Stewart, Purves.—Discussion on division of posterior spinal nerve-roots, Surg. 251

Stewart, T. Grainger.—Case of pseudo-bulbar paralysis of doubtful origin, Neur. 23

—— —— —— —— —— ——, probably disseminated sclerosis, Neur. 46

——, discussion on case of congenital brain defect, with stamping gait and tendency to fall backward, Neur. 34

—— —— on case of syringo-bulbia, Neur. 27

—— —— —— of toxic neuritis of doubtful nature, Neur. 22

——, and Holmes, Gordon, cerebellar tumours, referred to, Otol. 37

Steyr, epidemic of poliomyelitis in (1908). Epid. 209

Stockholm, outbreaks of milk-borne gastro-enteritis and diarrhœa at, Epid. 98

——, University of, lectures on balneology delivered at. since 1880, Baln. 116

Stokes-Adams symptoms in a lad, aged 18 (J. Galloway and W. J. Fenton), Clin. 39

—— —— —— ——, diagnosed from minor epilepsy, Clin. 41

Stomach, acute hæmorrhages into, Surg. 49

——, cancer of, administration of belladonna valuable in certain cases, Med. 33

—— ——, tuberculous invasion, Child. 226

——, catarrh attacking, Epid. 192

——, dilatation of, accompanying low blood-pressure, Med. 79

—— ——, following operations or shock, treatment by lavage and static charge, Electr. 41

—— ——, in atonic dyspepsia due to disturbances of nervous system, Electr. 40

—— ——, severe, success of electro-therapy in, Electr. 110

—— ——, treatment by farado-massage, Electr. 40

—— —— —— ——, support of intestines necessary under, Electr. 40

——, diseases, cases submitted to radioscopy, alleged danger of bismuth meals, Electr. 60

——, fibromatosis of, Path. 240

—— ——, similarity to hyperplastic tuberculosis of colon, Path. 240

——, fluids entering, rendered isotonic with blood, Baln. 39

——, lavage of, see *Lavage* (gastric)

——, motor insufficiency with hyperchlorhydria, treatment by electricity, Electr. 35

—— —— —— —— —— —— —— —— ——, methods, Electr. 36

—— musculature, spasm of, inhibited by belladonna, Med. 29, 30, 33

——, shape of, Electr. 53

——, ulcer of, beneficial action of ions of manganic acid, of zinc sulphate, or bichromate of potash, on, Electr. 42

—— ——, effect of oral administration of belladonna before meal, on acid and peptic values of stomach contents in, Med. 26, 29

Stomach, ulcer of, treatment by electricity and lavage, Electr. 41, 42
—— —— —— by rectal feeding injurious, Therap. 72, 73
—— —— —— (acute), formation of, influence of diet upon (C. Bolton), Path. 53-59
—— —— —— —— takes place more easily during period of digestion, Path. 56
—— —— ——, healing of, accelerated by milk diet, Path. 57, 58
—— —— —— ——, delayed by meat diet, Path. 57, 58
—— —— —— —— ——, by motor insufficiency, Path. 54
—— —— —— —— ——, by retention of food in stomach, Path. 54
—— —— —— ——, influence of diet upon (C. Bolton), Path. 53-59
—— —— ——, production of, experimental, in cats, by gastro-toxic serum, Path. 53, 54, 55
—— —— —— and chronic, treatment, dietetic, Path. 59
—— —— —— —— ——, principles of, Path. 58, 59
Stomach contents, acid-values of, effect of oral administration of belladonna on, before meal, Med. 25
—— —— —— —— —— —— —— —— —— —— ——, as associated with hyper-acidity, normal acidity, and hypo-acidity, Med. 25, 26
—— —— ——, reduced by administration of atropine, Med. 23, 24
—— ——, peptic values of, effect of oral administration of belladonna on, before meal, Med. 26-29
—— ——, volume of, not diminished by administration of belladonna, Med. 31
Stomach-tube, use of, in feeding experiments to determine portals of entry in phthisis, Child. 206, 212
Stomatitis complicated by necrosis of jaw, Child. 31
—— (pneumococcal) accompanied by conjunctivitis, Odont. 12
—— ——, case of (S. Spokes), Odont. 11
—— (ulcerative), in children (S. F. Rose), Child. 70
—— —— ——, ætiology, Child. 70
—— —— ——, bacteriology, Child. 73
—— —— ——, course of, Child. 71
—— —— ——, treatment, Child. 72
Stopcock, new form of, for gas and air administration (Frank Coleman), Anæsth. 40
Störck, Baron, first introduction of aconite into practice by (1762), Therap. 152
STORDY, T.—Case of absorption of a permanent molar due to an unerupted premolar, Odont. 37
Stott, outbreak of cow-pox in herd supplying milk to Brighton, reported by, Epid. 75, 99, 103
Stovaine, Therap. 56
STOWERS, J. H.—Discussion on case for diagnosis, Derm. 63
——, discussion on case of generalized eruption, probably mycosis fungoides, Derm. 21
—— —— on two cases of nævus unius lateris, Derm. 66
Strabismus (convergent, unilateral), percentage of age of commencement, Child. 89
—— (encephalic), liable to be confused with concomitant convergent type, Child. 92, 93
Strain, as factor in production of diseases of joints, Baln. 28
Strandgaard, on heredity in phthisis, quoted, Epid. 172
Strasburg, percentage incidence of gall-stones in post-mortem examinations at, Path. 88, 89
——, typhoid fever carriers at, Epid. 12, 19
Strathpeffer, mineral waters at, Baln. 15
——, neurasthenia benefited at, Baln. 42
STREET, A. F.—Discussion on calcareous (hard) drinking water, Baln. 128
Streptococci, differentiation of, Epid. 70
——, percentage of cases of mastitis in cows due to, large, Epid. 78
Streptococcic infection in human trypanosomiasis, Path. 15
Streptococcus and *Bacillus coli* causing puerperal infection, Obst. 167
—— and staphylococcus, infection by, compared in frontal sinus suppuration, Laryng. 132
—— *anginosus*, inoculation of, in goats, does not produce mastitis, Epid. 80
—— ——, invasion by, causing sore throat, Epid. 79

Streptococcus anginosus morphologically and culturally similar to *Streptococcus mastitidis*, Epid. 79
—— ——, virulence high, Epid. 79
—— ——, virulent effects produced by inoculation into mice, Epid. 79, 80
—— causing puerperal infection, Obst. 167, 188
—— commonest organism found in puerperal infection, Obst. 194
—— from fatal case of acute lymphadenitis, inoculation into goat, Epid. 85
—— —— —— —— —— —— ——, survival as saprophyte for seven months, Epid. 85
—— infection causing panophthalmitis, Path. 247, 248
—— in septicæmia hæmorrhagica, Surg. 48-57
—— *mastitidis*, Epid. 78
—— ——, cultivation of, Epid. 78
—— ——, inoculation of, in goats produces mastitis, Epid. 80
—— ——, morphologically and culturally similar to *Streptococcus anginosus*, Epid. 79
—— —— not pathogenic to man, Epid. 80, 81, 95, 103, 104
—— ——, virulence low, Epid. 79, 103
—— *pyogenes*, invasion by, causing sore throat, Epid. 79
—— ——, mastitis in cows infectious to man when due to, Epid. 95
—— ——, presence of, demonstrated in case of panophthalmitis, Odont. 32
—— ——, saprophytic in genital tract, Obst. 170
—— *salivarius*, Clin. 140, 141
Stress test, alternating (Arnold's), for cast and rolled gold plate, Odont. 60
String galvanometer, see *Galvanometer* (string)
Stroganoff, vaginal secretion in infants, quoted, Obst. 30
Strongylus contortus, *Strongylus pergracilis*, see *Nematodes*
Strophanthus, dosage of, Therap. 27
——, therapeutical value tested by accurate clinical records, Therap. 7-12
Struve, recommendation of egg and beer for young children after weaning, Child. 123
Strychnine, as "tonic" drug, Med. 67
——, dilute solution of, positive galvanic pole immersed in, for use in stomach for gastritis with vomiting, Electr. 41
——, effect of, on low blood-pressure, Med. 67, 74
——, therapeutical value tested by accurate clinical records, Therap. 7
—— and atropine, hypodermic injection before chloroform inhalation, Anæsth. 54
STUART-LOW, W.—Cases illustrating the treatment of frontal sinus suppuration, Laryng. 126
——, cystic adenoma of facial parotid gland, Laryng. 59
——, discussion on case of lingual thyroid, Laryng. 81
—— —— —— of nasopharyngeal fibrous polypus, Laryng. 61
—— —— on eight cases of mastoid operation; (Case I), Otol. 126; (Case IV), Otol. 130; (Case VI), Otol. 132
—— —— on treatment of frontal sinus discharge, Laryng. 147
——, epithelioma on the right palato-glossal fold in a man, aged 48, Laryng. 60
Stuart Period, rearing of infants during, Child. 116
STURRIDGE, E. J.—Discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 49
Subarachnoid space, invasion of, by *Trypanosoma gambiense*, Path. 6, 17
Subconscious ideas, Neur. 2
Subglottic stenosis after tracheotomy, case of (H. Barwell), Laryng. 6
Sublimate (corrosive), poisoning by, complicated by acute nephritis, Clin. 168
Submucous resection of nasal septum, instrument for more efficient removal of nasal spine in (H. Tilley), Laryng. 25
Suckling period of, allowed for in seventeenth and eighteenth centuries, Child. 119, 120
—— —— curtailments in eighteenth century, Child, 121
—— ——, progressive decline in, Child. 138
Sugar as article of diet for young children in eighteenth century, Child. 122, 123

Sugar in causation of dental caries, Odont. 96, 100, 102

——, viscosity of, in mouth, experimentally demonstrated, Odont. 96, 104

Sugar fermentation tests, destruction of bacteria by, Epid. 136

Sugar media employed in testing modifications in biological characteristics of *Bacillus typhosus*, Path. 44

Sugars, gas production of intestinal bacteria from, entirely or almost abolished, Path. 102, 103, 105, 109

Suggestion and surgical anæsthesia, Anæsth. 22

—— before operation, Crile's method, Anæsth. 22, 23

—— combined with chloroform anæsthesia, Anæsth. 22, 23

—— distinct from hypnotism, Anæsth. 23

—— in balneology, Baln. 5, 6

—— (post-hypnotic), see *Post-hypnotic influence*

—— (pre-anæsthetic), obtained by suggestion in waking state, Anæsth. 23

——, susceptibility to, racial, Anæsth. 23, 24

——, therapeutic abuse of, Baln. 5, 6

Sulph-hæmoglobinæmia, chronic, secondary polycythæmia not noted in, Clin. 86, 87

Sulphates, medical use and action of, Therap. 20-22

Sulphides as intestinal antiseptic in form of natural mineral waters, Therap. 19

—— (metallic), less useful than sulphur as intestinal antiseptics, Therap. 18

Sulphite and thiosulphate of sodium, medical use of, Therap. 19

Sulphocyanate, excretion of, under non-purin diet, Baln. 57

—— in saliva before and during taking of sulphur water, Baln. 57, 58

—— ——, estimation with ferric chloride in Meisling's colorimeter, Baln. 48

Sulphur and certain of its compounds, action and uses of, as intestinal antiseptics (R. B. Wild), Therap. 13-24

——, antiseptic power of, experiments, Therap. 17

——, depressant effects of, Therap. 24

——, effect of, on secretions of alimentary canal, Therap. 17, 18

—— in brain-tissue, Path. 196

—— in amaurotic idiocy (Tay-Sachs disease), diminution of lipoid and increase of extractive forms, Path. 198

——, insolubility of, Therap. 16

—— in treatment of dysentery and diarrhœa, rheumatism and gout, Therap. 15, 16

——, organic compounds of, in medicine, Therap. 22-24

——, vegetables containing, Therap. 22, 23

—— compounds in alimentary canal, chief action not purgation, but local antisepsis, Therap. 14, 15

Sulphur water, action on contents of stomach and small intestine, Baln. 56

—— ——, blood-pressure during taking of, Baln. 56

—— ——, comparative excretion of urine in fæces, before and after taking, Baln. 49

—— ——, effect on tissues oxidizing, Baln. 62

—— ——, excretion of exogenous uric acid during taking of, Baln. 50, 51, 52

—— —— —— of indican before and after taking, Baln. 55

—— —— —— of kreatinin before and after taking, Baln. 52, 53

—— —— —— of nitrogen during taking of, Baln. 50, 51

—— —— —— of phosphates before and after taking, Baln. 53, 54

—— ——, old (Harrogate), aperient action of, effect on low blood-pressure, Med. 61

—— ——, positive diuretic action of, Baln. 50

—— ——, strong (Harrogate), active agent or agents in, Baln. 59, 60

—— —— —— —— pharmacological effect (D. Brown), Baln. 45-52

—— —— —— presence of barium in, Baln. 60

—— ——, sulphocyanate in saliva before and during taking of, Baln. 57, 58

—— ——, therapeutical value of, Baln. 60

—— ——, used at Harrogate in treatment of colitis, Therap. 19

Sulphretted hydrogen in intestinal canal, antiseptic value of, Therap. 14, 15
Sulphurous acid, medical use of, Therap. 19
Sumatra, paratyphoid fever (A) in, Epid. 53, 54
Summer diarrhœa, association of *Bacillus enteritidis* with, Epid. 61
—— ——, *Bacillus paratyphosus* B not found associated with, Epid. 55
—— eruption, case of (S. E. Dore, for Sir Malcolm Morris), Derm. 4
Sun, rays of, exposure to, causing lupus erythematosus associated with macular atrophy, Derm. 144
Suprarenal extract, application in acute frontal sinusitis, Laryng. 127
—— —— in local anæsthesia, Therap. 53, 59-62
Surbiton, outbreak of milk-borne sore throat at, Epid. 91
Surface therapeutics, Baln. 99
Surgeons, operating, scepticism as to success in non-operative treatment of cancer, Electr. 50
Surgery, cold water dressings in, early advocacy of, Baln. 87
——, progress of (1811), illustrated by contents of *Medico-Chirurgical Transactions*, vol. ii. (J. Mitchell Bruce), Med. 147-156
Sutherland, G. A.—Case of cardiolysis for adherent pericardium, Child. 41
——, chronic tetany, Child. 17
——, discussion on case of congenital absence of muscles, Clin. 84
—— —— —— —— —— insufficiency of soft palate, Child. 24
—— —— —— —— —— pulmonary stenosis, Clin. 95
—— —— —— of enlargement of liver, Child. 55
—— —— —— of familial pigmentary dermo-fibromatosis, Clin. 76
—— —— —— of splenic enlargement, Child. 29
—— —— —— of splenomegaly (? congenital family cholæmia), Clin. 132
—— —— —— ——, with recurrent jaundice, Child. 69
—— —— on cerebral sclerosis and idiocy, Child. 185
—— —— on pyloric hypertrophy and spasm, Child. 185
—— and Burghard, F. F.—Treatment of splenic anæmia by splenectomy, Clin. 58
Sweden, epidemics of poliomyelitis in (1899), Epid. 200; (1905), Epid. 204
Swelling at junction of right vocal cord and apex of right vocal process (J. Donelan), Laryng. 80
——, formation of, as result of separation of periosteum by muscles, Surg. 143
—— —— —— —— —— ——, diagnosed as periosteal sarcoma, Surg. 143
——, pulsatile, in third and fourth left intercostal spaces, ? thoracic aneurysm (H. French), Clin. 22
Swellings, angiomatous, symmetrical, in tuberculous subject (J. H. Sequeira), Derm. 28
Swine fever, cause of, Epid. 59
Swinemünde (Baltic health resort), climate compared with that of Bournemouth, Baln. 19
Switzerland, teaching of hydrology in, Baln. 116
Sycosiform syphilide, case of (G. N. Meachen), Derm. 105
Sydenham, epidemic constitution of air, quoted, Epid. 190
—— —— epidemics and other diseases and states of the weather, quoted, Epid. 189
Symes-Thompson, H. E.—Case of infantilism, Clin. 45
Symington, Professor J., alteration of limits of rectum, by, Surg. 87
——, reasons against belief in appendiculo-ovarian ligament, Obst. 346
Symmers, St.C., and Whitla, Sir W., experiments to prove introduction of tuberculous infection by way of alimentary tract, Child. 205, 229
Symonds, C. J.—Case of cicatrizing sarcoma (shown by K. Digby), Derm. 126
Symonds, H. P.—Discussion on fractures in the neighbourhood of joints, Surg. 38
Symonds's rubber intubation apparatus, modification of, Laryng. 9
Symphysiotomy, Obst. 190, 208
——, date of first performance, Obst. 162
——, revival and decline, Obst. 162
——, risks of, Obst. 162

Synovial crackle, Med. 37
—— —— in dry synovitis, Med. 37
—— —— in later stage of tuberculous disease of joints, Med. 37
—— fluid, escape of, from knee- and elbow-joints, in relation to ossific process, Surg. 140
—— membranes, hyperæmia of, with villous hypertrophy, early changes in arthropathies, Baln. 24
Synovitis, auscultation in, joints silent during effusion, noisy after absorption of fluid, Med. 37
Syphilide, case for diagnosis (E. G. Graham Little), Derm. 52, 53
——, corymbose, case of (A. Shillitoe), Derm. 152
——, relapse (case for diagnosis) (S. E. Dore), Derm. 70
—— (sycosiform), case of (G. N. Meachen), Derm. 105
Syphilis (acquired), alopecia of, in boy (J. H. Sequeira), Derm. 79
—— ——, primary, of ear (external), Otol. 10
—— —— ——, of ear (middle), Otol. 10
—— ——, secondary, of ear (external), Otol. 10
—— —— —— —— ——, treatment, Otol. 10
—— —— —— —— (middle), Otol. 11
—— ——, tertiary, of ear (external), Otol. 12
—— —— —— —— —— ——, manifestations, Otol. 12
—— —— —— —— (middle), Otol. 12
——, alopecia areata in subject of (J. H. Sequeira), Derm. 150
——, and granuloma inguinale, Derm. 136
——, and sporotrichosis, Derm. 114
——, cause of othæmatoma, Otol. 104, 106
—— —— perforation of soft palate, with unusual features (W. J. Horne), Laryng. 116
——, causing choroiditis, Odont. 31
—— —— iridocyclitis, Odont. 28, 29
—— —— iritis, Odont. 28, 29
——, chyliform ascites, dropsy, and albuminuria, case of (H. Batty Shaw), Clin. 112
—— (congenital), case treated by "606," Child. 155, 166
—— —— and adenoids, Otol. 19
—— —— and deaf-mutism, Otol. 3, 21
—— —— and multiple cartilaginous exostoses, Med. 3
—— ——, associated with acne scrofulosorum (H. G. Adamson), Derm. 18
—— ——, deaf-mutism due to, rarity, Otol. 3
—— ——, of ear (external) Otol. 2
—— —— —— (middle) Otol. 2
—— —— —— —— with nerve deafness, Otol. 21
—— —— ——, perceptive portion, Otol. 3
—— —— —— —— ——, see also *Deafness* (congenital, syphilitic)
——, general correspondence with trypanosomiasis, Path. 2
—— (hereditary) causing cicatricial obliteration of nasopharynx (Dan McKenzie) Laryng. 104
—— (inherited), and "blue sclerotics," case of (J. D. Rolleston) Child. 96
—— ——, case of, treated by Ehrlich-Hata's "606," Derm. 10
—— —— in children, splenomegaly of (F. P. Weber), Child. 62
—— in relation to mental deficiency, preliminary communication on (E. B. Smith and A. W. G. Woodforde), Child. 166
—— —— to otology (A. H. Cheatle), Otol. 1-24
——, macular atrophy in patient suffering from (H. G. Adamson), Derm. 122
—— not a factor in amaurotic idiocy (Tay-Sachs disease), Path. 168
——, otalgia with history of, examination of post-nasal space in, Otol. 23
——, otosclerosis not due to, Otol. 3, 21
——, primary sore of vestibule of nose and adjoining skin (N. Patterson), Laryng. 84
——, rarely invades nervous system, Path. 2, 3

Syphilis, reinfection with, after five and a half years (A. Manuel), Clin. 30
——, relation of von Jaksch's anæmia to, Child. 157
—— (secondary), case in which fading roseola simulated ichthyosis (H. G. Adamson), Derm. 33
—— (tertiary) of ear, perceptive portion, Otol. 14
—— —— —— —— —— ——, complications and manifestations, Otol. 14-18
—— —— of labyrinth (probably), Otol. 14
——, treatment by mercury, oral administration succeeding where injections failed, Clin. 21
——, see also *Leucoderma syphiliticum*
Syphilitic, delayed healing of wound after mastoid operation on, Otol. 13
—— of thirty years' standing, nerve deafness in (A. H. Cheatle), Otol. 26
—— showing rhythmical elevations of soft palate accompanied by rhythmical clickings in Eustachian tube and tympanum on left side, Otol. 13
Syphilitic affection of eighth nerve, case of (J. E. R. McDonagh), Derm. 147
—— auto-reinfection, case of (J. E. R. McDonagh), Derm. 129
—— disease (congenital) of auriculo-ventricular bundle, Clin. 41, 42
—— —— of nose treated with "606" (G. N. Biggs), Otol. 121
—— keloid in man (G. W. Dawson), Derm. 3
—— lesions, keloids forming on cicatrices resulting from (J. M. H. MacLeod), Derm. 74
—— nasal ulceration causing obstruction of Eustachian tube, Otol. 12, 13
—— onychia, rare form of, case of (J. E. R. McDonagh), Derm. 55
—— osteitis of femur, case of (P. Maynard Heath), Child. 103
—— stricture of rectum (so-called), presumed cause of, Obst. 73
Syringo-bulbia, case of (H. R. Prentice), Neur. 25
Syringomyelia, cases of, Neur. 27, 28
——, with symptoms precipitated by trauma (E. G. Fearnsides, for H. Head), Neurol. 34

Tabes dorsalis, with early rectal crises, case of (J. P. Lockhart Mummery and James Collier), Clin. 117
——, gastric crisis in ? case of division of posterior roots (C. M. Hinds Howell), Neurol. 48
—— —— crises of, treatment by division of posterior spinal nerve-roots, Surg. 210
——, internal ear deafness and tinnitus in, Otol. 17
—— (juvenile), case of (Wilfred Harris, for L. G. Guthrie), Neur. 50
——, treatment by diathermic current, Electr. 7
——, unilateral deafness occurring in, Otol. 61
——, with lumbo-sacral disease, case of (E. F. Buzzard), Clin. 15
von Tabora, experimental work on auriculo-ventricular bundle, Therap. 97, 98
Tachycardia, paroxysmal, Med. 90-92, 96
—— ——, classes of, Med. 92, 96, 97
—— ——, electrocardiogram showing, Med. 91
Tæniæ coli, Surg. 92
Tanchou, "Du froid et ses applications dans les maladies" (1824), Baln. 87
Tapioca as food for children, first mention, Child. 126
TARGETT, J. H.—Discussion on chronic endometritis, Obst. 51
——, discussion on hydatid of uterus, Obst. 247
—— —— on retroflexion of gravid uterus with superimposed fibroid, Obst. 290
——, report on microscopic examination of hydatid of the uterus (A. E. Giles), Obst. 246
Tarrance, case of rupture of tympanic membrane, quoted, Otol. 94
Taste, disturbance of, following injury to chorda tympani nerve, Otol. 84
——, loss of, resulting from damage to chorda tympani, Otol. 93
——, perversion of, in case of internal ear deafness, Otol. 17
TATE, W. W. H.—Discussion on an ovarian tumour obstructing delivery, Obst. 257

Tawalha Mukhi (Flame's mouth), medicinal spring in Kangra District of Punjab, Baln. 117
Tawara, auriculo-ventricular bundle, quoted, Therap. 97
Tay, Waren, discovery of amaurotic idiocy by ; published under title, "Symmetrical changes in the region of the yellow spot in each eye of an infant," Path. 167
Tay-Sachs disease, see *Idiocy* (amaurotic)
Tayler, Herbert P.—Case of Colles's fracture, Electr. 72
Taylor, C. H. S., and Rolleston, H. D.—Mollities ossium of forty-five years' duration, Clin. 97
Taylor, Gordon.—Case of infection of the cavernous sinus due to oral sepsis, Odont. 113-118
Taylor, James.—Case of unilateral paralysis of ninth, tenth and eleventh cranial nerves (shown by P. W. Saunders), Neur. 51
——, discussion on case of pseudo-bulbar paralysis of doubtful origin, Neur. 25
—— —— —— of toxic neuritis of doubtful nature, Neur. 23
Tea, effect on low blood-pressure, Med. 62, 63, 77, 78
——, excessive consumption of, factor in causation of arterio-sclerosis, Med. 63
Teacher, specimen of very young tubal ovum, quoted, Obst. 320
Teats (ulcerated, in cows), Epid. 81
—— —— ——, association of outbreaks of milk-borne septic conditions with, Epid. 88
—— —— ——, infection from, through milk, causing human disease, Epid. 82
—— —— ——, isolation of diphtheria bacillus from, Epid. 82, 83
Teeth, caries of, see *Caries* (dental)
——, detergent action of bread on, experiments showing, Odont. 92, 93
——, effect of acid fermentation of bread on, Odont. 93, 94
—— —— of weak acid on, prevented by saliva, Odont. 95
—— extraction (painless) under hypnotism, Anæsth. 14, 19, 20
—— (filling of), communications as to, invited, Odont. 2
——, Hutchinsonian type of, in inherited syphilis, Child. 67
——, lime-salts in bread not harmful to, Odont. 97, 98
——, lodgment of fermentable, non-detergent carbohydrates in contact with, undisturbed by saliva, main source of caries, Odont. 97
——, mucous coating on, protective action, Odont. 94, 95, 96
——, phosphates not harmful to, Odont. 97
——, relation between the teeth and certain diseases of the skin and mucous membrane (W. K. Sibley), Odont. 71-84
—— —— of dental trouble to acne, Odont. 75
—— —— —— —— —— rosacea, Odont. 76
—— —— —— —— to alopecia areata, Odont. 74
—— —— —— —— to eczema, Odont. 76
—— —— —— —— to epithelioma of oral mucous membrane, Odont. 78
—— —— —— —— to erythema, Odont. 77
—— —— —— —— to leucoplakia buccalis, Odont. 79
—— —— —— —— to lichen planus, Odont. 79
—— —— —— —— to localized œdema, Odont. 77
—— —— —— —— to seborrhœa, Odont. 74
—— —— —— —— to ulceration of oral mucous membrane, Odont. 78
——, removal of plaques and masses of bacteria on and about, to prevent dental caries, Odont. 94, 101
——, skiagraphy of, Electr. 90
—— (supernumerary, multiple), preceding central incisor, case of (A. A. H. Johnson), Odont. 42
—— —— —— —— —— —— ——, treatment discussed and suggested, Odont. 47-50
—— —— ——, regarded as examples of atavism, Odont. 46, 47
Teething formerly regarded as important feature in early life, Child. 120

Teething, weaning formerly not undertaken till after completion of, Child. 119

Telangiectases, multiple, cutaneous, of recent origin (J. Galloway), Clin. 42

Telegraphy, wireless, rotary converters for, Electr. 83

Telephone, use of, injuring membrana tympani indirectly, Otol. 84

Temperatures needed to destroy pathogenic micro-organisms, Epid. 135

Temporal bone, compound fracture of, treatment, Otol, 101

—— ——, fracture of, specimens illustrating (G. J. Jenkins). Otol. 107

—— —— (right), healed fracture of, showing line of fracture in meatus (A. H. Cheatle), Otol. 26

—— ——, specimen of, from case of thrombosis of cavernous sinus (H. J. Davis), Otol. 27

—— ——, specimens of, showing types of fractures (A. H. Cheatle), Otol. 107

Temporo-sphenoidal (left) and frontal lobes, abscesses in, in case of chronic suppurative mastoiditis (P. Watson-Williams), Otol. 134

Tenby, health resort suitable for foreign invalids, Baln. 20

Tenderness, local, fixed idea of, hysterical symptoms, Neur. 8, 9

Tendon-sheaths, inflamed, friction-sounds heard over, by auscultation, Med. 38

Teratoma, ovarian (J. E. Gemmell), Obst. 285

Terebene, see *Menthol*, eucalyptol and terebene

Terrier, dangers of scopolamine, quoted, Anæsth. 44

—— and Péraire, injection of morphine and atropine before chloroform inhalation, quoted, Anæsth. 44

Terror, production of hypnoid state through, Neur. 13

Testicle, connexion with gout, presumed, Baln. 36

Test-meal, in control of effect of belladonna on acid-values of stomach contents, Med. 25

Tetanus, death from, following attempt to extract foreign body from ear, Otol. 82

Tetany, chronic (recurrent) (G. A. Sutherland), Child. 17

—— —— ——, dilatation of colon and putrefying fæces associated with, Child. 17

Teter, Dr., anæsthesia by nitrous oxide and oxygen, quoted, Anæsth. 3, 11

Thalasso-therapy, see *Health resorts, Marine*, British (Marine), German (Marine)

Therapeutics (physical), applications of, Baln. 70

—— ——, use of water and waters most important branch of, Baln. 75

——, plea for the study of; Presidential Address (A. R. Cushny, F.R.S.), Therap. 1-12

——, scepticism in, prevailing in middle nineteenth century, Baln. 84

Theromorphism defined, Path. 306

Thermal effects produced by high-frequency currents (F. Nagelschmidt), Electr. 1-8

Thermes, Dr., hypodermic administration of waters, quoted, Baln. 108

THIELE, F. H.—Discussion on indications for and technique of Cæsarean section and its alternatives, Obst. 191

Thigh (left), congenital nævoid condition of (W. H. Bowen), Child. 193

Thilenius, indication list for stay at marine health resorts, issued by, Baln. 17

Thomas and Hall, case of division of posterior spinal nerve-roots for gastric crises of tabes, Surg. 212

Thomas, H. O., abduction frame, modified, in fixation of lint after fractures about hip-joint, Surg. 14, 15

——, device for reducing lateral displacement of foot, contrived by, Surg. 21

——, forcible flexion in treatment of old dislocation of elbow, quoted, Surg. 10

—— knee-splint, Surg. 14, 15, 16

—— use of twisted metal splints for Colles's fracture by, Surg. 25

THOMAS, L. K.—Discussion on nasal reflex during anæsthesia, Anæsth. 29

THOMSON, F. G.—Discussion on balneological treatment of neurasthenia, Baln. 44

THOMSON, G.—Discussion on inflammatory eye conditions due to oral sepsis, Odont. 35

——, discussion on malocclusion of permanent molars, Odont. 112

—— —— on possible cause of facial asymmetry, Odont. 108

Thomson, H. Alexis, fibromatosis of stomach, quoted, Surg. 240

THOMSON, STCLAIR.—Cases illustrating the treatment of frontal sinus suppuration, Laryng. 123
——, complete stenosis of the larynx from epithelioma in a man, aged 23, Laryng. 119, 121
——, discussion on case for diagnosis, Laryng. 14, 15
—— —— on case of fungating carcinoma of œsophagus treated by radium, Laryng. 9
—— —— —— illustrating "dents de scie" of vocal cords, Laryng. 10
—— —— —— of leprosy, Laryng. 26
—— —— on cases illustrating results of removing nodules from cords by means of galvano-cautery (by indirect method), Laryng. 41
—— —— on empyema of antrum in man, aged 71, now malignant (sarcoma), Laryng. 30
—— —— on functional aphonia in a boy, aged 15, Laryng. 115
—— —— on new type of œsophago-gastroscope, Laryng. 73
—— —— on tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 112
—— —— on treatment of frontal sinus discharge, Laryng. 140
——, relationship of diseases of the nose and accessory sinuses to affections of the eye and orbit, Laryng. 161-163
——, scleroma of the nasopharynx in a Polish woman, Laryng. 7
THOMSON, THEODORE, C.M.G.—Discussion on diseases of the cow affecting milk, Epid. 99
——, discussion on notification of disease and epidemiology of poliomyelitis, Epid. 222
——, discussion on paratyphoid fever and "food-poisoning," Epid. 63
—— ——, on the problem of disposition in pulmonary tuberculosis, Epid. 178
—— —— on typhoid carriers and contact infection, Epid. 133, 143
——, first report on typhoid fever prevalence at Folkestone, quoted, Epid. 115
——, the enteric fever carrier, Presidential Address, Epidemiological Section, Epid. 1-26
Thoracic duct, fistula of, absorption of fats in rectal feeding in case of, Therap. 66
Thorax, auscultation of, knowledge of meaning of sounds heard in, how built up, Med. 36
THORNE, W. BEZLY.—Discussion on ergot; a clinical experimental study, Therap. 150
——, discussion on history of infant-feeding from Elizabethan times, Child. 141
—— —— on low blood-pressure, Med. 78
—— —— on Dr. Milne's paper, "Measles: its treatment and prevention," Child. 177, 184
—— —— on use and action of ergot, Therap. 150
Thrombosis following hysterectomy, Obst. 224
—— of cavernous sinus, case of, specimen of temporal bone from (H. J. Davis), Otol. 27
—— —— —— due to oral sepsis (G. Taylor), Odont. 113-118
—— —— —— due to suppuration in nose or ear, Laryng. 162
—— of lateral sinus following acute otitis media, operation recovery (W. J. Chichele Nourse), Otol. 56
—— —— —— (streptococcal), with early pulmonary metastasia, recovery (S. Scott), Otol. 30
—— (mural), diagnosis, Otol. 33, 34
Thrombus (polypoid), rarity of, Child. 201
—— —— —— (infective), in pulmonary artery and extending into right ventricle, mistaken for patent ductus arteriosus (T. R. Whipham), Child. 199
Thucydides, recovery of victims of plague of Athens after loss of extremities, recorded by, Child. 13
Thudichum, reason for high percentage of insane dying with gall-stones, Path. 91
Thyroid gland, administration in case of complete alopecia, Path. 301, 303
—— —— and chronic rheumatism, Baln. 27
—— ——, carcinoma of, with extensive involvement of lymphatic glands, operation three and a half years ago, no recurrence (Wilfred Trotter), Clin. 118
—— ——, deficiency, relationship of hairlessness (atrichosis) to, discussed, Path. 299, 300
—— ——, excision of one of lobes and isthmus of, in treatment of case of Graves's disease (C. H. Pring and A. Evans), Clin. 124
—— —— extract, administration to dachshund, Path. 299
—— —— —— (dry thyroideum siccum), action of, Path. 233-236
—— —— ——, feeding of rats with, producing exophthalmos in, Path. 235, 236

Thyroid gland, extract, forms of obesity benefited by, Path. 299
—— —— ——, (fresh) in congenital atrichosis in girl, Path. 301, 302
—— —— ——, hyper-medication of, in exophthalmic goitre, Clin. 124
—— —— —— in diseases of skin, Derm. 72
—— —— —— in ichthyosis in child (T. Colcott Fox), Derm. 72
—— —— —— in rheumatoid arthritis, Child. 35, 36
—— ——, functions of, disturbed, acromegaly and gigantism associated with, Odont. 26
—— —— hæmorrhage into, causing urgent dyspnœa and œdema of glottis in woman, aged 50 (Arthur Evans), Clin. 25
—— ——, inadequacy (juvenile myxœdema), associated with infantilism, case of (H. M. Fletcher), Child. 96
—— ——, insufficiency, relation of symmetrical lipomatosis to, Clin. 7
—— —— (lingual), case of (G. W. Badgerow), Laryng. 81
—— —— not palpable in case of lingual thyroid (W. G. Spencer), Laryng. 13
—— —— of dachshund, Path. 299, 300
—— —— of featherless fowl, Path. 301
—— —— of hairless rats, Path. 301
—— —— preparations, pro- and anti-, action of (W. Edmunds), Path. 233-236
—— ——, tissue in ovarian embryoma (R. Drummond Maxwell), Obst. 217
—— ——, tumour of, two unusual cases (R. J. Godlee), Clin. 119
—— —— ——, unobserved, dissemination of, Clin. 125
Thyroidism, acute, symptoms of, following removal of thyro-lingual cyst (H. J. Davis), Laryng. 27
Thyro-lingual cyst, removal from girl, operation followed by symptoms of acute thyroidism (H. J. Davis), Laryng. 27
Tibia, avulsion of tubercle of, Surg. 17, 18
—— —— ——, value of hinged knee-cage, Surg. 18
——, cyst (benign) of (H. A. Lediard), Clin. 132
—— —— ——, radiography, Clin. 133, 139
—— —— ——, treatment of case, Clin. 134
——, fracture of internal tuberosity of, resulting in genu valgum, Surg. 17
—— —— —— ——, treatment, Surg. 17
—— —— of spine of, Surg. 44
—— ——, long-standing, Surg. 17
——, necrosis of, specimens, Child. 202
——, split fracture of the lower end of, diagnosis and treatment, Surg. 18, 19
—— —— —— —— ——, in conjunction with fracture of the astragalus, Surg. 18
—— —— —— of lower end of, in conjunction with Potts's fracture, Surg. 18
——, suppurative periostitis of (acute), specimen showing, Child. 202
——, tubercle of, injury to, Surg. 44, 45
—— and femur from case of infantile scurvy, Child. 204
Tic douloureux, case of, illustrating anæsthesia produced by alcohol injection of superior maxillary and supra-orbital nerves (W. Harris), Neur. 52
Ticehurst, percentage incidence of gall-stones based upon post-mortem records of Guy's Hospital, recorded by, Path. 88, 89
Tidal current, induced, intragastric application, Electr. 37
TILLEY, HERBERT.—Discussion on case of endothelioma of palate, with metastatic growth in neck, treated by radium, Laryng. 95
——, discussion on complete stenosis of larynx from epithelioma in man, aged 33, Laryng. 121
—— ——, on empyema of maxillary antrum in boy, Laryng. 32
—— —— on large nasopharyngeal polypus removed from boy, Laryng. 33
—— —— on new type of œsophago-gastroscope, Laryng. 73
—— —— on report on fatal termination of case of deafness, Otol. 68
—— —— on stricture of lower end of œsophagus, Laryng. 38
—— —— on tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 110

TILLEY, HERBERT.—Discussion on treatment of frontal sinus discharge, Laryng. 138
——, discussion on Dr. Watson-Williams's osteo-plastic frontal sinus operations, Laryng. 157
—— improved form of Hay's pharyngoscope, Laryng. 108
—— —— instrument for more efficient removal of nasal spine in submucous resection of septum, Laryng. 25
——, plaster casts of unusual malformations of upper jaw, Laryng. 75
——, portion of rabbit-bone removed from right bronchus, wherein it had been impacted over three years, and caused symptoms of bronchiectasis, Laryng. 96
——, report on specimen of growth from nose by Morbid Growths Committee, Laryng. 18
——, series of foreign bodies removed by direct bronchoscopy and œsophagoscopy, and skiagrams relating to above, Laryng. 151
——, specimen of a pedunculated growth on an elongated uvula, Laryng. 107
——, specimens illustrating the removal of foreign bodies from the air-passages and the œsophagus, Clin. 152
——, tooth removed from floor of right antrum in patient whose corresponding nasal cavity was obstructed by nasal polypus, Laryng. 98
Tillmanns's family of multiple cartilaginous exostoses, Med. 4
Time, important factor in treatment of neurasthenia, Baln. 37
Tinea barbæ, "eczematoid" ringworm of fungus associated with, cultures of violet endothrix trichophyton from case of (H. G. Adamson), Derm. 126
Tin-miners, prevalence of phthisis among, how explained, Child. 218
Tinnitus following injury to head, Otol. 92
——, surgical treatment possible, Otol. 103
—— and deafness in early case of general paralysis, Otol. 18
—— and internal ear deafness in tabes, Otol. 17
TIRARD, N.—Discussion on after-history of albuminuria in adolescence, Med. 136
——, discussion on value of rectal feeding, Therap. 74
Tissues, evolution of, Lamarckian theory inadequate to explain, Path. 306
——, localization of and effects of diathermic currents in, Electr. 2, 3
——, metaplasia (transformation) of, two aspects of, Path. 306
——, powers of reaction possessed by, Baln. 92, 110; see also *Reaction*
Toast preferable to untoasted bread, Odont. 97
Tobacco-smoking as cause of arterio-sclerosis, question discussed, Med. 64, 65, 77
——, effect of on low blood-pressure, Med. 64, 65, 77
Tobacco-poisoning, subnormal blood-pressure due to, Med. 73
TOD, H.—Discussion on eight cases of mastoid operation: (Case I), Otol. 125; (Case III), Otol. 129; (Case V), Otol. 131
Todd, G., and Dean, G., isolation of diphtheria bacillus from ulcerated teats, quoted, Epid. 82, 85
Toe-nails, ringworm of, case (A. Shillitoe), Derm. 151
Tomes, C. S., F.R.S., hypertrophy of gums and alveolar processes, quoted, Odont. 23
Tongue, chronic glossitis ? cause (P. Watson-Williams), Laryng. 158
——, malignant disease of, two cases (P. R. W. de Santi), Laryng. 113, 114
——, nævus of, removal under application of diathermic current, Electr. 8
Tongue-depressor, with Geffer's gag, in operating for cleft palate, Surg. 174, 177
Tonsil, carcinoma (primary) of, case of (E. A. Peters), Laryng. 79
——, cyst of, microscopical section (W. H. Kelson), Laryng. 118
—— (left), ulceration on, case of (S. Hastings), Laryng. 74
——, sloughing; fatal hæmorrhage from internal carotid, Child. 204
Tonsillitis, tuberculous, case of (F. Rose), Laryng. 67
Tonsillolith in left supra-tonsillar fossa (C. A. Parker), Laryng. 12
——, specimen of (C. A. Parker), Laryng. 33
Tonsils, both, sarcomatous growth of, with multiple sarcomatoma in and beneath the skin (William Hill), Laryng. 57
——, enlarged, removal under application of diathermic current, Electr. 8

Tonsils and pharynx, extensive malignant ulceration of (L. H. Pegler), Laryng. 78
——, portals of entry of tuberculous infection, Child. 223, 227
——, tuberculosis of, in adults, Laryng. 68
—— —— of, in children, Laryng. 68
—— —— (bilateral), with tuberculous laryngitis (C. Graham), Laryng. 67
Tophi, true composition of, Baln. 126
"Tops and bottoms," early patent food for children, Child. 135
Tornwaldt's bursa, Laryng. 53, 54
—— disease, Laryng. 53
Torquay, health resort suitable for foreign invalids, Baln. 20
Torus palatinus, three cases of (W. de C. Prideaux), Odont. 39
Toulouse, University of, Chair of Medical Hydrology at, Baln. 113
Toxæmia (intestinal), temporary deafness and blindness due to (A. L. Whitehead), Otol. 121
Toxins cause of food-poisoning following eating of canned meats, Epid. 65, 66
Trachea, foreign bodies in, skiagraphy of, Electr. 91
Tracheo-bronchial glands, infection with tuberculosis by way of alimentary tract, Child. 221
Tracheo-laryngostomy for stenosis following tracheo-laryngotomy (W. Hill), Laryng. 109
Tragus, destruction of, in tertiary syphilis of external ear, Otol. 12
Trästena (Sweden), outbreak of poliomyletis in, due to school infection, Epid. 204, 205
Trauma precipitating symptoms in case of syringomyelia (E. G. Fearnsides, for H. Head), Neur. 34
—— (psychical) hysteria defined as, Neur. 10
Traumatism, cyst of jaw resulting from (H. A. T. Fairbank), Odont. 14
Treadgold, case of chloroma, quoted, Path. 68
Trélat's lance coudée in operating on cleft palate, Surg. 174
—— rugines in operating on cleft palate, Surg. 174
Trendelenberg, experimental work on auriculo-ventricular bundle, Therap. 98
Treves, epidemic of poliomyelitis at Upminster (1908), Epid. 210
Treves, Sir F., Bt., delimitation of rectum, quoted, Surg. 86
——, on pelvic colon, quoted, Surg. 86
TREWBY J. F.—A new inhaler for administering nitrous oxide gas by the nasal method, Anæsth. 12
Trichloracetic acid, growth of bacteria on, Path. 108
Trichophyton roseum and *ectothrix;* specimen of trichophytic invasion (A. Whitfield), Derm. 93
Trichosporum giganteum, Derm. 149
Troller, observations on reduction of gastric juice, quoted, Med. 24
Tropa-cocaine, Therap. 56
Trophic symptoms of hysteria, Neur. 17
Trophœdema, hysterical and functional (E. G. Fearnsides), Derm. 84
TROTTER, WILFRED.—Carcinoma of thyroid, with extensive involvement of lymphatic glands; operation three and a half years ago; no recurrence, Clin. 118
——, discussion on report of fatal termination of case of deafness, Otol. 68
Trypanosoma Brucei, Path. 7
—— *equiperdum* and dourine, or "mal de coit," Path. 2, 11
—— *gambiense*, history of discovery of, Path. 4, 5
—— ——, invasion of subarachnoid space, Path. 6, 7
—— ——, life-history in fly (*Glossina palpalis*), Path. 7-12
Trypanosome and spirochæte infections, comparative neuro-pathology (F. W. Mott. F.R.S.), Path. 1
Trypanosomiasis, general correspondence with syphilis, Path. 2
——, invasion of nervous system, Path. 2, 3
—— (human), ætiology. Path. 4-12
—— —— causal factors, sketch of discovery of, Path. 3, 4
—— ——, cerebral form of disease, Path. 15, 16

Trypanosomiasis (human), changes in central canal of spinal cord, Path. 30
—— —— —— in neural elements, Path. 31, 32
—— —— —— in small vessels and capillaries, Path. 30, 31
—— ——, clinical study of, Path. 12-39
—— ——, coitus as means of infection, Path. 11, 12
—— ——, curability, Path. 7, 17
—— ——, description of, Path. 13-18
—— ——, diagnostic importance of œdema and gland enlargement, Path. 13, 14, 18
—— ——, distribution of glia proliferation in, Path. 28, 29
—— ——, examination of nervous tissues of animals experimentally infected by trypanosomes, Path. 34-39
—— ——, examination of neuroglia cells in, Path. 24-28
—— ——, histological changes in the central nervous system, in, Path. 22-28, 35
—— —— —— —— in lymphatic glands, Path. 18-20
—— ——, infective sources other than *Glossina palpalis*, Path. 10-12
—— ——, invasion of subarachnoid space by organism, Path. 6, 17
—— ——, local infection followed by generalized blood and lymph infection, first stage, Path. 13, 14
—— ——, lowered resistance to microbial invasion, Path. 15
—— ——, microscopic examination of nerve-cells and fibres, Path. 32, 83
—— ——, mistaken at first for malaria, Path. 4, 13
—— ——, morbid changes in lymphatic structures, Path. 20, 21
—— ——, part played by carrier of disease (*Glossina palpalis*), Path. 7-12
—— ——, perivascularitis cause of lethargy in, Path. 34-37
—— ——, possible effects of arsenic compounds in treatment of, Path. 16, 17
—— ——, reappearance possibly due to latent intracellular forms, Path. 6, 7
—— ——, reservoir of infection, Path. 9, 10
—— ——, résumé of knowledge of (F. W. Mott, F.R.S.), Path. 1, 12
—— ——, sleeping sickness, lethargy, second stage, Path. 14-18
—— ——, sudden death in cases treated with arsenic, Path. 17
—— ——, vascular hæmorrhages in, Path. 31
Tryptophane, effects of, when administered by mouth, Baln. 55
—— —— —— by small intestine, experimentally, Baln. 56
TUBBY, A. H.—Asymmetry of the pelvis (Naegele), partial congenital suppression of left lateral wing of the sacrum, scoliosis, Child. 163
——, double congenital club-band of the radio-palmar variety, with absence of radius on both sides, Child. 164
Tube-holder, Electr. 86, 87
Tube-stand, adjuncts of, Electr. 86
——, component parts of, Electr. 85, 86
——, description of, Electr. 85
—— (efficient), essentials of, Electr. 85
—— (new), demonstration of (S. Melville), Electr. 31
——, rigidity of, essential, Electr. 85
——, universal safety and protective, for all kinds of fluoroscopy and radioscopy (Dr. Schmidt), Electr. 71
Tubercle of tibia, avulsion of, Surg. 17, 18
—— ——, injury to, Surg. 44, 45
Tuberculide (non-ulcerating) (?) Boeck's miliary sarcoid, following lupus vulgaris (J. H. Sequeira), Derm. 67
Tuberculin in pityriasis rubra pilaris, Derm. 24
—— test in diagnosis of tuberculosis, Epid. 32, 33
Tuberculosis, anaphylaxis in, question of, Child. 223
—— and sporotrichosis, Derm. 114
—— associated with symmetrical angiomatous swellings (J. H. Sequeira), Derm. 28

Tuberculosis (bovine), infection of children with, percentage, Child. 233, 234

—— (bovine and human), relative importance discussed, Child. 231

—— (bovine), infectivity proved experimentally, Epid. 49

—— —— —— ——, dosage more important factor than susceptibility, Epid. 49

—— ——, intimate relation to that in man, Epid. 46

—— ——, milk-borne, Epid. 88

——, carriers of, Epid. 33, 34, 44

—— in children, infection by alimentary tract, Child. 231

—— (chronic) of nose, larynx, and lungs, case of (C. A. Parker), Laryng. 64

—— (congenital), Child. 227

——, diagnosis by tuberculin test, Epid. 32, 33

——, experimental production by inoculation of material from erythema induratum and lichen scrofulosorum, Derm. 49

—— (generalized), proportion of deaths from, Child. 231

—— infection of infants and young children from subjects of, Child. 222, 223

—— —— by intestines more difficult than by lungs, Child. 216, 217

—— —— by tuberculous milk through alimentary tract, Child. 220, 221

—— (laryngeal), case of ? cured (C. Horsford), Laryng. 43

—— ——, in boy, aged 10 (W. J. Horne), Laryng. 116

—— ——, probable in case of pulmonary tuberculosis (case for diagnosis), Laryng. 15, 16

—— ——, treatment by diathermic current, Electr. 6

—— (latent), Epid. 34

—— ——, lymphatic glands, seat of infection in, Epid. 34

——, lupus starting point of, Child. 227

——, macular atrophy associated with, Derm. 123

—— of nose and cheek, case of (W. Hill), Laryng. 85

—— probable in case of laryngeal disease, presenting appearances of epithelioma (W. J. Horne), Laryng. 117

—— (pulmonary), action of aconite on pulse-rate in cases showing, Therap. 163, 178-183

—— ——, aspiration of tubercle bacillus not a general cause of, Child. 214

—— ——, asylum statistics, Epid. 180-182

—— ——, carriers of, Epid. 35

—— ——, comparative absence among coal-miners, how explained, Child. 219

—— ——, contagious nature of, early ideas of, Epid. 161

—— —— —— ——, nineteenth century investigations, Epid. 166

—— —— —— —— —— —— views, Epid. 162-166

—— —— —— ——, sixteenth and seventeenth century views of, Epid. 161, 162

—— ——, danger of hæmoptysis in middle-aged subjects of, at high altitudes, Med. 60, 76

—— ——, death-rate, diminution in, Epid. 27, 170, 171

—— ——, detection in early stages by examination of contacts, Epid. 30, 31, 38

—— ——, disposition to, definition, Epid, 151

—— —— ——, theory of, as found in great medical classics, Epid. 151-153

—— —— —— —— —— in medical writings of seventeenth and eighteenth centuries, Epid. 153-155

—— —— —— —— —— —— —— of nineteenth century, Epid. 156-160

—— ——, educational propaganda in, effect of, Epid. 29

—— ——, family predisposition to, Epid. 37, 47, 48

—— —— —— —— and family infection, Epid. 49

—— —— —— ——, influence of, Epid. 37, 44, 45

—— —— —— ——, mortality records of Pearl Life Assurance Company in relation to, Epid. 37

—— ——, frequency among grinders, tin-miners, and gold-miners, how explained, Child. 218

—— ——, growth of the doctrine of tubercle in nineteenth century, Epid. 156

—— ——, heredity in, Epid. 147, 184

—— ——, hygienic measures to counteract predisposition to, in children, Epid. 41, 42

Tuberculosis (pulmonary), infection by alimentary tract, Child. 205, 221, 229, 231
—— —— —— —— —— ——, experiments to prove, Child. 205, 229, 230
—— —— —— and disposition to, bacteriological and pathologico-anatomical evidence, Epid. 167, 168
—— —— —— ——, clinical evidence, Epid. 172-174
—— —— —— ——, epidemiological and statistical evidence, Epid. 168-172
—— —— ——, homes of poor chief centre of, Epid. 35
—— —— ——, immunity from, Epid. 35
—— —— —— by inhalation, Child. 228, 229
—— —— —— —— of dust, Child. 228
—— —— ——, marital, Epid. 45, 47
—— —— —— ——, co-efficient of correlation for existence of, values, Epid. 45
—— —— —— of members of staff of Mount Vernon Hospital, proportion shown, Epid. 36
—— —— —— promoted through inhalation of sharp spicules in dust, Child. 218
—— —— ——, tubercle bacillus in sputum as guide to, Epid. 46, 47, 48
—— —— —— with, of "contacts" with tuberculous patients, Epid. 35-37, 44, 46, 47
—— ——, introduction into country villages, Epid. 42
—— ——, local persistence, Epid. 42
—— ——, morbidity in localities exposed to strong prevalent rain-bearing winds, Baln. 17
—— ——, mortality from, diminution in, Epid. 27
—— ——, notification, compulsory, Epid. 29, 46
—— —— ——, universal, sole means of obtaining knowledge of incidence and case-mortality, Epid. 28
—— ——, prevalence of, Epid. 28
—— —— ——, due to malnutrition, Epid. 48
—— ——, prevention of (J. E. Squire), Epid. 27-50
—— —— —— by disinfection, Epid. 42
—— —— —— by home visitation to examine "contacts," Epid. 31, 38, 39
—— —— —— —— —— —— ——, agency of anti-tuberculosis dispensaries in, Epid. 39, 40
—— —— —— agency of medical inspection of school-children in, Epid. 40, 41
—— ——, probably due to pen infection, Child. 224
—— ——, problem of, considered from standpoint of disposition (W. Bulloch and Major Greenwood, jun.), Epid. 147-184
—— ——, rarity in mitral disease, Child. 226
—— ——, segregation of advanced cases, to prevent excessive dosage of infection, Epid. 49
—— ——, supervening on bronchitis in middle and old age, Epid. 35
—— ——, susceptibility to, due to acquired conditions, Epid. 48
—— ——, transmitted phthisical habitus, theory of, Epid. 172
—— ——, treatment by sanatoria, Epid. 29
—— —— and lichen scrofulosorum with atrophodermia striata et maculata (H. G. Adamson), Derm. 1
—— of soft palate, pharynx, and larynx (W. G. Howarth), Laryng. 66
—— of urinary tract due to sexual intercourse, Child. 224
—— of vocal cords in young man, case of (L. H. Pegler), Laryng. 76
——, portals of entry of infection in (L. Cobbett), Child. 205-234
——, relative susceptibility of various tissues and organs to, Child. 208
——, see also *Cæcum*, tuberculosis of ; *Colon*, tuberculosis of ; *Sigmoid*, tuberculosis of
Tuberculosis Commission, findings as to portals of entry in phthisis, Child. 217
Tuberculous disease of bones, joints, and glands, marine climate indicated for, Baln. 17
—— —— of hips and knee followed by osteo-arthritis of same joints, Baln. 24
—— —— of neck glands, production through pediculi of scalp, Child. 223, 224, 232
—— invasion of gastric carcinoma, Child. 226
—— laryngitis, with bilateral tuberculous disease of tonsils (C. Graham), Laryng. 67
—— nodules, disseminated, of skin (E. G. Graham Little), Derm. 48
—— tonsillitis, case of (F. Rose), Laryng. 67

Tuberculous ulcer of ileum perforated (L. E. C. Norbury), Clin. 50
—— ulceration of larynx, case of (L. H. Pegler), Laryng. 78
Tübingen, University clinic for hydrotherapy at, Baln. 87
Tuckey, C. Lloyd.—Discussion on hypnotism in relation to surgical anæsthesia, Anæsth. 19
Tulloch, Lieutenant, fatal infection by *Trypanosoma gambiense*, Path. 10
Tumour on back, probably dermoid (O. L. Addison), Child. 165
Tumour-cells, karyorrhexis of, in case of atypical chloroma, Path. 63, 64, 66
Tumours, uterine, in rabbits (A. E. Boycott), Path. 225-232
Turban on heredity in phthisis, quoted, Epid. 172
Turbinal, right middle, cystic (P. Watson-Williams), Laryng. 159
Turbinectomy, anterior middle, in acute frontal sinusitis, Laryng. 128, 129, 147
Turin, University of, lectures on medical hydrology in, Baln. 115
Turnbull, H. H.—Therapeutic use of the digitalis group, Therap. 25-34, 37
Turner, A. Logan.—Discussion on empyema of maxillary antrum in boy, Laryng. 32
—— —— on lupus of larynx in woman, Laryng. 35
Turner, P. D., suppurating fibroid after labour removed by Doyen's operation, quoted, Obst. 191
Turney, H. G.—Discussion on case of symmetrical lipomatosis, Clin. 5
——, discussion on cases of adiposis dolorosa, Clin. 5
——, on the arthropathies and osteo-arthritis, quoted, Baln. 24
Turtle, G. de B.—Discussion on word-blindness and idioglossia, Child. 52
Tuson, delimitation of rectum, quoted, Surg. 86
Tweedie, Alex. R., and Keith, Arthur.—Ectopia of the pituitary, with other congenital anomalies of the nose, palate, and upper lip, Laryng. 47-52
Tweedy, E. Hastings.—Discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 182
——, practice as regards removal of appendix in pelvic operations, Obst. 353
——, unjustifiability of craniotomy, quoted, Obst. 165
Twin tubal pregnancy (S. J. Cameron), Obst. 149
Twort, F. W., differentiation of bacilli producing food-poisoning, quoted, Epid. 70
——, fermentation of lactose by *Bacillus typhosus*, quoted, Epid. 142
——, mutation of micro-organisms, quoted, Epid. 124, 125
Tylosis in woman (Sir M. Morris and S. E. Dore), Derm. 108
Tympanic cavity, spontaneous hæmorrhage into, how produced, Otol. 84
—— ——, syphilitic endarteritis in blood-vessels of mucous membrane of, Otol. 18
—— membrane, injury to, causing rupture of membrane, Otol. 83
—— —— ——, causing suppurative otitis media, Otol. 83
—— —— ——, indirect, causing rupture, Otol. 84
—— —— ——, wider extension, Otol. 84
—— ——, malingering in form of self-injury to, Otol. 84
—— ——, perforation, traumatic, diagnosis from that caused by disease, Otol. 83, 84
—— ——, pressure that can be withstood by, Otol. 94, 105
—— ——, rupture of, Otol. 93-95
—— —— ——, causes, Otol. 93
—— —— ——, traumatic following direct and indirect violence, Otol. 94
Tympanum, contents of, injury to, during operations, Otol. 85
——, injuries to, Otol. 87
——, new growth of unknown nature in (W. H. Kelson), Otol. 77
——, and Eustachian tube on left side, rhythmical clickings in, accompanying rhythmical elevations of soft palate in syphilitic, Otol. 13
Typhoid fever, "ambulatory," Epid. 132
—— ——, association of *Bacillus enteritidis* with, Epid. 61
—— ——, bony formations in and around joints following, Baln. 24
—— ——, carrier of, source of outbreak at Brentry Certified Inebriate Reformatory, Epid. 13, 14, 15

Typhoid fever, carrier of, source of continued outbreaks at Folkestone (1893-1909) through milk distribution, Epid. 16, 18, 137, 138
—— —— —— —— —— —— —— —— —— ——, probability discussed, Epid. 115-118, 137, 138, 145, 146
—— ——, carriers of, Epid. 1-26
—— —— ——, and gall-stone diathesis, Epid. 12, 19
—— —— ——, apparent excess among females, Epid. 11, 12
—— —— ——, auto-infection, Epid. 134, 141
—— —— ——, bacteriological examination of excreta and blood, Epid. 118-123
—— —— —— —— —— —— ——, conducted exhaustively, Epid. 122
—— —— ——, cases detailed, Epid. 17, 11
—— —— ——, condition not necessarily dependent on previous attack of fever, Epid. 5
—— —— ——, definition, Epid. 2
—— —— ——, difficulties in proof of infection by (W. H. Hamer), Epid. 105-146
—— —— ——, doubtful cases, Epid. 123
—— —— ——, epidemics presumed due to, Epid. 2, 107-123
—— —— —— —— —— ——, questioned and discussed, Epid. 107
—— —— ——, examination of fæces, frequency, Epid. 14, 15
—— —— ——, in asylums, Epid. 119, 120, 134
—— —— ——, infective agency, difficulty of proof (W. H. Hamer), Epid. 105-132
—— —— —— —— ——, minimized, Epid. 19
—— —— ——, infectivity, degree of, estimated, Epid. 22
—— —— —— ——, intermission of, Epid. 17, 18
—— —— ——, investigations at Strasburg, Epid. 12, 19, 107, 108
—— —— —— —— at Washington, Epid. 13
—— —— ——, method of testing, Epid. 135
—— —— ——, non-effective, Epid. 124
—— —— ——, presence of *Bacillus typhosus* in, demonstrated by Weber's method, Epid. 120
—— —— ——, proportion of, Epid. 2, 3
—— —— ——, report on, to Local Government Board by Dr. J. C. Ledingham, Epid. 107
—— —— ——, suspected persons proving not to be, Epid. 121
—— —— ——, urine most infective, Epid. 135
—— ——, case of, with muscular hæmorrhage, Surg. 50
—— ——, contact infection, Epid. 127, 139, 141
—— ——, convalescents from, *Bacillus paratyphosus* B not found in fæces of, Epid. 55
—— ——, epidemic at Lincoln (1905), cases reported subsequently as an indication of "carrier" transmission, Epid. 8, 9
—— —— —— ——, secondary infections, Epid. 132
—— —— —— at Maidstone (1897), cases reported subsequently as an indication of "carrier" transmission, Epid. 8, 9
—— —— —— —— ——, secondary infection, Epid. 132
—— —— —— at Worthing (1893), cases reported subsequently as an indication of "carrier" transmission, Epid. 8, 9
—— —— —— —— ——, secondary infections, Epid. 132
—— ——, epidemics and outbreaks, origin of, difficulty of tracing, Epid. 105, 106
—— —— followed by recurrent attacks of osteitis (H. D. Rolleston), Clin. 156
—— ——, hysterical sequela of, Neur. 8
—— ——, incubation period, length of, Epid. 130
—— —— in Elham Rural District, case-rate low, Epid. 117, 123
—— —— in England and Wales, decline in mortality, Epid. 25
—— ——, infective agency, difficulty of proof, military instance, Epid. 121, 122
—— ——, inoculation against, see *Serum*, antityphoid
—— ——, mortality less in children, Epid. 10
—— ——, outbreak at Kilworth Camp, Epid. 109
—— —— —— at Lister Institute, Epid. 136

Typhoid fever, outbreak in Shoreditch, Epid. 131
—— —— —— in Southwark, Epid. 130
—— —— —— at Winchester, Epid. 131
—— ——, outbreaks due to fried fish, Epid. 134, 135, 141, 144, 145
—— —— —— of, at Aberdeen, origin discussed, Epid. 111-115
—— —— —— —— presumed to be due to milk, Epid. 110-115
—— —— —— of, difficulty in proving origin (W. H. Hamer), Epid. 105, 106
—— —— and Malta fever, mixed infections of, Epid. 118
—— —— and paratyphoid fever, mixed infections of, Epid. 118
—— ——, paratyphoid fever clinically indistinguishable from, Epid. 63
—— ——, pneumonia associated with, followed by pulmonary tuberculosis, Epid. 48
—— ——, prevalence at Lincoln (1881-1909), Epid. 7
—— —— —— at Maidstone (1881-1909), Epid. 7
—— —— —— at Worthing (1881-1909), Epid. 7
—— —— —— of, relation to weather, Epid. 190
—— ——, prophylaxis, campaign in South-west Germany (1903), Epid. 2, 10, 12, 20
—— ——, secondary invaders in, Epid. 117
—— ——, sequelæ of, vary, Epid. 194
—— ——, sex-incidence greater among males, Epid. 11
—— ——, transmission of, theories as to, Epid. 1, 2
—— ——, treatment by baths, Baln. 93
——, paratyphoid, and allied organisms, strains of, complement fixation with, studies in (H. R. Dean), Path. 251-278
Typhus fever, prevalence (local) in Mexico, Epid. 225, 226
—— —— —— (seasonal), resembles that of poliomyelitis, Epid. 225
—— ——, transmission by lice, Epid. 225, 226
—— —— —— ——, experimental, Epid. 225
Tyramine, reaction of arteries to, Therap. 89
——, use of, Therap. 115, 116
—— ——, illustrative cases, Therap. 128-130
Tyson, W. J.—Discussion on calcareous (hard) drinking water, Baln. 127
——, discussion on after-history of albuminuria in adolescence, Med. 130

Udders (cows'), quarters of, atrophy due to previous acute mastitis, Epid. 77
—— —— ——, unaffected, causal organisms of mastitis present in, Epid. 78, 79
—— —— —— —— in mastitis, milk drawn from unsafe for consumption, Epid. 79
Uhlenhuth, search for *Bacillus suipestifer*, by, Epid. 58
Uhthoff, symptoms of pseudoglioma, quoted, Path. 248
Ulceration associated with lupus erythematosus, case showing (J. M. H. MacLeod), Derm. 53
—— (trophic) over large formation of scar-tissue, case of (E. M. Corner), Clin. 160
—— (unusual) of skin, two cases showing (H. Batty Shaw), Clin. 110
Ulcers (callous, obstinate) on legs, with sclerodermia, greatly improving under mercurial inunction (A. W. Williams), Derm. 13
—— (icteric) of legs in pernicious anæmia (H. Batty Shaw), Clin. 110
—— —— —— —— ——, blood examination of, Clin. 111
Ulrich, J.—Fifty-six cases of cleft palate treated by operation, with special reference to the various methods of operation and the after-results, Surg. 173
Ultzmann and Hoffmann, "Atlas der Harnsedimente," Path. 135
Umber, rectal feeding followed by secretion of gastric juice, Therap. 72
Uncleanly habits, factor in causation of cancer, Electr. 51
Underwood, M., recommendation of red wine for young children, by, Child. 123
——, scientific methods in artificial feeding of infants first recognized by, Child. 125, 126
Undulatory current produced by Hampson's apparatus, Baln. 35
United States, epidemics of poliomyelitis in, Epid. 200-203

United States, epidemics of poliomyelitis in, map showing Epid. 202

—— ——, origin and progress of hydrotherapy in, Baln. 90, 97

University Hydrotherapeutic Institution, opening of, at Berlin, Baln. 6

Upminster, epidemic of poliomyelitis (1908) at, Epid. 210

Uranium, see *Acetate* and uranium

Urea, presence of, in diabetic urine, inquiry as to (1811), Med. 152

——, reaction of arteries to, Therap. 90

Ureters, constriction of, in cancer of uterus, leading to hydronephrosis, Obst. 77

——, isolation of, in abdominal panhysterectomy by Wertheim's method, Obst. 89

——, skiagraphy of, Electr. 90, 91

Uric acid, estimation by Hopkins and Ludwig-Salkowski processes, Baln. 47

—— —— (endogenous), excretion during taking of sulphur water, effect of doses of hypoxanthin on, Baln. 50

—— —— excretion in rheumatoid arthritis, Baln. 59

—— —— (exogenous), excretion during taking of sulphur water, Baln. 51, 52

—— ——, output in gout, Baln. 59

—— ——, salts of, solubility of, Baln. 122

—— —— sediments, crystals forming uric acid calculi compared with, Path. 133-136

—— —— ——, formation of, secretion of urine supersaturated in uric acid necessary before crystallization can take place in, Path. 138

—— —— storm, clinical picture of, Baln. 57

—— —— see also *Calculi* (urinary, uric acid)

Urinary sediments, crystalline rosettes and spheres of, Path. 142

—— tract, tuberculosis of, due to sexual intercourse, Child. 224

Urine, bacilli isolated from, comparative gas- and acid-forming powers in media of various sugars, Path. 97, 98, 107

——, *Bacillus typhosus* in, Epid. 2, 135

—— —— *paratyphosus* B not found in, Epid. 55

—— (diabetic) presence of urea in, inquiry as to (1811), Med. 152

——, diacetic acid and acetone in, during rectal feeding, Therap. 74, 75

——, effect on, in treatment with strong sulphur water, Baln. 45-62

——, excretion of, before and after taking sulphur water, Baln. 49

——, movement of, influencing production and growth of calculi, Path. 141

——, nitrogenous contents after rectal feeding, Therap. 68-70

—— of typhoid carriers most infective, Epid. 135

——, supersaturated in uric acid, secretion of, necessary before crystallization can take place in uric acid calculi, Path. 138

——, suppression (acute), treatment of, by incision and drainage of kidneys (W. Harris and W. H. Clayton-Greene), Clin. 161

——, total nitrogen in, estimation by Kjeldahl's method, Baln. 47

——, variation in saturation, effect on striation of masses in compact uric acid calculi, Path. 141

Urticaria, treatment by calcium salts, Baln. 121

—— pigmentosa in adult ? case of (E. G. Graham Little), Derm. 104

—— —— of macular type in infant (E. G. Graham Little), Derm. 103

Utero-parietal suture preliminary to Cæsarean section, Obst. 202, 203, 209

Uterus, action of ergot on, method of investigation, Therap. 117, 118

——, cancer of, case of, with dissections of pelvis showing distribution of lymphatic glands, Obst. 74, 75

—— ——, complicated by hydronephrosis, due to stricture of ureters, Obst. 77, 102

—— ——, constriction of ureters in, leading to hydronephrosis, Obst. 77

—— ——, diagnosis, significance of bleeding after menopause, Obst. 86, 105, 107

—— ——, extirpation of inter-iliac lymphatic glands in, Obst. 74, 75

—— ——, health of patients fair till late stages, Obst. 88

—— ——, involvement of inguinal glands in, Obst. 75, 76

Uterus, cancer of, involvement of sacro-uterine ligaments in, exaggerated, Obst. 72, 73
—— ——, matting of tissues in advanced cases, Obst. 72, 73, 102
—— ——, metastasis, glandular or visceral, post-mortem statistics showing percentage of cases free from, Obst. 71
—— ——, treatment by total abdominal hysterectomy (Wertheim's method), cases recorded, duration of symptoms, Obst. 86
—— —— —— —— —— —— ——, freedom from recurrence after, Obst. 101
—— —— —— —— —— —— ——, previous confirmation of disease by microscopic examination, Obst. 88
—— —— ——, operative, extirpation, of danger zones, Obst. 71
—— —— —— ——, pathological bases for (A. Leitch), Obst. 69-78
——, cancer of body of, average duration of life after appearance of first symptoms, Obst. 76, 101, 103, 105
—— —— ——, extent of removal, Obst. 76
—— —— ——, in patient, aged 29 (H. R. Spencer), Obst. 308-312
—— (cervix), dilatation of, before operation, method of, Obst. 309, 311, 312
—— ——, examination of films from, Obst. 171, 180, 206, 207
—— ——, glands of, see also *Glands* (cervical)
—— ——, ligation of, preliminary to abdominal hysterectomy, Obst. 178
—— ——, micro-organisms in, Obst. 193
——, eventration of, Obst. 199, 209
——, fibroid tumour of, complicating pregnancy, after-history of case (A. H. N. Lewers), Obst. 307
—— —— ——, intramural, treatment by myomectomy, case referred to, Obst. 12
—— —— ——, treatment by panhysterectomy, Obst. 116
——, fibromyoma of, causing acute intestinal obstruction (J. Barrie), Obst. 108
——, fundal incision into, methods in abdominal panhysterectomy, Obst. 178
—— (gravid) ruptured through scar of Cæsarean section (S. J. Cameron), Obst. 147
——, hydatid of (A. E. Giles), Obst. 245
—— ——, microscopical examination, Obst. 246
——, labour-pains in, constitution of, Therap. 120, 121
—— ——, frequency and duration of, Therap. 121
——, measurements of, in passive and active contraction, Therap. 118-120
——, mucosa, changes in normal pregnancy, cause of (James Young), Obst. 301
—— ——, in menstruation and pregnancy ; action on chorionic cells and function of decidua (James Young), Obst. 291
——, myoma of, operations for preservation of ovaries, Obst. 117, 118, 129, 134, 144, 145
——, myxosarcoma of, supposed case, Obst. 112
—— and ovaries, adeno-carcinoma of, case (M. A. Scharlieb), Obst. 359
——, parturient, indication for use of ergot, Therap. 123
——, pathological specimens, exhibited (1909-10), summarized, Obst. 13
——, polypi of, undergoing malignant degeneration, cases of (Professor Schottländer, shown by H. Macnaughton-Jones), Obst. 31
——, polypus (fibromyomatous) of, in girl (G. Blacker), Obst. 63
—— (pregnant), myomata of, successful enucleation possible, Obst. 290
—— ——, with superimposed fibroid, retroflexion of (C. E. Purslow), Obst. 288
——, retroflexed, in chronic endometritis, results of treatment by curetting, Obst. 47
——, rupture of, treated by suture, clinical history summarized, Obst. 12
——, sarcoma of, in patient, aged 25 (D. Drew), Obst. 111
——, septic infection at childbirth, causes of, Obst. 194
—— ——, grave risk in Cæsarean section, Obst. 209
——, swabbings from, determination of nature of pathogenic micro-organisms on, Obst. 171
——, toneless, cases of, Therap. 123, 137
——, tracts of lymphatic vessels from, to pelvic glands contained within broad ligaments, Obst. 76

Uterus, tumours of, in rabbits (A. E. Boycott), Path. 225-232
—— ——, operations for preservation of endometrium in, Obst. 118, 139, 143, 145
Utriculoplasty compared with hysterectomy for uterine hæmorrhage, Obst. 276
—— for uterine hæmorrhage, six cases of, one followed by pregnancy and labour (V. Bonney), Obst. 270
——, maintenance of menstrual function, Obst. 274
Uvula, elongated, pedunculated growth on (H. Tilley), Laryng. 107

Vaccine, autogenous, administered by mouth in treatment of case of infective endocarditis (A. Latham and G. L. Hunt), Clin. 14-21
—— —— —— —— —— —— —— ——, reasons for choice of method, Clin. 21
——. injection of, in case of leprosy with involvement of external ear, palate, pharynx, &c., Otol. 120
—— in treatment of chronic dermatitis of pinna due to infection by *Staphylococcus aureus* (C. E. West), Otol. 38
—— —— —— —— —— —— —— —— ——, dosage, Otol. 39 (footnote)
—— —— of membranous colitis complicated by pyorrhœa alveolaris, Clin. 140
—— of pyorrhœa alveolaris, preparation of and treatment by, Odont. 81, 82
—— therapy, empirical treatment of infective diseases, example of (C. Riviere), Therap. 39
—— —— in puerperal sepsis, Obst. 194, 200
—— ——, principles of, advances on empirical treatment suggested by consideration of (C. Riviere), Therap. 43
——, see also *Bacillus coli* vaccine
Vaccines (autogenous) in chronic frontal sinus suppuration, Laryng. 130, 138, 139
—— (mixed), see *Micrococcus catarrhalis* and Pneumococcus
Vaccinia, outbreak in herd of cows, infecting milkers, Epid. 75, 99, 103
Vagal reflex, under chloroform asphyxia, Path. 207-212
—— ——, under ether asphyxia, Path. 213
Vagina, bacteriology during labour, Obst. 169
—— —— —— pregnancy, Obst. 169
——, cysts of, Obst. 22
——, see also *Adenomatosis vaginæ*
——, development of, bearing on, of obstructing membrane (W. Blair Bell), Obst. 234
——, disinfection before and after Cæsarean section, Obst. 188
——, gangrene of, probably due to chemical effect of pink rubber ring pessary (A. E. Johnson), Obst. 323
——, glands found in, Obst. 22
—— —— ——, see also *Adenomatosis vaginæ*
——, hairpins impacted in, probably causing suppurating ovarian cyst (H. J. Paterson), Obst. 259
——, involvement in cancer originating in cervix, Obst. 78
—— —— —— —— ——, leading to further involvement of bladder, Obst. 78, 89
—— —— —— —— —— —— —— —— of rectum, Obst. 78
——, micro-organisms in, Obst. 193
——, pathological specimens, exhibited (1909-10), summarized, Obst. 13
——, sterilization during abdominal panhysterectomy, Obst. 178
——, treatment of top of, in abdominal panhysterectomy, Obst. 179
Vaginal cuff formed in Wertheim's operation, Obst. 78
—— secretion, acidity of, cause (W. Blair Bell), Obst. 234
—— —— in infants (G. Drummond Robinson), Obst. 26
—— —— ——, analysis, Obst. 29
Vahle, vaginal secretion in infants, quoted, Obst. 30
Vallet and Rimbaud, intermediate forms between *Bacillus coli* and *Bacillus typhosus*, observed by, Epid. 118

Vansteenberghe and Grysez, experiments to prove introduction of tuberculosis to lungs by way of alimentary tract, Child. 229, 230
Van der Wield, case of escape of cerebro-spinal fluid after injury to head, Otol. 91
Van Harrevelt and Lameris, outbreak of milk-borne diarrhœa in hospital at Stockholm, Epid. 98
Vapour baths, antiquity of, Baln. 85
—— ——, introduction into England, Baln. 85
Variation, anatomical, determined by function, essential doctrine of Lamarckism, Path. 305
Variations, fundamental origin due to changes in germ-cells, Path. 307
Varix of right superficial epigastric vein (L. McGavin), Clin. 51
Vaseline, liquid, mentholized, injection of, curing frontal sinus suppuration, Laryng. 147
Vasomotor centre, effect of baths on, Baln. 93
—— ——, paralysis of, cause of heart-failure in acute disease, Baln. 93
—— symptoms of hysteria, Neur. 17
Vegetables, bread diet should be supplemented by, Odont. 98
—— containing sulphur, Therap. 22, 23
——, unclean or decaying, as source of infection of sporotrichosis, Derm. 115
Venesection to relieve dyspnœa in case of pneumococcal bronchitis, Med. 102
Ventnor, health resort suitable for foreign invalids, Baln. 20
Ventricle (cardiac), when isolated, can beat with independent rhythm, Therap. 97
—— —— (right), infective polypoid thrombus in pulmonary artery, extending into right ventricle, mistaken for patent ductus arteriosus (T. R. Whipham), Child. 199
——, see also *Fibrillation*, electric effects in, ventricular
"Ventricle-stripping operation" for relief of "whistling" and "roaring" in horses, illustrated by specimens of equine larynx and records of cases, demonstrations of, instruments (Frederick Hobday), Laryng, 87
Ventricles (cardiac) and auricles, isolated independent rhythm of heart-beat, Therap. 97
Ventricular complex, Med. 85-88, 93
—— ——, normal, Med. 91
—— contraction, point of origin, Med. 87
—— —— premature, Med. 88-90
—— —— ——, electrocardiograms showing, Med. 89, 90
—— representative, electrocardiogram showing, Med. 86
Ventricular voice in woman, aged 28 (F. F. Muecke), Laryng. 106
Vernon-Harcourt regulator used for chloroform inhalation, preceded by hypodermic injection of scopolamorphine and atropine, Anæsth. 50, 53, 56
Veronal, hypodermic injection before chloroform inhalation, Anæsth. 44
Vertebræ (cervical), skiagraphy of, Electr. 90
Vertebral column and inner border of scapula, bony connexion between, in Sprenge's deformity, detected by radiography, Electr. 69
Vertigo, accompanying onset of congenital syphilitic deafness, Otol. 4, 5, 7
—— (acute), with repeated falls always to left, during two months (H. J. Davis) Otol. 71
—— associated with unilateral deafness, probably complete (Dan McKenzie), Otol. 60
—— (aural) in tertiary syphilis affecting perceptive portion of ear, Otol. 15
—— following injury to head, Otol. 92
—— (labyrinthine) (Meniére's symptoms—non-infective) treated by operation (G. J. Jenkins), Otol. 116
——, surgical treatment possible, Otol. 103
——, with vomiting accompanying onset of congenital syphilitic deafness, Otol. 4, 5, 7
Verworn, nature of Nissl substance, quoted, Path. 175
Vichy, freedom from typhoid outbreak, an argument against infective agency of "carriers," Epid. 19
Vichy douche (Harrogate), effect on low blood-pressure, Med. 71

Vicious circle created by impaired digestion, Baln. 39
Vierordt, Professor, on physical methods of treatment, quoted, Baln. 71
Villages (country), introduction of pulmonary tuberculosis into, Epid. 42
Villemin on tuberculous disposition, quoted, Epid. 158, 159
Vinaj, Professor, Lecturer on Medical Hydrology in the University of Turin, Baln. 115, 116
Virchow, ætiology of multiple cartilaginous exostoses, quoted, Med. 3
——, attitude towards Darwinian theory, Path. 306
——, identity of Charcot's disease with osteo-arthritis, Baln. 24
——, on tuberculous disposition, quoted, Epid. 158
——, "ossific diathesis" of, Surg. 137
——, "Transformation and descent," Path. 306
Viscera, congenital transposition, case of (F. P. Weber), Child. 33
Visceral crises, division of posterior spinal nerve-roots for, Surg. 206
—— ——, pain of, simulating that of organic disease, Surg. 227
Viscosimeter, Determann's clinical, Clin. 88
Visick, H.—Discussion on drugs used as local anæsthetics in the mouth, Therap. 59
Vision, field of, hysterical restriction, Neur. 4
Vitality, lowered, and post-operative chest troubles, causal relationship between, Anæsth. 6
Vitreous, meningococcus in, in cases of associated metastatic ophthalmitis and arthritis of left wrist-joint, Path. 242, 243, 249
Vittel, waters of, contain much calcareous matter, Baln. 124
—— ——, harmless to sufferers from oxaluria, Baln. 124
Vocal cord (left) ulceration of, in male, aged 67, case for diagnosis (G. C. Cathcart), Laryng. 63
—— —— (right) and apex of vocal process, swelling at junction of (J. Donelan), Laryng. 80
—— cords, "dents de scie" of, case illustrating (W. J. Horne), Laryng. 10
—— ——, inequality of planes of, in pachydermia laryngis, Laryng. 63
—— ——, nodules on, removal by galvano-cautery (indirect method), cases illustrating results (E. A. Peters), Laryng. 40
—— ——, tuberculosis of, case (L. H. Pegler), Laryng. 76
Voelcker, A. F.—Discussion on absence of abdominal respiratory movements in pericarditis, Med. 48
——, discussion on auscultation of joints, Med. 39
Vogl, A., on instruction in hydrotherapy, quoted, Baln. 114
Voice (ventricular), in woman, aged 28 (F. F. Muecke), Laryng. 106
Voit and Bauer, absorption of proteins by rectum, quoted, Therap. 64
Volcanic action, connexion of medicinal springs with, Baln. 106
—— eruption, attenuated thermal waters of same nature as, Baln. 107
Volk, macular atrophy in syphilis, quoted, Derm. 124
Vomiting as complication or effect of use of digitalis, Therap. 30, 35-38
—— complicating rectal feeding, Therap. 72
—— following operations under anæsthetics, Anæsth. 2
—— (post-anæsthetic), causes of, Anæsth. 9, 10
—— ——, prevention of, Anæsth. 10
——, stubborn, in gastritis, methods of electricity to control, Electr. 41
——, under inhalation anæsthesia preceded by hypodermic injection of drugs, Anæsth. 50-53
—— with vertigo, accompanying onset of congenital syphilitic deafness, Otol. 4, 5
Vulva, sarcoma of (A. H. N. Lewers). Obst. 64
—— ——, microscopical examination, Obst. 66
—— —— (melanotic) of (Clifford White), Obst. 66
—— —— ——, report of Pathology Committee, Obst. 68
Vulvitis (leukoplakic) and kraurosis, communications on (1909-10), summarized, Obst. 4

WAGGETT, E. B.—Discussion on case of acute inflammation of mastoid treated by Bier's method, Otol. 59
—— discusssion on case of lateral sinus thrombosis, Otol. 33
—— —— on empyema of maxillary antrum in boy, Laryng. 32
—— —— on syphilis in relation to otology, Otol. 23
—— —— on treatment of frontal sinus discharge, Laryng. 148
—— —— on two cases of cerebellar abscess, Otol. 63
WAINWRIGHT, L.—Discussion on calcareous (hard) drinking water, Baln. 127
Waking state, suggestion in, useful in supplying pre-anæsthetic suggestion, Anæsth. 23
—— ——, suggestive anæsthesia obtained in, Anæsth. 19
Waldeyer, views on appendicular ovarian ligament, Obst. 343
Walker, E. W. Ainley, differentiation of streptococci, quoted, Epid. 70
——, mutation of micro-organisms, quoted, Epid. 124
WALLACE, A. J.—Discussion on indications for, and technique of, Cæsarean section and its alternatives, Obst. 202
——, ovarian tumours obstructing delivery, quoted, Obst. 255
WALLACE, J. SIM.—Experiments and observations on bread, with special reference to the causation and prevention of dental caries, Odont. 90-104
WALLIS, Sir F. C.—Discussion on operative treatment of cancer of rectum, Surg. 122
WALLIS, G. F. C.—Discussion on diseases of nose and accessory sinuses in relation to affections of eye and orbit, Laryng. 164-166
WALTERS, C. F., and FINZI, N. S.—Tumour of the cheek, Laryng. 160
WALTON, A. J.—Discussion on fractures in neighbourhood of joints, Surg. 39
Wandless, atypical examples of amaurotic idiocy (Tay-Sachs disease) in family five-sixths Irish, recorded by, Path. 168, 169
WARD, L. E. BARRINGTON.—Discussion on treatment of cleft palate, Surg. 191
WARDE, W. B.—Discussion on case for diagnosis, Derm. 63
WARNER, J. A., and FOX, W.—Case for diagnosis, Derm. 40
—— ——, report on case for diagnosis (sarcoma cutis), Derm. 81
Warnock, percentage incidence of gall-stones based upon post-mortem records of Peckham House Asylum, recorded by, Path. 88
WARREN, R.—Discussion on fractures in neighbourhood of joints, Surg. 27
WARRINGTON, W. B.—Discussion on case of toxic neuritis of doubtful nature, Neur. 22
—— —— cases of spondylose rhizomélique and spondylitis with progressive muscular contracture, Neur. 44
——, observations on experimental division of posterior spinal nerve-roots, quoted, Surg. 210, 219, 220
——, persistent rigidity of spinal column in spondylitis, quoted, Baln. 32
Warry, J. King, outbreak of milk-borne sore throat at Hackney, reported by, Epid. 91
Wartmann and Boehm, experiments on use of aconite, made by, Therap. 157
Warts upon face, liability to develop epithelioma or rodent ulcer, Electr. 51
Washington, U.S.A., typhoid fever carriers at, Epid. 19
Wassermann, use of bacillary extracts introduced by, Path. 252
Wassermann reaction in mental deficiency, results, Child. 171
Water as vehicle of energy, Baln. 81
——, branches of medical doctrine of, Baln. 74
——, different kinds of, earliest references to effects on constitution, Baln. 1, 2
—— (distilled), addition of calcium salts to, Baln. 122, 123
—— —— —— —— —— ——, lessens solvent action of water on sodium biurate, Baln. 123
—— (drinking), calcium salts in, Baln. 128
—— ——, calcareous hard, constipation attributed to, Baln. 125
—— —— —— ——, effects of, Baln. 119, 120
—— —— —— —— ——, on horses, Baln. 122
—— —— —— ——, in relation to gout, Baln, 119, 120
—— —— —— ——, influence alleged to be baneful, Baln. 119

Water (drinking), calcareous hard, influence alleged to be baneful, contradicted, Baln. 123
—— —— —— —— —— in health and disease, Baln. 119-128
—— —— —— ——, mortality of towns of Great Britain supplied with less than that of those supplied with soft water, Baln. 123
—— —— —— ——, production of calculus through, questioned, Baln. 124
—— —— (soft), entails no immunity to gout or rheumatism, Baln. 124
—— —— ——, possesses no special advantages over hard, Baln. 123
——, first factor in spa treatment, Baln. 72
——, fluidifying action on solids, Baln. 107
——, ionization in, Baln. 107
——, sewage-contaminated, bathing in, possible source of infection of poliomyelitis, Epid. 206
——, use of, most important branch of physical therapeutics, Baln. 75
Waters (arsenical), colloidal arsenic in, Baln. 105
—— —— of La Bourboule, hypodermic administration, Baln. 108
—— (natural), isotonicity with blood-serum, Baln. 108
—— of Bagnoles d'Orne, Baln. 72
—— of infiltration, Baln. 106
—— ——, physical properties of, Baln. 74
——, primitive or virgin, Baln. 106, 107
—— (sulphurous), colloidal matters in, Baln. 105
—— (thermal), attenuated volcanic eruption, Baln. 107
—— ——, colloidal metallic solutions in, Therap. 104, 105
——, use of, most important branch of physical therapeutics, Baln. 75
Water-cure, establishment by Priessnitz, Baln. 84
Water-pap, food for weaned children in eighteenth century, Child. 121, 122
Water supply at Folkestone, analysis, Baln. 127, 128
—— —— of London, Commission to investigate (1851), findings of, Baln. 121
WATERHOUSE, H. F.—Discussion on surgical treatment of cancer of rectum, Surg. 112
Watering-places (Marine), see *Health resorts* (Marine)
WATSON, C. GORDON.—Case of resection of cæcum and ascending colon, showing chronic hypertrophy of mucous membrane, with stenosis simulating cancer, Path. 237
——, discussion on case of Graves's disease, Clin. 125
—— —— on case of septic gastritis, Clin. 141
—— —— on operative treatment of cancer of rectum, Surg. 129
WATSON-WILLIAMS, P.—Case of carcinoma of larynx, Laryng. 93
——, case of malignant neoplasm of the larynx, Laryng. 16
—— —— of malignant stricture of the œsophagus, Laryng. 83, 153
——, chronic glossitis ? cause, Laryng. 158
—— —— suppurative mastoiditis, with abscesses in the left temporo-sphenoidal and frontal lobes, Otol. 134
——, cystic middle turbinal (right), Laryng. 159
——, demonstration of direct bronchoscopy on a patient, Laryng. 151
——, discussion on bacteriology of chronic post-nasal catarrh, Otol. 51
—— —— on case for diagnosis, Laryng. 57
—— —— —— of antrum suppuration: nasal antrostomy followed by blocking of nasal duct, Laryng. 103
—— —— —— of bilateral tuberculous disease of tonsils, Laryng. 69
—— —— —— of endothelioma of palate, with metastatic growth in neck, treated by radium, Laryng. 95
—— —— —— of lateral sinus thrombosis, Otol. 34
—— —— —— of lingual thyroid, Laryng. 13
—— —— —— of nasopharyngeal fibrous polypus, Laryng. 62
—— —— —— of party-wall cancer of larynx and œsophagus, Laryng. 101
—— —— —— —— pharyngeal cancer, Laryng. 55

WATSON-WILLIAMS, P.—Discussion on case of recurring aphonia in a woman, aged 36, Laryng. 82

——, discussion on case of removal of rabbit-bone from right bronchus, Laryng. 98

—— —— —— —— of upper jaw in man, aged 71, Laryng. 86

—— —— —— of swelling at junction of right vocal cord and apex of vocal process, Laryng. 80

—— —— —— of temporary deafness and blindness due to intestinal toxæmia, Otol. 123

—— —— —— of tuberculosis of vocal cords in young man, Laryng. 78

—— —— —— of ulceration of soft palate, Laryng. 12

—— —— on cases illustrating results of removing nodules from cords by means of galvano-cautery (by indirect method), Laryng. 41

—— —— on complete stenosis of the larynx from epithelioma in a man, aged 23, Laryng. 120, 121

—— —— on deafness due to occlusion of Eustachian tubes by scar-tissue, Otol. 110

—— —— on diseases of nose and accessory sinuses in relation to affections of eye and orbit, Laryng. 166

—— —— on ectopia of pituitary, Laryng. 53-54

—— —— on empyema of maxillary antrum in boy, Laryng. 32

—— —— on equine larynx and "ventricle-stripping," Laryng. 89, 91

—— —— on instrument for efficient removal of nasal spine in submucous resection of septum, Laryng. 25

—— —— on large nasopharyngeal polypus removed from boy, Laryng. 33

—— —— on laryngeal tuberculosis, cure (?), Laryng. 43

—— —— —— —— in a boy, aged 10, Laryng. 116

—— —— on lupus of larynx in woman, Laryng. 35

—— —— on new pattern expanding œsophagoscope, Laryng. 58

—— —— on new type of œsophago-gastroscope, Laryng. 73

—— —— on origin of cancer in larynx, Laryng. 23

—— —— on removal of thyro-lingual cyst, Laryng. 28

—— —— on specimen of pedunculated growth on an elongated uvula, Laryng. 108

—— —— on stricture of lower end of œsophagus, Laryng. 38

—— —— on tracheo-laryngostomy for stenosis following tracheo-laryngotomy, Laryng. 112

—— —— on two cases of malignant disease of tonsil, pharynx, and tongue, Laryng. 114

—— —— on two specimens of quadrilateral cartilage (nasal septum); removed by submucous section, Laryng. 6

——, further notes on case of epithelioma laryngis, Laryng. 153

——, patients illustrating results of Dr. Watson-Williams's osteoplastic radical frontal sinus operations, Laryng. 154-157

——, pemphigus of pharynx and conjunctiva, Laryng. 158

——, the present position of the treatment of purulent discharge from the sinuses; an address introductory to a discussion on the subject, Laryng. 127-138

——, Presidential Address (Laryngological Section), Laryng. 1

——, remarks at presentation of memorial to Sir F. Semon, Laryng. 150

——, tertiary specific ulcer of pharynx, Laryng. 158

Watson-Williams's plastic operation in chronic frontal sinus suppuration, Laryng. 134, 135, 148

—— —— —— —— —— —— ——, summary of cases treated by, Laryng. 136-138

de Watteville current in treatment of gastric disorders, Electr. 41

WAUGH, G. E.—Discussion on treatment of cleft palate, Surg. 186

Wax, aseptic, packing cavity with, after operation for frontal sinus suppuration, Laryng. 146, 148

Weaning after cutting of first teeth, Child. 119, 120

—— by the moon, superstition regarding, Child. 119

——, method of accustoming children to artificial food after, Child. 119

——, water-pap food for children after, in eighteenth century, Child. 122

Weather, states of, relation of epidemics and other diseases to, Epid. 189

WEBB, J. CURTIS.—Discussion on diathermic treatment, Electr. 9
Weber, cartilaginous exostoses and multiple enchondromata, quoted, Med. 3
Weber, fracture of skull followed by labyrinthine suppuration proving fatal twenty-one months later, case quoted, Otol. 89
WEBER, F. PARKES.—Chronic (artificial?) skin eruption in a patient whose case has been followed for fourteen years, Derm. 43
——, congenital heart disease with congenital malformation of the external ear, Child. 161
—— —— —— —— without murmur, and with a family history of congenital cyanosis, Child. 159
—— congenital transposition of viscera, Child, 33
——, discussion on after-history of albuminuria in adolescence, Med. 138
—— —— on case of anorexia nervosa, Child. 80
—— —— —— of congenital syphilis treated by "606," Child. 159
—— —— —— of congenital xanthoma (endothelioma), Derm. 77
—— —— —— of erythema ab igne, Derm. 89
—— —— —— —— —— ——, or livedo reticularis with pigmentation, Derm. 47
—— —— —— of infantilism, Clin. 46
—— —— —— of inherited syphilis and "blue sclerotics," Child. 99
—— —— —— of large cavernous nævus of foot, Clin. 84
—— —— —— of multiple cutaneous telangiectases of recent origin, Clin. 44
—— —— —— of persistent jaundice, Child. 62
—— —— —— of splenomegaly (? congenital family cholæmia), Clin. 130
—— —— —— of spondylitis deformans, Clin. 29
—— —— —— of symmetrical lipomatosis, Clin. 6
—— —— —— of tropho-neurotic (?) separation of nails, Derm. 137
—— —— —— of unilateral pigmentary nævus, Derm. 59
—— —— —— of urgent dyspnœa and œdema of glottis, Clin. 25
—— —— on cases of adiposis dolorosa, Clin. 6
—— —— —— of familial pigmentary dermo-fibromatosis, Clin. 75
—— —— —— showing unusual ulceration of skin, Clin. 112
—— —— on "congenital elephantiasis" of arm, Child. 24
—— —— on fibromatous tumours, Derm. 102
—— —— on rigidity of spine, Clin. 148
—— —— on treatment of acute suppression of urine by incision and drainage of kidneys, Clin. 168
—— —— —— of splenic anæmia by splenectomy, Clin. 70
—— —— on tumour of rectus abdominis in child, Child. 8
—— —— on ulcerative stomatitis in children, Child. 73
—— —— on varix of right superficial epigastric vein, Clin. 51
——, lichen scrofulosorum imitating psoriasis, Derm. 93
——, nævus verrucosus linearis, Child. 32
——, splenomegaly with recurrent jaundice, ending in hepatic cirrhosis and ascites, with remarks on the splenomegaly of inherited syphilis in children, Child. 62
——, and DORNER, G.—Congenital obliteration (or congenital atresia) of bile-ducts with hepatic cirrhosis, Child. 41-50
—— ——, case of congenital pulmonary stenosis with special consideration of the nature of the secondary blood changes, Clin. 85-94
WEBER, Sir HERMANN.—Discussion on outlines of medical hydrology (Hyde Memorial Lectures, Lecture I), Baln. 80
——, on constipation, quoted, Baln. 125
——, translation of Braun's "Curative effects of baths and waters" (1874), made by, Baln. 65
Webster, action of atropine on heart, quoted, Anæsth. 54, 55
Wechsberg and Neisser, functions of free amboceptors, Path. 276
Weil, E., association of secondary polycythæmia of congenital heart disease with erythroblastic reaction in bone-marrow, quoted, Clin. 93

Weintraud, percentage of albumin in blood and blood-serum in erythræmia, recorded by Clin. 92
Weismann, A., climatic influence in relation to transmissibility of acquired characters, quoted, Path. 307
——, denial of transmissibility of acquired characters, Path. 280
Welch, bovine tuberculosis infection, quoted, Child, 231
Wellington College, prevalence of measles at, Child. 175
Wells, S., Russell.—Discussion on multiple cartilaginous exostoses, Med. 21
Welsh miners, difficulty of anæsthetizing, Anæsth. 5, 11
Werdnig-Hoffmann type of progressive spinal muscular atrophy of infants and young children (F. E. Batten), Clin. 115, Neur. 54, 67
Wernicke, paratyphoid carriers, quoted, Epid. 57
Wertheim, Professor E., personal cases operated on by his method of abdominal panhysterectomy, primary mortality, Obst. 95
—— —— —— —— —— —— —— —— ——. ultimate results, Obst. 98
——, practice as regards removal of appendix in pelvic operations, Obst. 352
—— method of abdominal panhysterectomy, Obst. 78
—— —— —— ——, cases operated on by (C. P. Childe), Obst. 79-100
Wesley, John, "Primitive Physick, or an easy and natural method of curing most diseases" (1747), Baln. 82
West, C. Ernest.—Bacteriology of chronic post-nasal catarrh; a preliminary study of fifty cases, Otol. 43-50
——, case of chronic infective labyrinthitis, Otol. 39
——, chronic dermatitis of the pinna due to infection by *Staphylococcus aureus*, under treatment by vaccine, Otol. 38
——, discussion on case of acute inflammation of mastoid treated by Bier's method, Otol. 59
—— —— —— of acute vertigo with repeated falls always to the left, Otol. 72
—— —— —— of caries and necrosis following mastoiditis due to scarlet fever, Otol. 70
—— —— —— of chronic otitis media suppurativa, Otol. 66
—— —— —— of congenital malformation of auricle and meatus, Otol. 25
—— —— —— of extradural cerebellar abscess, Otol. 30
—— —— —— of fistula of ampulla, Otol. 76
—— —— —— of infiltration of both auricles, Otol. 38
—— —— —— of lateral sinus thrombosis, Otol. 34
—— —— —— of unilateral deafness associated with vertigo, Otol. 61
—— —— on deafness due to occlusion of Eustachian tubes by scar-tissue, Otol. 110
—— —— on large tuberculous polyp removed from middle ear of infant, Otol. 42
—— —— on syphilis in relation to otology, Otol. 19
—— —— on three specimens of chronic middle-ear suppuration, Otol. 112
—— —— on two cases of cerebellar abscess, Otol. 63
West, Samuel.—Case of acute suffocative catarrh of pneumococcal origin and fatal (pneumococcal) bronchitis; a second case of pneumococcal bronchitis, unilateral, of long duration and not fatal, with other analogous cases and remarks, Med. 101
——, discussion on after-history of albuminuria in adolescence, Med. 133
West Indies, Obeah system in, Anæsth. 23, 24
Westmacott, F. H.—Cast of cavity in the left mastoid process of a woman, aged 75, filled with cholesteatoma—smooth polished surfaces, Otol. 124
——, discussion on large tuberculous polyp removed from middle ear of infant, Otol. 42
—— —— on new growth of unknown nature in tympanum, Otol. 77
—— —— on unilateral optic neuritis and complete ophthalmoplegia externa resulting from acute sphenoidal sinusitis, Laryng. 30
——, eight specimens of the otic element of the Arctic whale, Otol. 134
——, sequestration of bony labyrinth in a boy, aged 4, Otol. 123
Westminster Hospital, B. family from, showing multiple cartilaginous exostoses, Med. 4
Weston-super-Mare, Baln. 18

Westphalia, epidemic of poliomyelitis in (1908), Epid. 207
Wet-nurses, choice of, Child. 118, 119
——, decline of, Child. 125
——, in Elizabethan period, Child. 112, 113
——, mothers of illegitimate children selected for, by preference, Child. 124
——, superstitions regarding, Child. 113
——, zenith of prosperity, Child. 123, 124
Wet pack in neurasthenia, Baln. 43
Whale (Arctic), otic element of, eight specimens (F. H. Westmacott), Otol. 124
Whipham T. R.—Case of enlargement of the liver, Child. 54
——, case of patent ductus arteriosus, Child. 31
——, discussion on case of rheumatoid arthritis, Child. 38
—— —— on Dr. Milne's paper, "Measles: its treatment and prevention," Child. 179
—— —— on portals of entry in phthisis, Child. 226
——, heart showing an infective polypoid thrombus in the pulmonary artery and extending into the right ventricle, mistaken for patent ductus arteriosus, Child. 199
——, pathological specimen of the heart in a case of congenital malformation, Child. 3
White, Clifford.—A fœtus with congenital absence of the sacrum, Obst. 279
——, melanotic sarcoma of the vulva, Obst. 66
—— —— —— ——, report of Pathology Committee, Obst. 68
White, W. Hale, gall-stones as factor in causation of primary carcinoma of gall-bladder and bile-ducts, quoted, Path. 93
——, percentage incidence of gall-stones in post-mortem examinations, quoted, Path. 88
Whitehead, A. L.—Discussion on case of fistula of the ampulla, Otol. 77
——, discussion on deafness due to occlusion of Eustachian tubes by scar-tissue, Otol. 110
—— —— on eight cases of mastoid operation (Case I), Otol. 126; (Case III), Otol. 129; (Case IV), Otol. 130
—— —— on syphilis in relation to otology, Otol. 20
——, notes of a case of temporary deafness and blindness due to intestinal toxæmia, Otol. 121
Whitelocke, R. H. A.—Discussion on fractures in neighbourhood of joints, Surg. 26
——, discussion on myositis ossificans traumatica, Surg. 157
—— —— on portals of entry in phthisis, Child. 225
Whitfield, A.—Atrophic lichen planus with cornu cutaneum, Derm. 11; discussion (p. 12): Dr. Kenneth Wills, Dr. Whitfield (reply)
——, case of parakeratosis variegata? Derm. 30
——, cultures of *Trichophyton roseum* and *ectothrix*, and specimen of trichophytic invasion, Derm. 93
——, discussion on cases for diagnosis, Derm. 25, 35, 43, 70, 95
—— —— on case of acute lupus erythematosus, Derm. 51
—— —— —— —— —— pityriasis of scalp, Derm. 142
—— —— —— of disseminated tuberculous nodules of skin, Derm. 49
—— —— —— of erythema ab igne, or livedo reticularis, Derm. 47, 89
—— —— ——, of folliculitis decalvans, Derm. 145
—— —— —— of pityriasis rubra pilaris, Derm. 24
—— —— —— of sycosiform syphilide, Derm. 106
—— —— on fibromatous tumours, Derm. 102
—— —— on macular atrophy associated with lupus erythematosus, Derm. 144
—— —— on report on case for diagnosis (sarcoma cutis), Derm. 82
—— —— on syphilitic affection of eighth nerve, Derm. 148
—— —— on unusual case of linear nævus, Derm. 39
——, series of ringworm cultures, illustrating the eczematoid ringworm of the hands and feet, Derm. 111
Whitla, Sir W., and Symmers, StC., experiments to prove introduction of tuberculosis by way of alimentary tract, Child. 205, 229
Wholemeal bread, Odont. 97, 99

Whooping-cough, prevalence of, relation to weather. Epid. 190
Wick, Dr. L., lecturer on balneology and climatology at Vienna, Baln. 115
Wickman, epidemics of poliomyelitis in Sweden (1899 and 1905), recorded by, Epid. 200, 204
——, monograph on Heine-Medin's disease, quoted, Child. 94
WILD, R. B.—Action and uses of sulphur and certain of its compounds as intestinal antiseptics, Therap. 13-24
——, discussion on therapeutic use of digitalis group, Therap. 36
WILLIAMS, A. W.—A very extensive case of lupus in an elderly woman, Derm. 96
——, case of angiokeratoma, Derm. 95
—— —— of keratosis of finger, Derm. 32
—— —— for diagnosis, Derm. 95
——, epithelioma involving the whole of the lower lip and the entire skin of the chin, showing a close clinical resemblance to actinomycosis, Derm. 14
——, gonorrhœal keratosis of hands and feet, Derm. 12
——, sclerodermia with obstinate callous ulcers on the legs greatly improving under mercurial inunction, Derm. 13
WILLIAMS, C. THEODORE, M.V.O.—Discussion on outlines of medical hydrology (Samuel Hyde Memorial Lectures—Lecture III, the Medicinal Spring), Baln. 118
—— discussion on tuberculosis and its prevention, Epid. 43
Williams, J. Whitridge, anaerobic germs in puerperal infection, Obst. 167
——, indications for craniotomy, quoted, Obst. 166
WILLIAMS, LEONARD.—Discussion on calcareous (hard) drinking water, Baln. 128
——, discussion on case of Graves's disease, Clin. 124
—— —— on osteo-arthritis and chronic rheumatism, Baln. 35
——, on internal secretions, quoted, Baln. 27
——, on risks of over-stimulating or bracing climate, quoted, Baln. 69
WILLIAMS, LLEWELLYN.—Discussion on paratyphoid fever and "food-poisoning" (communication by letter), Epid. 63
WILLIAMSON, OLIVER K.—Case of secondary optic atrophy due to a (?) cerebellar tumour, Child. 109
——, discussion on low blood-pressure, Med. 76
WILLS, KENNETH.—Discussion on case of acute lupus erythematosus, Derm. 51
——, discussion on case of atrophic lichen planus with cornu cutaneum, Derm. 12
Wilson, acid and gas-forming power of bacillus isolated from urine, quoted, Path. 97
WILSON, Professor ERNEST and W. H.—A new induction coil, Electr. 79-83
Wilson, G., outbreak of milk-borne sore throat at Rugby, Epid. 89
Winchester, outbreak of typhoid fever at, Epid. 131
Winds, strong, prevalent, rain-bearing, phthisis morbidity in localities exposed to, Baln. 17
Windscheid, Professor, lecturer on hydrotherapy at Leipzig, Baln. 115
Wine, recommended for young children in eighteenth century, Child. 123
WINGFIELD, H.—Discussion on hypnotism in relation to surgical anæsthesia, Anæsth. 21
Winterbottom, case of foreign body remaining in ear for sixty years, reported by, Otol. 82
Winter Gardens, open-air galleries should be substituted for at British spas, Baln. 15
Winternitz, Professor W., demonstrations and lectures on hydrotherapy at Kaltenleutgeben, Baln. 115
——, lifework of, Baln. 86
——, modus operandi of baths, quoted, Baln. 93
——, on hydrotherapy in England, quoted, Baln. 97
——, physiological and clinical basis of hydrotherapy, quoted, Baln. 77, 86, 89
Wolff's law as to structure and function of bone, Surg. 2, 4
Wolffian tubules, derivation of adenomatosis vaginæ from, negatived, Obst. 24
Women, administration of open ether to, results satisfactory, Anæsth. 51
——, appendicitis in, Obst. 347
—— ——, adhesions between wall of appendix and cæcum, Obst. 347
——, arterio-sclerosis in, principal factor producing, Med. 63

Women, chief sufferers from adiposis dolorosa, Clin. 7
——, habitual constipation in, Obst. 328
——, hypodermic injection of drugs before inhalation anæsthesia in, Anæsth. 50, 51
——, liability to chronic rheumatism at menopause, Baln. 28, 34
——, long appendix in, Obst. 348
Wood, Douglas.—Congenital deformity of the palate, Child. 5
Wood, Neville.—British health resorts for foreign invalids, Baln. 11-21
——, on freshness of water of mountain streams. quoted, Baln. 72
Woodforde, A. W. G.—Specimens from a case of "delayed chloroform poisoning," Child. 81
——, and Smith, B. E.—A preliminary communication on syphilis and mental deficiency, Child. 166
Woodhall Spa, Baln. 15
Woodhouse, A. Clayton.—Discussion on case of absorption of permanent molar due to unerupted premolar, Odont. 38
——, discussion on case of multiple supernumerary teeth preceding central incisor, Odont. 47
—— on model of maxilla with exostoses, Odont. 41
Woodman, E. Musgrave.—Discussion on case of tabes dorsalis, with early rectal crises, Clin. 118
——, ectopia vesicæ in a female child, Clin. 157
Wooldridge's experiments as to independent heart-beat in auricles, Therap. 97
Wooldridge, Professor E. H.—Discussion on diseases of cow affecting milk in relation to human disease, Epid. 102
Word-blindness co-existent with high mental development, Child. 52
—— (congenital), intermittent, case of (E. Pritchard), Child. 51
Workington, epidemic of poliomyelitis at (1910), Epid. 212
Wormwood, smearing of nipples with, to overcome child's dislike to strange food, Child. 119
Worry, effect on low blood-pressure, Med. 57, 79, 80
Worth, C., percentage of age of commencement of unilateral convergent squint, Child, 89
Worthing, typhoid fever at (1881-1909), Epid. 7
—— —— ——, epidemic at, 1893, cases reported subsequently as an indication of carrier transmission, Epid. 8, 9
—— —— —— —— ——, secondary infections, Epid. 132
Wrexham, outbreak of food-poisoning at, Epid. 65
—— —— of gastro-enteritis at, due to *Bacillus paratyphosus*, Epid. 57
Wright, Sir A. E., F.R.S., determination of nature of pathogenic micro-organisms on uterine swabbings by, Obst. 171, 212
——, successful case of vaccine treatment of frontal sinus suppuration, under, Laryng. 139
Wright, A. J. M.—Case for diagnosis, Laryng. 56
——, demonstration of direct bronchoscopy on a patient, Laryng. 151
——, discussion on diseases of the nose and accessory sinuses in relation to affections of eye and orbit, Laryng. 164
——, œdema of conjunctiva from acute frontal sinusitis, Laryng. 160
——, paralysis of right third nerve following ethmoidal operation, Laryng. 159
——, results of orbital cellulitis following ethmoidal operation, Laryng. 159
Wright, J. A.—Case of cretinism, Child. 189
——, cerebellar ataxia, Child. 190
——, child with a peculiar gait (? functional ataxia), Child. 191
—— congenital heart disease (pulmonary stenosis), cerebral hemiplegia, Child. 190
——, demonstration of pathological specimens, Child. 204
Wright, K.—Discussion the problem of disposition in pulmonary tuberculosis, Epid. 184
Wright, William, advocacy of cold-bath treatment of fevers in Scotland, by (*c.* 1780), Baln. 83
Wrisberg, pars intermedia of, degenerative changes in Otol. 15, 17
Wrist, Colles's fracture of, Surg., 29, 30
——, hyper-extension of, in treatment of fracture of scaphoid, Surg. 3
Wrist-joint, left, arthritis of, association of metastatic ophthalmitis with, Path. 242, 243

Würzburg, University of, lectures on physical therapy and balneology at, Baln. 115
Wynter, W. Essex.—Absence of abdominal respiratory movement as an indication of pericarditis, Med. 40-50, 149
——, case of chloroma, quoted, Path. 67
——, discussion on rigidity of spine, Clin. 149
——, Graves's disease in a boy, commencing at the age of 10, Clin. 155
——, two cases of Henoch's purpura, Clin. 48

Xanthoma, congenital (endothelioma), case of (J. E. R. McDonagh), Derm. 76
—— diabeticorum, Derm. 77
X-ray and bismuth, gastric and intestinal spasm visible under observation by, Med. 29, 31, 32
—— examination in mollities ossium of forty-five years' duration, Clin. 99, 104
—— —— of benign cyst of tibia, Clin. 133, 139
—— tube, fixed, with patient movable, Electr. 85
—— ——, hardness of, determination by Bauer's qualimeter (W. Deane Butcher), Electr. 71
—— ——, see also *Tube-stand*
X-rays, action intensified and exposure to shortened by dusting over ulcerated surfaces with carbonate or oxychloride of bismuth, Electr. 57
——, alterations produced by in areas remote from those under treatment, Electr. 53
——, detection of atheroma of aorta by, Electr. 26, 29
——, diagnosis of fractures about joints by, Surg. 17-19, 21, 36
—— —— ——, by, limitations, Surg. 1, 2, 31, 32
——, difference in quality, how demonstrated, Electr. 58
——, dosage of, Electr. 54, 63, 68
——, examination by, in detection of thoracic aneurysm, Electr. 22, 23
—— ——, inhibition of action of diaphragm seen under, Med. 44
—— —— of œsophagus under (A. C. Jordan), Electr. 13-30
—— —— —— ——, method, Electr. 13, 14
—— —— —— ——, by means of bismuth meal, Electr. 14-23, 27
—— ——, see also *Screen examination*
——, exposure to, prolonged and unscientific, malignant growths resulting from, Electr. 53
——, hard better than soft, in treatment of cancer, Electr. 62, 63
—— in diagnosis of fracture of patella, Surg. 69-82
——, indications against operative treatment, ascertained by, Surg. 36
—— (secondary), Electr. 60
—— ——, action of, Electr. 57
—— ——, derived from arsenic, therapeutic effects, Electr. 67
—— —— —— from silver, Electr. 67
—— —— —— ——, therapeutic effects, Electr. 67
—— ——, treatment by, Electr. 63
——, " selective action " of, criticized, Electr. 52, 68
—— —— ——, discussed, Electr. 61, 62
——, treatment by, good results probably due to stimulation of normal cells, Electr. 53
—— ——, technique (F. Fowler), Electr. 61
—— —— in acute lupus erythematosus, not indicated, Derm. 51
—— —— of cancer (J. Hall-Edwards), Electr. 47-69
—— —— ——, cases showing good results, Electr. 64
—— —— ——, illustrative cases, Electr. 55, 56
—— —— ——, method employed (J. Hall-Edwards), Electr. 54
—— —— ——, selection of cases, Electr. 62
—— —— ——, two chief factors in, Electr. 60
—— —— —— of breast encouraging, Electr. 60
—— —— of epithelioma, Electr. 58
—— —— ——, of back of hand, Derm. 142

X-rays, treatment of epithelioma, preliminary to operation, Electr. 58
—— —— of hyperkeratosis of palms and soles, Derm. 18
—— ——, of malignant disease, Electr. 52
—— —— —— ——, symptomatic results encouraging, Electr. 52
—— —— —— ——, ultimate results so far disappointing, Electr. 52
—— —— of ringworm, followed by acute pityriasis of scalp, Derm. 141
—— ——, of rodent ulcer, effect of. Electr. 61
——, value in diagnosis, testimony to, Med. 150

Yarrell, W., specimen of hairless Egyptian variety of *Canis familiaris*, recorded by, Path. 298
YEARSLEY, P. MACLEOD.—Discussion on injuries of ear, Otol. 104
——, discussion on syphilis in relation to otology, Otol. 21
Yellow spot, see *Macula lutea*
YOUNG, E.—Discussion on osteo-arthritis and chronic rheumatism, Baln. 36
Young, H. H., operative treatment of vesical diverticula, quoted, Surg. 162
YOUNG, J.—The uterine mucosa in menstruation and pregnancy, the action of the chorionic cells and the function of the decidua, Obst. 291
Young, T. (1815), on tuberculous contagion, quoted, Epid. 162, 163
—— ——, on the tuberculous disposition, quoted, Epid. 157
YULE, Udny.—Discussion on the problem of disposition in pulmonary tuberculosis, Epid. 178

Zadro, injection of veronal before inhalation anæsthesia, quoted, Anæsth. 44
Zehmisch, percentage of absorption of carbohydrates in rectal feeding, recorded by, Therap. 65
Ziegler, P., treatment of congenital atrichosis in girl with fresh thyroid extract, quoted, Path. 301, 302
Zinc oxide, large percentage present in red rubber pessaries, Obst. 324-326
—— salts, escharotic effect of, Obst. 326
—— sulphate, electrical introduction of, in treatment of gastric ulcer, Electr. 42
Zurbelle, practice as regards removal of appendix in surgical operations, Obst. 352
Zürich, lectures on physical treatment at, Baln. 116
Zweifel, P., practice as regards removal of appendix in pelvic operations, Obst. 352

JOHN BALE, SONS & DANIELSSON, LTD., 83-91, Great Titchfield Street, London, W.

BINDING SECT. AUG 2 1967

R
35
R7
v.4
pt.3

Biological
& Medical
Serials

Royal Society of Medicine,
London
Proceedings

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY